



Evaluation Report for Category B, Subcategory 4.1 Application

Application Number: 2007-8789
Application: B.4.1 (Conversion to full registration without consultation)
Product: Nufarm Mecoprop-p Technical Acid
Registration Number: 27631
Active ingredients (a.i.): Mecoprop P-Isomer (present as acid) (MEP)
PMRA Document Number: 1902219

Background

During the re-evaluation of the active ingredient mecoprop (racemic: 50/50 R/S isomers), the PMRA had identified significant data gaps for racemic mecoprop that would have to be addressed in order to bring the supporting database up to modern standards (Re-evaluation Decision Document for Mecoprop, RRD2004-09). At the time, rather than generating the required data to support continuing registration, the registrants of technical racemic mecoprop had decided to discontinue sales of the racemic form of mecoprop and replace it with a specific isomer of mecoprop known as mecoprop-p.

Purpose of Application

The purpose of this application was to convert Marks Mecoprop-p Technical Acid (Registration Number 27441) to full registration. This application was assessed at the same time as conversion applications for Nufarm Mecoprop-p Technical Acid (Registration Number 27631) and A.H. Marks MCPP-p 600 Liquid Herbicide (Registration Number 28563).

Furthermore, there were approximately 60 associated end-use products assessed for conversion from conditional to full registration. The conversion of these applications was dependent on the conversion of the above three applications.

Chemistry Assessment

The chemistry requirements have been fulfilled.

Health Assessments

Toxicology Summary

The PMRA conducted a detailed review of the toxicological database for mecoprop-p. The database consists of an array of laboratory animal (*in vivo*) and cell culture (*in vitro*) toxicity studies currently required for health hazard assessment purposes. The studies were carried out in accordance with currently accepted international testing protocols and Good Laboratory Practices. The scientific quality of the data is acceptable, and the database is considered adequate to characterize the toxicity of this chemical.

Available acute, short-term, and long-term toxicity data comparing mecoprop-p, mecoprop racemate, mecoprop-p dimethylamine (DMA), and mecoprop-p ethylhexylester (EHE) showed no significant differences in toxicity potential. Systemic toxicity findings in laboratory animals were similar. *In vivo* and *in vitro* dissociation/degradation studies demonstrated that mecoprop-p DMA and mecoprop-p EHE were readily hydrolyzed to mecoprop-p. The degradation study findings supported the view that the toxicity effects observed in studies tested with mecoprop-p DMA and mecoprop-p EHE are mostly due to mecoprop-p. Therefore, toxicity data generated with mecoprop racemate, mecoprop-p DMA, and mecoprop-p EHE can be used to support the registration of mecoprop-p.

Laboratory studies demonstrated that the absorption of mecoprop-p was rapid and extensive following oral administration in the rat. Peak plasma concentrations were seen shortly after exposure. The compound was excreted rapidly in urine with most of the administered radioactivity (AR) collected in urine within 24 hours. Fecal elimination constituted about 4-12% of the AR. A total of >77% of the AR was eliminated within 24-48 hours. No radioactivity was detected in expired air. Tissue distribution of radioactivity was extensive, but the level declined rapidly. The total radioactivity remaining in tissues was low and there was no evidence of accumulation when the rats were killed at 168 hours after dosing. It was concluded that mecoprop-p was absorbed rapidly and extensively, and excreted rapidly in urine, either unchanged, as hydroxylated mecoprop-p, or in the form of one of up to seven minor metabolites. There were no notable gender differences in the metabolic profile of mecoprop-p in the rat.

In the rat, mecoprop-p is moderately to highly acutely toxic by the oral route, but is of low acute toxicity by the dermal and inhalation routes of exposure. Mecoprop-p is extremely irritating to the rabbit eye affecting the cornea, but is only slightly irritating to the rabbit skin. Mecoprop-p is not a skin sensitizer when tested in the guinea pig.

In short- and long-term dietary toxicity studies in mice, rats, and dogs, mecoprop-p induced systemic toxicity at high dose levels. Systemic toxicity invariably involved reduced food intake and lowered body weight and body-weight gains. The liver, kidneys, and the adrenals were the target organs. Liver effects were characterized by higher weights and cellular changes. Although the kidney weights were higher in mecoprop-p exposed rodents, there were no accompanying gross or histopathological changes. The effects in the adrenals were observed only at a high dose level in the rat, and the changes included discolouration and lipid accumulation.

After repeated dermal administration of mecoprop-p in the rabbit for 21 days, only minor local dermal effects were evident, and there was no systemic toxicity up to the limit dose.

In vivo/in vitro genotoxicity studies of mecoprop-p assessing gene mutation, chromosome aberration, and unscheduled DNA synthesis showed negative genotoxic findings. However, tests with mecoprop racemate in *in vitro* chromosome aberration and *in vivo* sister chromatid exchange in Chinese hamster bone marrow cells gave equivocal findings because some positive findings were observed at cytotoxic levels while others were not reproducible.

A two-year combined dietary toxicity and oncogenicity study on mecoprop (racemic mixture) in the rat is deficient because the dose levels tested were not high enough; the highest dose level tested did not produce any effects on mortality, clinical signs, ophthalmoscopy, haematology, clinical chemistry, urinalysis, or gross and histopathology. A second carcinogenicity study of mecoprop-p in the rat was later conducted. Sufficiently high dose levels were tested and the findings showed that mecoprop-p was not carcinogenic in the rat. Two studies were also conducted to assess the oncogenic potential of mecoprop-p in the mouse. In the first study, the highest dose level was terminated early at around one year and the mice at this level were killed without histopathological examination despite the presence of subcutaneous palpable masses in the majority of the high-dose mice. In the second study, a higher incidence of liver adenoma and carcinoma was recorded in the females. Combining the data from the two mouse oncogenicity studies demonstrated that mecoprop-p induced liver neoplasms in the female mouse but not in the male.

Reproductive toxicity data were generated for mecoprop racemate in the rat. There were no effects on the parental animal or on reproductive performance. There were no significant differences in birth, viability, and gestation indices. However, at the highest dose level, there was an increase in pup deaths and lower weight gains during lactation days zero to four. The effects occurred at a dose that did not induce maternal toxicity.

Teratology data on mecoprop racemate in rats, and mecoprop-p in the rat and rabbit did not demonstrate any evidence of teratogenicity. For the racemate, developmental toxicity (higher post-implantation loss, lower foetal weight and foetal crown/rump length) occurred at maternally toxic dose levels. For mecoprop-p, the highest dose level tested in the rabbit did not induce any maternal or developmental toxicity. It appeared that the maximum tolerable dose was not reached in the rabbit teratology study. However, data from other short- and long-term toxicity studies indicated that the highest dose level tested in the rabbit teratology study should be close to an effect level. Thus, the rabbit teratology study is considered adequate. In the rat, the highest dose level tested resulted in lower maternal body weight and increase minor skeletal variations in the pups.

Acute and short-term neurotoxicity data of mecoprop-p in the rat did not demonstrate any evidence of neurotoxicity. There were no triggers in the toxicological database to warrant a study to investigate developmental neurotoxicity.

In summary, mecoprop-p is moderately to highly toxic by the oral route. It is of low toxicity by the dermal and inhalation routes. Mecoprop-p is highly irritating to the eye, but is slightly to moderately irritating to the skin. Mecoprop-p is not a dermal sensitizer. Repeated exposures to mecoprop-p in laboratory animals cause liver, kidney, and adrenal changes, together with reduced body-weight gain at high dose levels. Mecoprop-p is not genotoxic, neurotoxic, or teratogenic. Oncogenicity data in the mouse indicated that mecoprop-p induced a higher incidence of liver tumours in the female. Reproductive toxicity study of the racemate in the rat showed that there was an increase in pup deaths at a dose level that did not cause maternal toxicity.

Results of the acute and repeat exposure tests conducted on laboratory animals with mecoprop-p technical, along with the toxicology endpoints for use in the human health risk assessment, are summarized in Tables 1, 2, and 3 of Appendix I.

Incident Reports

Starting April 26, 2007, registrants have been required by law to report incidents, including adverse effects to health and the environment, to the PMRA within a set time frame. Information on the report of incidents can be found on the PMRA website.

As of April 30, 2010, 49 incident reports involving humans (29) and domestic animals (20) were received by the PMRA. All incidents involved end-use products containing 2,4-D, dicamba, or 2,4-dichloroprop-P, and mecoprop-p as the active ingredients. In no cases were products containing mecoprop-p as the sole active ingredients involved. In most cases, exposure was dermal via accidental contact with the products or of lawns treated with weed control products and fertilizers; direct ingestion of the products was not confirmed or witnessed.

Causality has not been established for the effects noted in the incident reports for mecoprop-p exposure; the symptoms noted in the reports were non-specific and could be attributed to the exposure of a combination of chemicals. In all cases, the observed effects were of short duration.

In 2007, the EPA reviewed various databases such as the Office of Pesticides Program Incident Data System, Poison Control Center, California Department of Pesticide Regulation and the National Institute of Occupational Safety and Health's Sentinel Event Notification system for Occupational Risks and did not identify any human incidents involving mecoprop-p (EPA Reregistration Eligibility Decision (RED) for Mecoprop-p (MCP), August 29, 2007).

PCPA Hazard Characterization

For assessing risks from potential residues in food or from products used in or around residential areas or schools, the *Pest Control Products Act* (PCPA) requires the application of an additional 10-fold factor to threshold effects to take into account completeness of the data with respect to the exposure of, and toxicity to, infants and children and potential pre- and post-natal toxicity. A different factor may be determined to be appropriate on the basis of reliable scientific data.

With respect to the completeness of the toxicity database, extensive data are available for mecoprop-p and mecoprop racemate, including developmental toxicity studies in rats and rabbits and a reproductive toxicity study in rats.

