Pest Management Regulatory Agency
Agence de réglementation de la lutte antiparasitaire

Proposed Acceptability for Continuing Registration

PACR2006-05

Re-evaluation of the Lawn and Turf Uses of the Herbicide (4-chloro-2-methylphenoxy)Acetic Acid (MCPA)

The purpose of this document is to inform registrants, pesticide regulatory officials and the Canadian public that Health Canada's Pest Management Regulatory Agency (PMRA) has re-evaluated the lawn and turf uses of the herbicide 4-chloro-2-methylphenoxy acetic acid, commonly known as MCPA. This Proposed Acceptability for Continuing Registration document provides a summary of the data and information reviewed as well as the rationale for the proposed regulatory decision.

The PMRA has concluded that the use of MCPA to treat lawns and turf does not entail an unacceptable risk of harm to human health or the environment, provided that the mitigation measures proposed in this document are adopted. Standard precautionary statements and label improvements are also required.

By way of this document, the PMRA is soliciting comments from interested parties on the proposed regulatory decision for lawn and turf uses of MCPA. The PMRA will accept written comments on this proposal up to 60 days from the date of publication of this document to allow interested parties an opportunity to provide input into the proposed decision. All comments should be forwarded to Publications at the address below.

(publié aussi en français)

28 April 2006

This document is published by the Alternative Strategies and Regulatory Affairs Division, Pest Management Regulatory Agency. For further information, please contact:

Publications
Pest Management Regulatory Agency
Health Canada
2720 Riverside Drive
A.L. 6605C
Ottawa, Ontario
K1A 0K9

Internet: pmra_publications@hc-sc.gc.ca
www.pmra-arla