With respect to identified concerns relevant to the assessment of risk to infants and children, no fetotoxicity was identified in the rat and rabbit developmental toxicity studies at or below maternally toxic doses. Fetotoxic effects were observed in rats at high dose levels and included increased post-implantation loss and delayed maturation. These effects were only seen at dose levels causing decreased body weight and food consumption in the dams. In the rat reproductive toxicity study with the racemate, adverse offspring effects involving the increase in pup deaths during lactation days zero to four and lower pup weight gain were identified. The effects occurred at a maternally non-toxic dose and pup mortality was considered a serious endpoint. The effects were considered to have arisen from prolonged repeated exposure. Therefore, the PCPA factor has been retained at 10-fold for repeat exposure scenarios when using the rat reproductive toxicity assay to establish the point of departure. In acute exposure scenarios, the risk was considered well characterized and there was no pre- or post-natal toxicity that could be attributed to a single dose. Accordingly, the PCPA factor was reduced to 1-fold.

Cancer Potency Factor

The carcinogenicity study of mecoprop-p in mice demonstrated a higher incidence of liver tumours in the female. The exact mode of action of carcinogenesis is not clearly determined. Therefore, linear low dose extrapolation (q_1^*) is recommended for cancer risk assessment ($q_1^* = 1.59 \times 10^{-3} \text{ (mg/kg bw/d)}^{-1}$).

Determination of Acute Reference Dose (ARfD)

General population:

The assessment of an acute reference dose for mecoprop-p for the general population is based on the NOAEL of 175 mg/kg bw established in the acute rat neurotoxicity study. At the LOAEL of 350 mg/kg bw, decreased body-weight gain and clinical signs were evident. Use of this study for ARfD determination is relevant because of the duration of exposure. The standard uncertainty factors (10-fold for interspecies extrapolation and 10-fold for intraspecies variability) have been applied. As previously discussed, the PCPA factor has been reduced to 1-fold. The composite assessment factor (CAF) is 100. The ARfD proposed is calculated according to the following formula:

$$\text{ARfD} = \frac{\text{NOAEL}}{\text{CAF}} = \frac{175 \text{ mg/kg bw}}{100} = 1.75 \text{ mg/kg bw}$$

The selected ARfD is also considered to be appropriate for the acute aggregate exposure and risk assessment for the general population.

Females aged 13-49:

To estimate acute dietary risk, the rat developmental toxicity study is selected for the appropriateness of exposure duration. At the LOAEL of 100 mg/kg bw/d, there were effects on the maternal animals affecting food intake and body-weight gains shortly after the initiation of

dosing. Pregnant animals appear to be more sensitive in terms of effects on body-weight gain. There was no indication of a similar sensitivity in the young. Based on the NOAEL of 50 mg/kg bw/d established in this study, and the CAF of 100-fold as discussed in the previous section (standard uncertainty factors of 10-fold for interspecies extrapolation and 10-fold for intraspecies variability, and the reduced PCPA of 1-fold), the proposed ARfD is calculated according to the following formula:

$$\text{ARfD (Females 13-49)} = \frac{50 \text{ mg/kg bw/d}}{100} = 0.5 \text{ mg/kg bw}$$

The selected ARfD is also considered to be appropriate for the acute aggregate exposure and risk assessment for females aged 13-49.

Determination of Acceptable Daily Intake (ADI)

All populations:

To estimate dietary risk from repeat exposure, the reproductive toxicity study of the mecoprop racemate in the rat is considered relevant for the duration of exposure as well as for the toxicity endpoints of concern. Offspring toxicity was demonstrated at the LOAEL 50 mg/kg bw/d. The effects included decreased body-weight gain and an increase in pup mortality during days one to four of the lactation period. The NOAEL for offspring toxicity established in this study is 10.3 mg/kg bw/d. For ADI determination, the standard uncertainty factors of 10-fold for interspecies extrapolation and 10-fold for intraspecies variability were applied. As discussed previously, the 10-fold PCPA factor has been retained. The CAF is therefore 1000. In the determination of the ADI for all populations, the identified concerns relevant to the assessment of risk to infants and children are taken into consideration.

The ADI proposed is calculated according to the following formula:

$$\text{ADI} = \frac{\text{NOAEL}}{\text{CAF}} = \frac{10.3 \text{ mg/kg bw/d}}{1000} = 0.01 \text{ mg/kg bw/d}$$

Maximum Residue Limit(s)

Residue data for mecoprop-p in cereal grains were submitted to support the conversion to full registration of this active on several end-use product labels. Residue data from field trials conducted in/on barley, corn and wheat were assessed in the framework of this application. In addition, a processing study in treated wheat was also assessed to determine the potential for concentration of residues of mecoprop-p into processed commodities.

Based on the maximum residues observed in crops treated according to label directions, maximum residue limits (MRLs) to cover residues of mecoprop-p in/on crops will be established as shown in Table 1. Residues in processed commodities not listed in Table 1 are covered under established MRLs for the raw agricultural commodities (RACs).

Table 1. Summary of Field Trial and Processing Data Used to Establish Maximum Residue Limit(s) (MRLs)

| Commodity | Application Method/ Total Application Rate | PHI (days) | Residues | | Experimental Processing Factor | Currently Established MRL | Recommended MRL |
|---------------|---|------------|---------------|-----|--------------------------------|---------------------------|---|
| | | | Min | Max | | | |
| Barley grain | Postemergence foliar application/ 1050 g a.e./ha | 53-79 | All <0.02 ppm | | No concentration observed | Under GMRL of 0.1 ppm | 0.02 ppm (for all crops of Crop Group 15; Cereal grain) |
| Corn (K+CWHR) | | 61-79 | | | | | |
| Corn grain | | 114-147 | | | | | |
| Wheat grain | | 58-104 | | | | | |

Based on the dietary burden and residue data, MRLs of 0.01 ppm in milk, 0.02 ppm in eggs, fat and meat of cattle, goats, hogs, horses, poultry and sheep and 0.05 ppm in meat by-products of cattle, goats, hogs, horses, poultry and sheep to cover residues of mecoprop will be established.

Following the review of all available data, MRLs for crops and livestock are recommended to cover residues of mecoprop-p. Residues in these crop/livestock commodities at the established MRLs will not pose an unacceptable risk to any segment of the population, including infants, children, adults and seniors.

Occupational and Residential Risk Assessment

Toxicological Endpoints

Non-dietary oral ingestion:

For children who might be exposed to mecoprop-p in treated lawns for 1-7 days through incidental ingestion, the NOAEL of 175 mg/kg bw established in the acute rat neurotoxicity study is used. Lower body-weight gain was identified at the LOAEL of 350 mg/kg bw/d. The standard uncertainty factors (10-fold for interspecies extrapolation and 10-fold for intraspecies variability) and the reduced PCPA factor of 1-fold as described previously provide a target margin of exposure (MOE) of 100.

Short-term dermal exposure:

Adequate 21-day dermal toxicity studies with mecoprop-p in the rat and rabbit are available. A NOAEL of 1000 mg/kg bw/d was established in both studies. Use of this endpoint is considered protective of all sub-populations, including nursing infants and unborn children of exposed female workers. The standard uncertainty factors (10-fold for interspecies extrapolation and 10-fold for intraspecies variability) and the reduced PCPA factor of 1-fold as described previously provide a target MOE of 100.

Short-term inhalation exposure:

No short-term inhalation toxicity data are available. The appropriate endpoint for short-term inhalation exposure for all sub-populations is determined by the NOAEL of 50 mg/kg bw/d established in the rat developmental toxicity study. Lower body-weight gain was identified at the LOAEL of 100 mg/kg bw/d. The standard uncertainty factors (10-fold for interspecies extrapolation and 10-fold for intraspecies variability) as described previously provide a target MOE of 100.

Intermediate- and long-term dermal and inhalation exposure:

To estimate dermal and inhalation risk from intermediate- and long-term repeat exposure, the reproductive toxicity study of the mecoprop racemate in the rat is considered relevant for the duration of exposure as well as for the toxicity endpoints of concern. Offspring toxicity was demonstrated at the LOAEL 50 mg/kg bw/d. The effects included decreased body-weight gain and increased pup mortality during days one to four of the lactation period. The NOAEL for offspring toxicity established in this study is 10.3 mg/kg bw/d. The standard uncertainty factors (10-fold for interspecies extrapolation and 10-fold for intraspecies variability) have been applied. The worker population could include females of child-bearing age (13-49 years old). For this reason, an additional factor of 10-fold has been applied to protect nursing infants and unborn children of exposed female workers. This additional factor was applied in consideration of the seriousness of the endpoint (i.e. pup mortality) in the absence of maternal toxicity. The target MOE is 1000.

Environmental Assessment

Fate and Behaviour in the Environment

Table 2 summarizes the fate and behaviour of mecoprop-p in the terrestrial environment.

Mecoprop-p has a water solubility >250 g/L at pH 7.0 and pH 10 indicating it is very soluble in soil water under environmental conditions. Mecoprop-p is not expected to bioaccumulate in terrestrial biota as the log K_{ow} is 0.48. The measured vapour pressure of mecoprop-p at 20°C (3.0×10^{-5} mm Hg) indicates it has low volatility and the calculated Henry's law constant of $1/H = 2.4 \times 10^6$ indicates mecoprop-p is not expected to volatilize from moist soil surfaces. The dissociation constant (pK_a) of 3.68 indicates mecoprop-p will exist as an anion at environmentally relevant pHs and, therefore, is expected to be mobile in soil under environmental conditions. Mecoprop-p does not absorb light at wavelengths greater than 300 nm and thus, is not expected to undergo direct phototransformation in the terrestrial environment.

Hydrolysis is not an important route of transformation as mecoprop-p is stable in water at pH 5, pH 7 and pH 9 after 31.3 days. On soil, phototransformation is not an important route of transformation as the first-order half-life of mecoprop-p is 32 days under artificial light. In aerobic soil, the DT_{50} of mecoprop-p is 6.1-11.7 days which indicates that mecoprop-p is non-persistent in aerobic soil. The major transformation product in the aerobic soil studies is CO_2 (>25% of applied radioactivity) with 4-chloro-2-methylphenol as a minor transient transformation product (<3%). Other minor transformation products were also observed, but never exceeded 3% of the applied radioactivity and were never identified. Biotransformation in anaerobic soils is not a route of transformation of mecoprop-p and it is persistent under these conditions.

Under laboratory conditions, mecoprop-p has medium to high mobility in soil on the basis of the adsorption K_{oc} values of 135-167. Under field conditions, mecoprop-p is non-persistent as the DT_{50} values are 2.3-4.3 days. Residues of mecoprop-p were detected to the 61.5-76.9 cm soil depth (after the second application) which indicated that residues are mobile in the soil profile.

Table 2 Fate and behaviour of mecoprop-p (MCP-P) in the terrestrial environment.

| Property | Test substance | Value / Observation | Comments |
|---|----------------|--|--|
| Physical and Chemical Properties | | | |
| Vapour pressure at 20°C | MCP-P | 3.0×10^{-5} mm Hg | Low volatility |
| Henry's law constant at 20°C | MCP-P | $1/H = 2.4 \times 10^6$ $K = 1.0 \times 10^{-8}$ atm · m ³ /mole | Not expected to volatilize from moist soil |
| Ultraviolet (UV) / visible spectrum | MCP-P | No absorption at wavelengths >300 nm | Not expected to undergo direct phototransform in the terrestrial environment |
| Solubility in water at 20°C (g/L) | MCP-P | pH 4: 6.65 pH 7: >250 pH 10: >250 | Very soluble in soil water at environmental pHs |
| n-Octanol/water partition coefficient (log K_{ow}) | MCP-P | 0.48 (pH 7, 25°C) | Not expected to bioaccumulate in terrestrial biota |
| Dissociation constant (p K_a) | MCP-P | 3.68 (20°C) | Exists as an anion at environmental pH, therefore, expected to be mobile in soil |
| Abiotic transformation | | | |
| Hydrolysis | MCP-P | Stable to hydrolysis at pH 5, pH 7 and pH 9 | Hydrolysis is not an important route of transformation |
| Phototransformation on soil | MCP-P | half-life = 32 days | Phototransformation is not an important route of transformation on soil under environmental conditions |
| Biotransformation | | | |
| Biotransformation in aerobic soil | MCP-P | $DT_{50} = 6.1-11.7$ days $DT_{90} = 20.3-38.8$ days | Non-persistent in aerobic soil |
| Biotransformation in anaerobic soil | MCP-P | No biotransformation | Persistent under anaerobic soil conditions as based on anaerobic biotransformation of the racemic mecoprop |
| Mobility | | | |
| Adsorption / desorption in soil | MCP-P | $K_{oc} = 135-167$ | Medium to high mobility in soil |
| Soil leaching | MCP-P | $R_f = 0.86$ | Mobile in soil |
| Field studies | | | |
| Field dissipation | MCP-P | $DT_{50} = 2.3-4.3$ days | Non-persistent in soil under field conditions |
| Field leaching | MCP-P | Residues leached to the 76.9 cm soil depth | Residues of MCP-P are expected to leach through the soil profile |

Table 3 summarizes the fate and behaviour of mecoprop-p (MCP-P) in the aquatic environment.

Mecoprop-p has a water solubility >250 g/L at pH 7.0 and pH 10 indicating it is very soluble in water under environmental conditions. Mecoprop-p is not expected to bioaccumulate in aquatic biota as the log K_{ow} is 0.48. The measured vapour pressure of mecoprop-p at 20°C (3.0×10^{-5} mm Hg) indicates it has low volatility and the calculated Henry's law constant of $1/H = 2.4 \times 10^6$ indicates mecoprop-p is not expected to volatilize from bodies of water. Mecoprop-p does not absorb light at wavelengths greater than 300 nm and, thus, is not expected to undergo direct phototransformation in the aquatic environment.

Hydrolysis is not an important route of transformation as mecoprop-p is stable in water at pH 5, pH 7 and pH 9 after 31.3 days. In water, phototransformation is not an important route of transformation as the first-order half-lives of mecoprop-p are 9.8 days (pH 5), 14.4 days (pH 7) and 13.8 days (pH 9). Under aerobic aquatic conditions, mecoprop-p is slightly persistent to moderately persistent as the DT_{50} s are 14.8-60.4 days. Under anaerobic aquatic conditions, mecoprop-p does not undergo biotransformation and, thus, is not a route of transformation. On the basis of its K_{oc} values of 135-167, mecoprop-p is not expected to partition into the sediment of aquatic systems.

Table 3 Fate and behaviour of mecoprop-p (MCP-P) in the aquatic environment.

| Study type | Test material | Value | Comments |
|---|---------------|--|--|
| Physical and Chemical Properties | | | |
| Vapour pressure at 20°C | MCP-P | 3.0×10^{-5} mm Hg | Low volatility |
| Henry's law constant at 20°C | MCP-P | $1/H = 2.4 \times 10^6$ $K = 1.0 \times 10^{-8}$ atm · m ³ /mole | Not expected to volatilize from bodies of water |
| Ultraviolet (UV) / visible spectrum | MCP-P | No absorption at wavelengths >300 nm | Not expected to phototransform directly in the aquatic environment |
| Solubility in water at 20°C (g/L) | MCP-P | pH 4: 6.65 pH 7: >250 pH 10: >250 | Very soluble in water at environmental pHs |
| n-Octanol/water partition coefficient (log K_{ow}) | MCP-P | 0.48 (pH 7, 25°C) | Not expected to bioaccumulate in aquatic biota |
| Abiotic transformation | | | |
| Hydrolysis | MCP-P | Stable to hydrolysis at pH 5, pH 7 and pH 9 | Hydrolysis is not an important route of transformation |
| Phototransformation in water | MCP-P | 9.8 days (pH 5) 14.4 days (pH 7) 13.8 days (pH 9) | Phototransformation is not an important route of transformation in the aquatic environment |
| Biotransformation | | | |
| Biotransformation in aerobic water systems | MCP-P | $DT_{50} = 14.8-60.4$ days | Slightly persistent to moderately persistent |
| Biotransformation in anaerobic water systems | MCP-P | No biotransformation | Not a route of transformation |

Monitoring Data

Mecoprop has been detected in Canadian surface waters and groundwater. For the ecoscenario exposure estimates, the acute concentration (2.9 µg/L) was estimated by determining the 95th percentile of the maximum concentration detected in each monitoring study/site whereas the chronic concentration (0.40 µg/L) was estimated by determining the 95th percentile of the arithmetic means of all samples at each site (detects and non-detects) from the monitoring studies (Appendix II).

Environmental Risk Characterization

The environmental risk assessment integrates the environmental exposure and ecotoxicology information to estimate the potential for adverse effects on non-target species. This integration is achieved by comparing exposure concentrations with concentrations at which adverse effects occur. Estimated environmental exposure concentrations (EECs) are concentrations of pesticide in various environmental media, such as food, water, soil and air. The EECs are estimated using standard models which take into consideration the application rate(s), chemical properties and environmental fate properties, including the dissipation of the pesticide between applications. Ecotoxicology information includes acute and chronic toxicity data for various organisms or groups of organisms from both terrestrial and aquatic habitats including invertebrates, vertebrates, and plants. Toxicity endpoints used in risk assessments may be adjusted to account for potential differences in species sensitivity as well as varying protection goals (i.e. protection at the community, population, or individual level).

The risk assessment first utilizes a deterministic evaluation that integrates the environmental exposure represented by the EEC and the environmental toxicity as represented by the most sensitive test species to determine the likelihood of adverse ecological effects. One method of achieving this integration is through the estimation of a *Risk Quotient* (RQ). The RQ is usually calculated by comparing a threshold toxicity endpoint, usually a LC₅₀, LD₅₀, EC₅₀, EC₂₅, NOEC or NOEL for the most-sensitive test species, to an EEC based on the maximum cumulative application rate. The mathematical relationship among RQ, toxicity endpoint and the EEC is:

$$RQ = EEC \div \text{toxicity endpoint}$$

In addition, uncertainty factors are applied to the acute toxicity endpoints to account for interspecies variability. For fish and amphibians, the LC₅₀ is divided by an uncertainty factor of ten. For terrestrial and aquatic invertebrates, algae and aquatic vascular plants, the LC₅₀ or EC₅₀ is divided by an uncertainty factor of two.

For describing the risk associated with the RQ, the Level of Concern (LOC) is considered. The LOC is equal to a RQ of 1.0 and functions as the cut-off criteria for estimating risk. Thus, if the LOC is exceeded (RQ>1) then, a concern is identified. For RQ < 1.0, there is a negligible risk as the LOC is not exceeded. In cases where the LOC is exceeded, a refined assessment is conducted in which the risk is based on exposure to mecoprop-p through spray drift and surface runoff.

Risks to Terrestrial Organisms

Table 4 summarizes the risks to terrestrial organisms resulting from the application of mecoprop-p. The effect endpoints considered in the risk assessment for terrestrial organisms are summarized in Table 5 of Appendix 1.

For earthworms and bees, mecoprop-p is not expected to pose an appreciable risk ($RQ < 1$) under field conditions.

For wild birds feeding on contaminated food on-field, mecoprop-p poses an acute oral risk ($RQs = 1.1-3.1$), an acute dietary risk ($RQs = 1.2-9.7$) and a risk to reproduction ($RQ = 1.3$) in some feeding guilds. For wild birds feeding on contaminated food off-field, mecoprop-p poses an acute dietary risk ($RQ = 1.2$) to one feeding guild. Although in these cases the LOC is exceeded ($RQ > 1$) in birds feeding either on-field or off-field, the risk is based on the assumption that the bird's diet consists entirely of mecoprop-p-contaminated food, as expressed by the estimated daily exposure values. Under field conditions, however, it is not expected that birds would be feeding exclusively in fields treated with mecoprop-p or entirely in areas bordering these treated fields. In addition, the risk assessment does not consider feeding preference or avoidance behaviour toward contaminated food as these data are not available. On this basis, the risk to birds may be overestimated for field conditions.

For small mammals feeding on contaminated food on-field, mecoprop-p poses an acute oral risk ($RQs = 1.3-4.5$), a subchronic dietary risk ($RQs = 1.1-24.7$) and a risk to reproduction ($RQs = 1.2-7.6$) in some feeding guilds. For small mammals feeding on contaminated food off-field, mecoprop-p poses a subchronic dietary risk ($RQs = 1.1-3.0$) to some feeding guilds. Although in these cases the LOC is exceeded ($RQ > 1$) in small mammals feeding either on-field or off-field, the risk is based on the assumption that the animal's diet consists entirely of mecoprop-p-contaminated food (as expressed by the EDE values). Under field conditions, however, it is not expected that small mammals would be feeding exclusively in fields treated with mecoprop-p or entirely in areas bordering these treated fields. In addition, the risk assessment does not consider feeding preference or avoidance behaviour toward contaminated food as these data are not available. On this basis, the risk to small mammals may be overestimated for field conditions.

The greatest risk was shown in non-target terrestrial plants ($RQ = 9-35$) in areas bordering treated fields where exposure to mecoprop-p results from off-target spray drift.

Table 4 Summary of the risk to terrestrial organisms.

| Organism | Exposure | Endpoint value | RQ | LOC Exceeded |
|----------------------|---------------|-----------------------------------|----------------|--------------|
| Invertebrates | | | | |
| Earthworm | Acute | 247 mg a.e./kg soil | 0.001 0.002 | No No |
| Bee | Oral | LD ₅₀ = 5.6 kg a.e./ha | 0.19 0.24 | No No |
| | Contact | LD ₅₀ = 56 kg a.e./ha | 0.019 0.024 | No No |
| Birds | | | | |
| Bobwhite quail | Acute | 46 mg a.e./kg bw/d | 1.1-3.1 | Yes |
| Mallard duck | Acute Dietary | 14.6 mg a.e./kg bw/d | 1.2-9.7 | Yes |

| Organism | Exposure | Endpoint value | RQ | LOC Exceeded |
|------------------------|--------------------|--|----------|--------------|
| Japanese quail | Reproduction | 111.2mg a.e./kg bw/d | 1.3 | Yes |
| Mammals | | | | |
| Rat | Acute | 70 mg a.e./kg bw/d | 1.3-4.5 | Yes |
| Mouse | Subchronic Dietary | 12.7 mg a.e./kg bw/d | 1.1-24.7 | Yes |
| Rat | Reproduction | 41.4 mg a.e./kg bw/d | 1.2-7.6 | Yes |
| Vascular plants | | | | |
| Vascular plants | Vegetative vigour | 0.0069 kg a.e./ha (HD ₅ of EC ₅₀ S) | 9-35 | Yes |

Risks to Aquatic Organisms

Table 5 summarizes the risks to aquatic organisms resulting from the application of mecoprop-p. The endpoints considered in the risk assessment for aquatic organisms are summarized in Table 6 of Appendix 1.

In freshwater invertebrates, fish, amphibians and algae, there were no appreciable risks (RQs < 1) associated with the application of mecoprop-p.

In marine/estuarine plants, mecoprop-p poses a risk as the RQs were 1.4-3.5 and hence, exceeds the LOC.

Table 5 Summary of Risk to Aquatic Organisms.

| Species | Exposure | Endpoint value (mg a.e./L) | RQ | LOC Exceeded |
|---|----------|-------------------------------|---------|--------------|
| Freshwater Invertebrate (<i>Daphnia magna</i>) | Acute | 41 | <1 | No |
| Freshwater Invertebrate (<i>Daphnia magna</i>) | Chronic | 11.1 | <1 | No |
| Bluegill Sunfish (<i>Lepomis macrochirus</i>) | Acute | 9.1 | <1 | No |
| Rainbow Trout (<i>Oncorhynchus mykiss</i>) | Chronic | 49.6 | <1 | No |
| Amphibians | Acute | 9.1 | <1 | No |
| Freshwater Algae (<i>Navicula pelliculosa</i>) | Acute | 0.12 | <1 | No |
| Marine algae (<i>Skeletonema costatum</i>) | Acute | 0.0085 | 1.4-3.5 | Yes |

Toxic Substances Management Policy Considerations

The Toxic Substances Management Policy (TSMP) is a federal government policy developed to provide direction on the management of substances of concern that are released into the environment. The TSMP calls for the virtual elimination of Track 1 substances [those that meet all four criteria outlined in the policy, i.e., persistent (in air, soil, water and/or sediment), bio-accumulative, primarily a result of human activity and toxic as defined by the *Canadian Environmental Protection Act*].

During the review process, mecoprop-p and its transformation products were assessed in accordance with the PMRA Regulatory Directive DIR99-03¹ *The Pest Management Regulatory Agency's Strategy for Implementing the Toxic Substances Management Policy*. Substances associated with the use of mecoprop-p were also considered, including transformation products formed in the environment, and contaminants and formulants in the technical product and the end-use product. Mecoprop-p and its transformation products were evaluated against the following Track 1 criteria: persistence in soil ≥ 182 days; persistence in water ≥ 182 days; persistence in sediment ≥ 365 days; persistence in air ≥ 2 days; bioaccumulation $\log K_{ow} \geq 5$ or BCF ≥ 5000 (or BAF ≥ 5000). In order for mecoprop-p or its transformation products to meet Track 1 criteria, the criteria for both bioaccumulation and persistence (in one media) must be met. The technical product and end-use product, including formulants, were assessed against the contaminants identified in the *Canada Gazette*, Part II, Volume 139, Number 24, pages 2641–2643: *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern, Part 3 Contaminants of Health or Environmental Concern*. The PMRA has reached the following conclusions (Table 4 of Appendix I summarizes the comparison to TSMP Track 1 criteria):

- Mecoprop-p does not meet Track 1 criteria.
- Mecoprop-p does not meet the Track 1 criterion for persistence because the half-life values in soil (6.1-11.7 days) and water (14.8-48.5 days), do not exceed the Track 1 criterion for soil and water.
- Mecoprop-p does not meet the Track 1 criterion for bioaccumulation, as its octanol-water partition coefficient ($\log K_{ow} = 0.48$) is below the Track 1 criterion.
- Mecoprop-p (technical grade) does not contain any by-products or microcontaminants that meet the TSMP Track 1 criteria. Impurities of toxicological concern are not expected to be present in the raw materials nor are they expected to be generated during the manufacturing process.

Formulants and Contaminants of Health or Environmental Concern

During the review process, contaminants in the technical and formulants and contaminants in the end-use products are compared against the *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern* maintained in the *Canada Gazette*². The list is used as described in the PMRA Notice of Intent NOI2005-01³ and is based on existing policies

¹ DIR99-03, *The Pest Management Regulatory Agency's Strategy for Implementing the Toxic Substances Management Policy*

² *Canada Gazette*, Part II, Volume 139, Number 24, SI/2005-114 (2005-11-30) pages 2641–2643: *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern* and in the order amending this list in the *Canada Gazette*, Part II, Volume 142, Number 13, SI/2008-67 (2008-06-25) pages 1611-1613. *Part 1 Formulants of Health or Environmental Concern, Part 2 Formulants of Health or Environmental Concern that are Allergens Known to Cause Anaphylactic-Type Reactions and Part 3 Contaminants of Health or Environmental Concern*.

³ NOI2005-01, *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern under the New Pest Control Products Act*.

and regulations including: DIR99-03; and DIR2006-02⁴, and taking into consideration the Ozone-depleting Substance Regulations, 1998, of the *Canadian Environmental Protection Act* (substances designated under the Montreal Protocol). The PMRA has reached the following conclusions:

Technical grade mecoprop-p and the end-use product A.H. MARKS MCPPP-p 600 Liquid Herbicide do not contain any formulants or contaminants of health or environmental concern identified in the *Canada Gazette*, Part II, Volume 139, Number 24, pages 2641–2643: *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern*.

Value Assessment

A value assessment was not required for this application.

Conclusion

The PMRA has assessed all available information and is able to support the conversion of Marks Mecoprop-p Technical Acid (Registration Number 27441) to full registration.

⁴ DIR2006-02, PMRA Formulants Policy.

List of Abbreviations

| | |
|------------------|--|
| 1/H | Henry's Law Constant |
| µg | micrograms |
| a.e. | acid equivalent |
| ADI | acceptable daily intake |
| AR | administered radioactivity |
| ARfD | acute reference dose |
| atm | atmosphere |
| BAF | Bioaccumulation Factor |
| BCF | Bioconcentration Factor |
| bw | Body weight |
| CAF | composite assessment factor |
| cm | centimetres |
| CO ₂ | Carbon dioxide |
| d | day |
| DMA | dimethylamine |
| DMAS | dimethylamine salt |
| DNA | deoxyribonucleic acid |
| DT ₅₀ | dissipation time 50% (the time required to observe a 50% decline in concentration) |
| DT ₉₀ | dissipation time 90% (the time required to observe a 90% decline in concentration) |
| EC ₂₅ | effective concentration on 25% of the population |
| EC ₅₀ | effective concentration on 50% of the population |
| EDE | estimated daily exposure |
| EEC | estimated environmental exposure concentration |
| EHE | 2-ethylhexylester |
| EPA | United States Environmental Protection Agency |
| g | gram(s) |
| h | hour(s) |
| ha | hectare(s) |
| HD ₅ | hazard dose 5% |
| HDT | highest dose tested |
| Hg | mercury |
| HGPRT | hypoxanthine-guanine phosphoribosyl transferase |
| kg | kilogram(s) |
| K _{oc} | organic-carbon partition coefficient |
| K _{ow} | octanol-water partition coefficient |
| L | litre(s) |
| LC ₅₀ | lethal concentration to 50% |
| LD ₅₀ | lethal dose to 50% |
| LOAEL | lowest observed adverse effect level |
| LOC | level of concern |
| LOD | limit of detection |
| m ³ | metre(s) cubed |
| MCPP | mecoprop |
| MCPP-P | mecoprop-P |

| | |
|-------------------------|-------------------------------------|
| MAS | maximum average score |
| mg | milligram(s) |
| MIS | maximum irritation score |
| mL | millilitre(s) |
| mm | millimetre(s) |
| MOE | margin of exposure |
| MRL | maximum residue limits |
| MTD | maximum tolerated dose |
| NA | not applicable |
| ND | not determined |
| nm | nanometres |
| NOAEL | no observed adverse effect level |
| NOEC | no observed effect concentration |
| NOEL | no observed effect level |
| NZW | New Zealand white |
| PCPA | <i>Pest Control Product Act</i> |
| PHI | post harvest interval |
| p <i>K</i> _a | dissociation constant |
| PMRA | Pest Management Regulatory Agency |
| ppm | parts per million |
| q ₁ | linear low dose extrapolation |
| RAC | raw agricultural commodity |
| RED | Reregistration Eligibility Decision |
| R _f | retention factor |
| RQ | risk quotient |
| TSMP | Toxic Substances Management Policy |
| US | United States |
| UV | ultraviolet |

Appendix I Tables and Figures

Table 1 Acute Toxicity of mecoprop-p

| ACUTE STUDIES - TECHNICAL (mecoprop-p) | | | | |
|---|---|---|--|------------------|
| Study type | Species, strain (test compound) | Results | Comments | Reference |
| Oral | rat, Wistar (mecoprop D-form) | LD ₅₀ ♂ = 1327; ♀ >681<1000 ♂♀ = 1050 mg/kg bw | Moderate toxicity WARNING - POISON | 1097063 |
| | Rat, Sprague-Dawley (mecoprop-p) | LD ₅₀ : ♂+♀ = 431 mg/kg bw | Highly toxic DANGER - POISON | |
| | Rat, Sprague-Dawley (mecoprop-p) | LD ₅₀ : ♂ = 803 (710-698) ♀ = 756 (651-861) ♂+♀ = 775 (666-885) mg/kg bw | Moderate toxicity WARNING - POISON | |
| | Rat, Sprague-Dawley (mecoprop-p DMA) | LD ₅₀ : ♂ = 834 (657-1064) ♀ = 696 (542-891) ♂+♀ = 763 (642-907) mg/kg bw | Moderate toxicity WARNING - POISON | |
| | Rat, Sprague-Dawley (mecoprop-p DMA) | LD ₅₀ : ♂ = ~ 903 ♀ = ~ 903 ♂+♀ = >681 <1000 mg/kg bw | Moderate toxicity WARNING - POISON | |
| | Rat, Sprague-Dawley (mecoprop-p EHE) | LD ₅₀ : ♂ = 1300 (1100-1600) ♀ = 1400 (1200-1900) ♂+♀ = 1400 (1200-1600) mg/kg bw | Slight toxicity | |
| Dermal | rat, Wistar (mecoprop D-form) | LD ₅₀ >4000 mg/kg bw | Low toxicity | 1190440 |
| | Rat, CD (mecoprop-p) | LD ₅₀ >2000 mg/kg bw | Low toxicity | |
| | Rat, Sprague-Dawley (mecoprop-p) | LD ₅₀ >2000 mg/kg bw | Low toxicity | |
| | Rat, Wistar (mecoprop-p DMA) | LD ₅₀ >4000 mg/kg bw | Low toxicity | |
| | Rabbit, NZW (mecoprop-p DMA) | LD ₅₀ >4000 mg/kg bw | Low toxicity | |
| | Rat, NZW (mecoprop-p EHE) | LD ₅₀ >2000 mg/kg bw | Low toxicity | |
| Intraperitoneal | Rat, Wistar (mecoprop D-form) | LD ₅₀ : ♂ >316 <464; ♀ = 383 ♂+♀ = 383 mg/kg bw | Not applicable | |
| Inhalation (4 h nose/head only) | Rat, Wistar (mecoprop D-form) | LC ₅₀ >5.6 mg/L (actual) | Low toxicity | |
| | rat, Sprague-Dawley (mecoprop-p) | LC ₅₀ >0.87 mg/L (actual) | Low toxicity | |
| | Rat, Wistar (mecoprop-p DMA) | LC ₅₀ ♂ = ~3.6; ♀ = 5.24~ ♂+♀ = 4.71 (3.89-6.45) mg/mL | Low toxicity | |
| Inhalation (4 h whole-body) | rat, Sprague-Dawley (mecoprop-p DMA) | LC ₅₀ >4.68 mg/L (actual) | Low toxicity | |
| | rat, Sprague-Dawley (mecoprop-p EHE) | LC ₅₀ >4.66 mg/L (actual) | Low toxicity | |
| Eye irritation | rabbit, New Zealand white (NZW) (mecoprop D-form) | MAS = 70/110 MIS at 1, 24, 48, 72 h = 38, 62, 66, 89, respectively | Severely irritating DANGER CORROSIVE TO EYES | |
| | rabbit, NZW (mecoprop-p) | MIS at 1, 24, 48, 72 h = 48, 48, 50, 61, respectively | Severely irritating DANGER – EYE IRRITANT | |
| | rabbit, white Vienna (mecoprop-p DMA) | | Severely irritating DANGER – EYE IRRITANT | |

| ACUTE STUDIES - TECHNICAL (mecoprop-p) | | | | |
|---|--|---|--|------------------|
| Study type | Species, strain (test compound) | Results | Comments | Reference |
| Skin irritation | rabbit, NZW (mecoprop D-form) | MIS: at 1, 24, 48 h, and days 7 and 14 after patch removal were 3.2, 2.3, 2.3, 1.0, 0, respectively | Moderately irritating (exposure period was prolonged for 24 h instead of the guideline requirement of 4 h) | |
| | rabbit, NZW (mecoprop-p) | MIS: 0 | Non-irritating | |
| | rabbit, NZW (mecoprop-p) | MIS: at 1, 24, 48, 72 h, and days 7, 10, 13 = 0.8, 0.8, 0.7, 0.5, 0.3, 0.2, 0 (maximum = 8), respectively | Slightly irritating | |
| | rabbit, white Vienna (mecoprop-p DMA) | MIS: at 4, 24, 48, 72 h = 1.67, 0, 0, and 0 (maximum = 8), respectively | Mildly irritating CAUTION - SKIN IRRITANT | |
| | rabbit, NZW (mecoprop-p DMA) | MAS = 0.5/8 MIS: at days 1, 2, 3, 4 after dosing were 0.5, 0.5, 0.33, and 0 (maximum = 8), respectively | Slightly irritating | |
| Skin sensitization (Open epicutaneous test) | guinea pig, Pirbright White Dunkin Hartley (mecoprop racemate) | Negative | (Deficient - no positive control data) | |
| Skin sensitization (maximization) | guinea pig, Pirbright White Dunkin Hartley (mecoprop D-form) | Negative | Not a skin sensitizer | |
| | guinea pig, Pirbright White Dunkin Hartley (mecoprop-p) | Negative | Not a skin sensitizer | 1185283 |
| | guinea pig, Pirbright White Dunkin Hartley (mecoprop-p DMA) | Negative | Not a skin sensitizer | |
| Skin sensitization (Buehler) | guinea pig, albino (mecoprop-p) | Negative | Not a skin sensitizer | |
| | guinea pig, Dunkin Hartley (mecoprop-p DMA) | Positive | Potential skin sensitizer | |
| | guinea pig, Dunkin Hartley (mecoprop-p EHE) | Positive after 1 st challenge Negative after 2 nd challenge Results difficulty to interpret | Equivocal | |
| ^a MIS = maximum irritation score; ^b MAS = maximum average score for 24, 48 and 72 hours | | | | |

Table 2 Short- and Long-term Toxicity Profile of Technical Mecoprop-p and Mecoprop Racemate

| SHORT-TERM TOXICITY [mecoprop-p (D-form of mecoprop), mecoprop racemate] | | | |
|---|---|---|------------------|
| Study type | Species, strain / test compound / dose levels | Results and comments | Reference |
| 4-week dietary | mouse, B6C3F1 / mecoprop racemate 0, 100, 300, 900, 2700 ppm ♂: 0, 20, 63, 181, 614; ♀: 0, 31, 79, 267, 951 mg/kg bw/d | NOAEL ♂+♀ = 900 ppm; ♂ = 181; ♀ = 267 mg/kg bw/d LOAEL ♂+♀ = 2700 ppm; ♂ = 614; ♀ = 951 mg/kg bw/d (liver pathology) | |
| | mouse, B6C3F1 / mecoprop racemate 0, 2700, 4500, 7000 ppm ♂: 0, 764, 1800, 3913; ♀: 0, 1009, 2101, 4308 mg/kg bw/d | NOAEL not established LOAEL ♂+♀ = 2700 ppm ♂ = 764; ♀ = 1009 mg/kg bw/d (liver pathology) | |
| 7-week dietary | Rat, Sprague-Dawley / mecoprop racemate and D-form racemate: 0, 50, 400 ppm ♂: 0, 4.41, 30.1; ♀: 0, 4.77, 37.5 mg/kg bw/d | NOAEL = 400 ppm (HDT) racemate: ♂ = 30.1, ♀ = 37.5 mg/kg bw/d D-form: ♂ = 35.2, ♀ = 38.0 mg/kg bw/d | |

| SHORT-TERM TOXICITY [mecoprop-p (D-form of mecoprop), mecoprop racemate] | | | |
|---|--|--|--|
| Study type | Species, strain / test compound / dose levels | Results and comments | Reference |
| | D-isomer: 0, 50, 400 ppm ♂: 0, 4.41, 35.2; ♀: 0, 4.76, 38.0 mg/kg bw/d | | |
| 90-day dietary | mouse; B6C3F1 / mecoprop-p 0, 100, 1000, 2500 ppm ♂: 0, 20, 224, 739; ♀: 0, 30, 330, 925 mg/kg bw/d | NOAEL ♂+♀ = 100 ppm; ♂ = 20, ♀ = 30 mg/kg bw/d LOAEL ♂ +♀= 1000 ppm; ♂ = 224, ♀ = 330 mg/kg bw/d (liver and kidney pathology) | 1185284 |
| | Rat, Wistar / mecoprop racemate 0, 50, 150, 450 ppm ♂: 0, 3.8, 11.3, 33.7; ♀: 0, 4.4, 13.3, 38.9 mg/kg bw/d | NOAEL > 450 ppm; ♂ = 33.7, ♀ = 38.9 mg/kg bw/d (HDT) MTD not reached | |
| | Rat, Wistar / mecoprop racemate and mecoprop D-form racemate: 0, 200, 800, 3200 ppm ♂: 0, 16.5, 68, 391; ♀: 0, 18.2, 76, 399 mg/kg bw/d D-isomer: 0, 200, 400, 800, 1600, 3200 ppm ♂: 0, 15.6, 32, 68, 146, 403 ♀: 0, 18.4, 38, 76, 170, 404 mg/kg bw/d | NOAELs - 800 ppm racemate: ♂ = 68; ♀ = 76 mg/kg bw/d D-isomer: ♂ = 68; ♀ = 76 mg/kg bw/d LOAELs – racemate: 3200 ppm; ♂ = 391; ♀ = 398 mg/kg bw/d (bw) D-isomer: 1600 ppm; ♂ = 146; ♀ = 170 mg/kg bw/d (bw) | |
| 90-day dietary / neurotoxicity | rat, Wistar / mecoprop-p 0, 75, 500, 2500 (♂), 3000 (♀) ppm ♂ = 0, 5.4, 35.2, 189; ♀ = 0, 6.0, 41.1, 240 mg/kg bw/d | NOAEL = 500 ppm; ♂ = 35; ♀ = 41 mg/kg bw/d LOAEL ♂ = 2500 ppm; 189 mg/kg bw/d ♀ = 3000 ppm; 240 mg/kg bw/d (pathology of liver and adrenal) | 1185225 |
| 1-year dietary | dog, beagle / mecoprop-p 0, 60, 180, 600 ppm ♂ = 0, 1.8, 5.2, 18.3; ♀ = 0, 2, 5.7, 19 mg/kg bw/d | NOAEL = 180 ppm; ♂ = 5.2; ♀ = 5.7 mg/kg bw/d LOAEL = 600 ppm; ♂ = 18.3; ♀ = 19 mg/kg bw/d (bw) | 1185285 |
| 21-day dermal | rabbit, NZW / mecoprop-p 0, 10, 100, 1000 mg/kg bw | NOAEL: 1000 mg/kg bw/d | |
| | rat, Wistar / mecoprop-p 0, 12, 120, 1000 mg/kg bw | NOAEL: 1000 mg/kg bw/d | |
| CHRONIC TOXICITY AND ONCOGENICITY | | | |
| 18-month dietary oncogenicity | Mouse, B6C3F1/CrlBR / mecoprop-p 0, 25, 250, 2500 ppm ♂ = 0, 4, 40, 592; ♀ = 0, 4, 46, 732 mg/kg bw/d | NOAEL: ♂ = 40 mg/kg bw/d (HDT); ♀ = 4 mg/kg bw/d LOAEL: ♂ = not established; ♀ = 46 mg/kg bw/d (kidney pathology) Oncogenic potential not adequately assessed | 1185226 |
| | Mouse, B6C3F1/CrlBR / mecoprop-p 0, 700 (♂), 800 (♀) ppm ♂ = 0, 122; ♀ = 0, 209 mg/kg bw/d | NOAEL: not established higher incidence of liver adenoma and carcinoma in ♀ | |
| 2-year dietary/ oncogenicity | rat, Wistar / mecoprop racemate 0, 20, 100, 400 ppm ♂ = 0, 1.1, 5.5, 22.2; ♀ = 0, 1.4, 6.9, 27.9 mg/kg bw/d | No systemic toxicity; inadequate dose levels tested (MTD not reached) Oncogenicity: no effects at dose levels tested | 1185227 1185228 1185324 1185348 |
| | rat, Wistar / mecoprop-p 0, 100, 600, 1200 ppm ♂ = 0, 5.3, 32.0, 64.6; ♀ = 0, 6.6, 39.9, 81.7 mg/kg bw/d | NOAEL: ♂ = 600 ppm, or 32 mg/kg bw/d ♀ = 100 ppm, or 6.6 mg/kg bw/d LOAEL: ♂ = 1200 ppm, or 64.6 mg/kg bw/d (bw) ♀ = 600 ppm, or 40 mg/kg bw/d (bw) no evidence of oncogenicity | |
| REPRODUCTION AND DEVELOPMENTAL TOXICITY | | | |
| 2-generation reproductive toxicity | rat, Wistar / mecoprop racemate 0, 20, 100, 500 ppm | Parental systemic toxicity NOAEL > 500 ppm, or ♂ >47.3; ♀ >50.7 mg/kg bw/d Reproductive toxicity: NOAEL = 500 ppm, or 47.3 mg/kg bw/d Offspring toxicity: NOAEL = 100 ppm / 9.3 mg/kg bw/d LOAEL = 500 ppm, or 47.3 mg/kg bw/d (higher pup mortality during lactation days 0-4; but birth, viability, and lactation indices not affected) | 1185229 1382211 |

| SHORT-TERM TOXICITY [mecoprop-p (D-form of mecoprop), mecoprop racemate] | | | |
|---|--|--|-------------------------------|
| Study type | Species, strain / test compound / dose levels | Results and comments | Reference |
| Developmental toxicity | rat, Sprague-Dawley / mecoprop racemate 0, 20, 50, 125 mg/kg bw/d | NOAEL, maternal toxicity = 20 mg/kg bw/d developmental toxicity = 50 mg/kg bw/d LOAEL, maternal toxicity = 50 (bw, food) developmental toxicity = 125 mg/kg bw/d No evidence of teratogenicity | |
| | Rat, Wistar / mecoprop-p 0, 20, 50, 100 mg/kg bw/d | NOAEL, maternal and developmental toxicity = 50 mg/kg bw/d LOAEL, maternal and developmental toxicity = 100 mg/kg bw/d No evidence of teratogenicity | 1185230 |
| | rabbit, Himalayan / mecoprop-p 0, 5, 20, 50 mg/kg bw/d | NOAEL, maternal and developmental toxicity = 50 mg/kg bw/d No evidence of teratogenicity | 1185323 1185349 1185350 |
| SPECIAL TOXICITY AND METABOLISM/TOXICOKINETICS | | | |
| acute neurotoxicity | rat, Wistar / mecoprop-p 0, 175, 350, 700 mg/kg bw | Acute systemic toxicity: NOAEL = 175 mg/kg bw; LOAEL = 350 mg/kg bw (bw, clinical signs) No evidence of acute neurotoxicity | 1185286 |
| 90-day neurotoxicity | | See short-term study data | |
| Metabolism | Rat, Wistar / mecoprop-p | Absorption: rapid and extensive; plasma peak concentrations seen 2-4 h post-dosing. Distribution: tissue distribution was extensive, but the level declined rapidly and no evidence of bioaccumulation. Excretion: rapid; mainly in the urine; fecal elimination low; not eliminated in expired air. Metabolism: minimal; unchanged parent compound the main compound excreted. | 1185287 |
| GENOTOXICITY | | | |
| Study | Species and strain or cell type / Test compound / Concentrations or doses | Results | Reference |
| Gene mutations in bacteria <i>in vitro</i> | <i>Salmonella typhimurium</i> strains TA 98, TA 100, TA 1535, TA 1537, TA 1538 / mecoprop racemate | negative | |
| | <i>Salmonella typhimurium</i> strains TA 98, TA 100, TA 1535, TA 1537 / mecoprop-p | negative | 1185335 |
| | <i>Salmonella typhimurium</i> strains TA 98, TA 100, TA 1535 and TA 1537 / mecoprop-p | negative | |
| | <i>Salmonella typhimurium</i> strains TA 98, TA 100, TA 1535 and TA 1537 / mecoprop-p DMA | negative | |
| | <i>Salmonella typhimurium</i> strains TA 98, TA 100, TA 1535 and TA 1537 / mecoprop-p DMA | negative | |
| Gene mutations in mammalian cells <i>in vitro</i> | Chinese hamster ovary cells (HGPRT locus) / mecoprop-p | negative | 1185344 |
| | Chinese hamster ovary cells (HGPRT locus) / mecoprop-p DMA | negative | |
| | Chinese hamster ovary cells (HGPRT locus) / mecoprop-p EHE | negative | |
| Chromosome aberrations <i>in vitro</i> | human blood lymphocytes / mecoprop-p | positive at cytotoxic concentrations ≥ 300 $\mu\text{g/mL}$ without S9 activation in assay 1 negative all concentration levels with (assay 1 and 2) or without S9 (assay 1) clastogenic at cytotoxic doses +S9 | 1185345 1185346 |
| | human blood lymphocytes / mecoprop-p | positive at cytotoxic doses with S9 activation | |
| | human blood lymphocytes / mecoprop-p DMA | clastogenic at higher doses with S9 activation | |

| SHORT-TERM TOXICITY [mecoprop-p (D-form of mecoprop), mecoprop racemate] | | | |
|---|--|---|------------------|
| Study type | Species, strain / test compound / dose levels | Results and comments | Reference |
| | human blood lymphocytes / mecoprop-p EHE | clastogenic at high dose with S9 activation | |
| <i>In vivo</i> chromosome aberration in | Chinese hamster bone marrow cells / mecoprop D-form | negative | |
| | Chinese hamster bone marrow cells / mecoprop racemate | Positive at the high dose | |
| <i>In vitro/in vivo</i> unscheduled DNA synthesis | rat, Wistar / mecoprop-p 0, 50, 200, 500, 500 mg/kg bw | negative | 1185347 |
| <i>In vivo</i> sister chromatid exchange | Chinese hamster (bone marrow cells) / mecoprop racemate | negative | |
| <i>In vivo</i> mouse micronucleus assay | mouse, CD-1 / mecoprop-p 24 h: 0, 20, 100, 500 mg/kg bw; 48 & 72 h: 0, 2600 mg/kg bw | negative | |
| | mouse, CD-1 / mecoprop-p DMA 0, 144, 288, 576 mg/kg bw | negative | |
| | mouse, CD-1 / mecoprop-p EHE 0, 782, 1564, 3128 mg/kg bw | negative | |

Table 3 Toxicology Endpoints for Use in Health Risk Assessment for Mecoprop-p

| Exposure scenario | NOAEL, mg/kg bw/d | Study | Endpoint | CAF | ARfD / ADI / target MOE |
|---|--------------------------|---------------------------|---|------------|--------------------------------|
| Acute dietary (ARfD), general population | 175 | Acute rat neurotoxicity | Body weight, clinical signs, motor activity | 100-fold | 1.75 |
| Acute dietary (ARfD), ♀ 13-49 | 50 | Rat developmental | Body weight | 100-fold | 0.5 |
| Chronic dietary (ADI), all population | 10.3 | Rat reproductive toxicity | Offspring toxicity | 100-fold | 0.01 |
| Incidental ingestion, children | 50 | Rat developmental | Body weight | | 100 |
| Acute 1-7 day dermal | 1000 | 21-day rabbit dermal | No toxic effects | | 100 |
| Acute 1-7 day inhalation | 50 | Rat developmental | Body weight | | 100 |
| Acute aggregate oral/inhalation | 50 | Rat developmental | Body weight | | 100 |
| Acute aggregate dermal | 1000 | 21-day rabbit dermal | No toxic effects | | 100 |
| Short-term dermal | 1000 | 21-day rabbit dermal | No toxic effects | | 100 |
| Short-term inhalation | 50 | Rat developmental | Body weight | | 100 |
| Intermediate- and long-term dermal and inhalation | 10.3 | Rat reproductive toxicity | Offspring toxicity | | 1000* |
| CAF = standard 100-fold (10-fold for interspecies extrapolation and 10-fold for intraspecies variability) X reduced PCPA factor of 1-fold | | | | | |
| * the 10-fold PCPA factor retained | | | | | |

Table 4 Toxic Substances Management Policy Considerations-Comparison to TSMP Track 1 Criteria

| TSMP Track 1 Criteria | TSMP Track 1 Criterion value | | Active Ingredient Endpoints | Transformation Products Endpoints |
|---|------------------------------|--|---|--|
| Toxic or toxic equivalent as defined by the <i>Canadian Environmental Protection Act</i> ¹ | Yes | | Yes | CO ₂ was the major transformation product in aerobic soil; 4-chloro-o-cresol (4-CC) was a minor transformation product that was short-lived in the upper soil strata. |
| Predominantly anthropogenic ² | Yes | | Yes | |
| Persistence ³ : | Soil | Half-life ≥ 182 days | Half-life = 6.1-11.7 days | |
| | Water | Half-life ≥ 182 days | Half-life = 14.8-48.5 days | |
| | Sediment | Half-life ≥ 365 days | ND | |
| | Air | Half-life ≥ 2 days or evidence of long range transport | Half-life or volatilisation is not an important route of dissipation and long-range atmospheric transport is unlikely to occur based on the vapour pressure (3.0×10^{-5} mm Hg) and Henry's Law Constant ($K = 1.0 \times 10^{-8}$ atm · m ³ / mole). | |
| Bioaccumulation ⁴ | Log K _{OW} ≥ 5 | | 0.48 | |
| | BCF ≥ 5000 | | Value or not available | |
| | BAF ≥ 5000 | | Value or not available | |
| Is the chemical a TSMP Track 1 substance (all four criteria must be met) | | | No, does not meet TSMP Track 1 criteria. | No, does not meet TSMP Track 1 criteria. |
| <p>¹All pesticides will be considered toxic or toxic equivalent for the purpose of initially assessing a pesticide against the TSMP criteria. Assessment of the toxicity criterion may be refined if required (i.e., all other TSMP criteria are met).</p> <p>²The policy considers a substance “predominantly anthropogenic” if, based on expert judgement, its concentration in the environment medium is largely due to human activity, rather than to natural sources or releases.</p> <p>³ If the pesticide and/or the transformation product(s) meet one persistence criterion identified for one media (soil, water, sediment or air) then the criterion for persistence is considered to be met.</p> <p>⁴Field data (e.g., BAFs) are preferred over laboratory data (e.g., BCFs) which, in turn, are preferred over chemical properties (e.g., log K_{OW}).</p> | | | | |

Table 5 Effects of Mecoprop-p (MCPP-P) on terrestrial organisms.

| Organism | Exposure | Test substance | Endpoint value | Degree of toxicity |
|---|----------------------------------|-----------------------------------|--|-----------------------------------|
| Invertebrates | | | | |
| Earthworm (<i>Eisenia foetida</i>) | Acute | MCPP-P DMAS | LC ₅₀ 494 mg a.e./kg soil | |
| Honey bee (<i>Apis mellifera</i>) | Oral | racemic MCPP ^a acid | LD ₅₀ > 10 µg a.e./bee (LD ₅₀ > 11.2 kg a.e./ha) | Relatively non-toxic ^b |
| | Contact | racemic MCPP ^a acid | LD ₅₀ > 100 µg a.e./bee (LD ₅₀ > 112 kg a.e./ha) | Relatively non-toxic ^b |
| Carabid beetle (<i>Pterostichus cupreus</i>) | | | | |
| Birds | | | | |
| Bobwhite quail (<i>Colinus virginianus</i>) | Acute oral | MCPP-P acid | LD ₅₀ = 462 mg a.e./kg bw | Moderately toxic ^c |
| | Acute dietary | MCPP-P DMAS | LC ₅₀ > 4630 mg a.e./kg diet | Slightly toxic ^c |
| | Reproduction | | NA | |
| Mallard duck (<i>Anas platyrhynchos</i>) | Acute oral | MCPP-P acid | NA | |
| | Acute dietary | MCPP-P acid | LC ₅₀ > 2585 mg a.e./kg diet | Slightly toxic ^c |
| | | MCPP-P DMAS | LC ₅₀ > 3908 mg a.e./kg diet | Slightly toxic ^c |
| Reproduction | | NA | | |
| Japanese quail (<i>Coturnix japonica</i>) | Reproduction | MCPP-P DMAS | NOEC = 827 mg a.e./kg diet | |
| Mammals | | | | |
| Rat | Acute oral | MCPP-P acid | LD ₅₀ = 700 mg a.e./kg bw | Slightly toxic ^c |
| | Dietary (90-day) | MCPP-P acid | NOEC = 800 mg a.e./kg diet | |
| | Reproduction (two generation) | MCPP-P acid | NOEC = 500 mg a.e./kg diet | |
| Mouse | Dietary (90-day) | MCPP-P acid | NOEC = 100 mg a.e./kg diet | |
| Vascular plants | | | | |
| Vascular plant | Seedling emergence | MCPP-P acid | <p>EC₂₅ = 0.02 - >1.18 kg a.e./ha EC₅₀ = 0.04 - >1.18 kg a.e./ha HD₅ of EC₅₀s = 0.0296 kg a.e./ha (seedling emergence)</p> <p>EC₂₅ = 0.50 - >7.87 kg a.e./ha EC₅₀ = 0.73 - >7.87 kg a.e./ha (percent survival)</p> <p>EC₂₅ = 0.0090 - 0.86 kg a.e./ha EC₅₀ = 0.024 - >1.18 kg a.e./ha HD₅ of EC₅₀s = 0.0227 kg a.e./ha (plant height)</p> <p>EC₂₅ = 0.0046-0.61 kg a.e./ha EC₅₀ = 0.0091-0.99 kg a.e./ha HD₅ of EC₅₀s = 0.0081 kg a.e./ha (plant dry weight)</p> | |

| Organism | Exposure | Test substance | Endpoint value | Degree of toxicity |
|----------|-------------------|-----------------|--|--------------------|
| | Vegetative vigour | MCPPP-P DMAS | EC ₂₅ = 0.016 - >1.18 kg a.e./ha EC ₅₀ = 0.03- > 1.18 kg a.e./ha (plant height) EC ₂₅ = 0.0045 - >1.18 kg a.e./ha EC ₅₀ = 0.011- > 1.18 kg a.e./ha HD ₅ of EC ₅₀ s = 0.0069 kg a.e./ha (plant dry weight) | |

^a Toxicity data on racemic mecoprop used as surrogate data for mecoprop-p (MCPP-P) isomer

^b Atkins *et al.* (1981) classification

^c US EPA classification

NA - data not available

Table 6 Effects of Mecoprop-p (MCPP-P) on aquatic organisms.

| Organism | Exposure | Test substance | Endpoint value | Degree of toxicity ^b |
|--|-------------------|---|--|---------------------------------|
| Freshwater species | | | | |
| <i>Daphnia magna</i> | Acute | MCPP-P acid | EC ₅₀ >90 mg a.e./L (mortality) | Slightly toxic |
| | | MCPP-P acid | EC ₅₀ >82 mg a.e./L (immobility) | Slightly toxic |
| | Chronic (21-d) | Racemic MCPP- DMAS (EP) ^a | NOEC = 11.1 mg a.e./L | |
| Rainbow trout (<i>Oncorhynchus mykiss</i>) | Acute | MCPP-P DMAS | LC ₅₀ >123 mg a.e./L | Practically non-toxic |
| | Chronic | MCPP-P acid | LC ₅₀ = 95.7 mg a.e./L NOEC = 49.6 mg a.e./L | Practically non-toxic |
| Bluegill sunfish (<i>Lepomis macrochirus</i>) | Acute | MCPP-P acid | LC ₅₀ >91 mg a.e./L | Slightly toxic |
| Freshwater green algae (<i>Pseudokirchneriella subcapitata</i>) | Acute | MCPP-P DMAS (EP) | EC ₅₀ = 220 mg a.e./L | |
| | | MCPP-P DMAS (EP) | EC ₅₀ = 108 mg a.e./L (biomass); EC ₅₀ >317 mg a.e./L (growth rate) | |
| | | MCPP-P DMAS | EC ₅₀ = 159 mg a.e./L (biomass and growth rate) | |
| | | MCPP-P acid | EC ₅₀ = 249 mg a.e./L (biomass); EC ₅₀ >672 mg a.e./L (growth rate) | |
| Freshwater blue-green algae (<i>Anabaena flos-aquae</i>) | Acute | MCPP-P DMAS | EC ₅₀ = 1.2 mg a.e./L (biomass) | |
| Freshwater diatom (<i>Navicula pelliculosa</i>) | Acute | MCPP-P DMAS | EC ₅₀ = 0.24 mg a.e./L (biomass) | |
| Duckweed (<i>Lemna</i> sp.) | Acute (7-d) | MCPP-P DMAS | EC ₅₀ = 18.7 mg a.e./L (biomass) | |
| | Acute (14-d) | MCPP-P DMAS | EC ₅₀ = 1.9 mg a.e./L (biomass) | |

| Organism | Exposure | Test substance | Endpoint value | Degree of toxicity ^b |
|--|----------|----------------|--|---------------------------------|
| Marine species | | | | |
| <i>Skeletonema costatum</i> (marine alga) | Acute | MCPP-P DMAS | EC ₅₀ = 17 µg a.e./L (biomass) | |

^a Toxicity data on racemic mecoprop used as surrogate data for mecoprop-p (MCPP-P) isomer

^b US EPA classification

Appendix II Monitoring Data

Water Monitoring Data

A search for mecoprop¹ water monitoring data in Canada resulted in a number of samples with detections being reported. The Federal Provincial and Territorial representatives from all of the provinces and territories in Canada were contacted, requesting water monitoring data for mecoprop. In addition, requests were submitted to Environment Canada, the Department of Fisheries and Oceans and the drinking water subcommittee through Health Canada. A response was received by all provinces and territories indicating that either monitoring data were not available or the available data were submitted.

US databases were searched for detections of mecoprop. Data on residues present in water samples taken in the US are important to consider in the Canadian drinking water assessment given the extensive monitoring programs that exist in the US. Runoff events, local use patterns, site specific hydrogeology as well as testing and reporting methods are probably more important influences on residue data rather than Northern versus Southern climate. As for the climate, if temperatures are cooler, residues may break down more slowly, on the other hand if temperatures are warmer, growing seasons may be longer and applications may be more numerous and frequent.

Mecoprop was not analyzed for in the US databases searched.

Approach for Evaluation

Data from Canadian monitoring studies in which mecoprop was quantified are summarized in Table 4 of Appendix I.

For both the ecoscenario assessment and the drinking water assessment, information was extracted from the available sources, tabulated and sorted into categories as follows:

- Residues in known drinking water sources (both surface and groundwater);
- Residues in ambient water that may serve as a drinking water source (both surface and groundwater);
- Residues in ambient water that are unlikely to serve as a drinking water source.

An important limitation of the monitoring data set is that, in many cases, the data were not accompanied with use data for mecoprop. For instance, the rate applied, when the application occurred and weather conditions prior to sampling were generally not known or reported. Without this information, it is difficult to conclude if non-detects were a result of non-transport or more simply a result of inappropriate timing of sampling. In addition, because the data are sparse and concentrations vary in time and space, the maximum concentration reported is unlikely to be the absolute maximum concentration that would be observed in Canada. Factors that may result in higher concentrations being detected include application at higher rates, precipitation and some areas/soils are simply more prone to leaching and/or run off. Sampling at

1 MCPP-P is the herbicidally active isomer of mecoprop. For analytical purposes the term “mecoprop” is used rather than specifying the isomer analyzed.

intervals immediately following application would increase the likelihood that the maximum concentration would be detected.

Thus, it is likely mecoprop was not used in some of the areas monitored, and that higher concentrations of mecoprop may occur in other areas not monitored. The mecoprop monitoring data likely underestimate the peak exposure because of the following limitations:

In general, the data are sparse in both time and location. In some of the studies available, mecoprop was analyzed in samples that were taken from non-mecoprop use areas. Mecoprop use information from the areas surrounding where the samples were collected is often not available.

Sampling in some of the studies was conducted during periods when mecoprop is not applied in Canada (i.e., October through March).

The concentrations of pesticides in surface water are directly related to the frequency and timing of monitoring in relation to pesticide application and runoff events. Therefore, timing and frequency of sampling is likely to be the most important factor influencing the concentration detected and the frequency of detections. Samples are often taken at arbitrary time intervals (i.e., once a month, once a week) and are unlikely to capture the absolute maximum concentration of mecoprop.

The following statistics are used to interpret the information available in each dataset and are summarized in Table 4 of Appendix I.

The detection frequency provides an indication of how often positive detections occur within the given data set. Detection frequency is primarily determined by the limits of detection and is influenced by pesticide use patterns and application rates. Consequently, a wide range of detection frequencies is likely to be expected.

The 95th percentile concentration is calculated and reported. Maximum values should also be considered, especially when the 95th percentile is not available, which occurs when there are insufficient detections to calculate a 95th percentile.

The maximum concentration is reported and is used to determine the 95th percentile concentration to estimate an acute exposure value.

The arithmetic mean with non-detects considered at $\frac{1}{2}$ LOD is used to determine the 95th percentile concentration to estimate a chronic exposure value.

Ecoscenario Exposure Estimates from Monitoring Data

The acute and chronic exposure estimates for mecoprop in Canadian surface water are presented in Table 1. The acute exposure value was estimated from monitoring data by determining the 95th percentile of the maximum concentration detected in each monitoring study/site. The chronic exposure value was estimated by determining the 95th percentile of the arithmetic means of all samples at each site (detects and non-detects) from the monitoring studies. The samples with values less than the LOD were given a value of ½ LOD. Groundwater data and data from water distribution systems were not included in the ecoscenario assessment.

Table 1 Concentrations of Mecoprop in Surface Water Estimated from Available Monitoring Data

| Acute Concentration (µg/L)* | Chronic Concentration (µg/L)** |
|---|--------------------------------|
| 2.9 | 0.40 |
| * 95 th percentile of the maximum detected concentrations from surface water monitoring studies | |
| **95 th percentile of the mean concentration for each study site including ½ LOD for non-detects | |

Drinking Water Exposure Estimates from Monitoring Data

A search for mecoprop water monitoring data in Canada and the US resulted in a number of samples with detections being reported. A detailed summary of the Canadian and US water monitoring studies is provided in Table 4 Appendix I. Calculated EECs for acute and chronic exposure from groundwater and surface water are presented in Table 2.

Table 2 Concentrations of Mecoprop in Drinking Water Estimated from Available Monitoring Data

| Ground Water | | Surface Water | |
|---|----------------------|-------------------|----------------------|
| Acute EEC (µg/L)* | Chronic EEC (µg/L)** | Acute EEC (µg/L)* | Chronic EEC (µg/L)** |
| 0.069*** | 0.003*** | 2.09 | 0.36 |
| * 95 th percentile of the maximum detected concentrations from surface water monitoring studies | | | |
| **95 th percentile of the mean concentration for each study site including ½ LOD for non-detects | | | |
| *** based on one monitoring study only | | | |

References

1.0 Chemistry

| PMRA Document Number | Reference |
|----------------------|---|
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| 1760405 | Mecoprop-p Summary Dossier (A H Marks/ Nufarm Limited) Document J Confidential Information, DACO: 2.11.4 CBI |
| 1760406 | 2007, The Analysis of 5 Batches of R(+) 2-(4-chloro-2-methylphenoxy)propionic acid, DACO: 2.13.3 CBI |
| 1760407 | 2009, A discussion on the formation of MCPA as an impurity of concern in MARKS Mecoprop-p Technical Acid; Reg. No. 27441, DACO: 2.13.4 CBI |
| 1760409 | 2009, Analysis for Tetra-to Octa-Chlorinated Dioxins and Fruans in Seven Batches of (+)--2-(4-chloro-2-methylphenoxy) propanoic acid (MCPP-P, Mecoprop-p), DACO: 2.13.4 CBI |
| 1760410 | 2009, Analysis for Tetra-to Octa-Chlorinated Dioxins and Fruans in Seven Batches of (+)--2-(4-chloro-2-methylphenoxy) propanoic acid (MCPP-P, Mecoprop-p), DACO: 2.13.4 CBI |
| 1760412 | 2009, Analysis for Tetra-to Octa-Chlorinated Dioxins and Fruans in Seven Batches of (+)--2-(4-chloro-2-methylphenoxy) propanoic acid (MCPP-P, Mecoprop-p), DACO: 2.13.4 CBI |
| 1760414 | 2009, Analysis for Tetra-to Octa-Chlorinated Dioxins and Fruans in Seven Batches of (+)--2-(4-chloro-2-methylphenoxy) propanoic acid (MCPP-P, Mecoprop-p), DACO: 2.13.4 CBI |

2.0 Human and Animal Health

| PMRA Document Number | Reference |
|----------------------|--|
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| 1418481 | 1985. Report of the study of the acute dermal toxicity on the rat based on OECD of Duplosan KV/BAS 037 29 H. Report RZ-No: 85/061. PMRA DER 93745. |
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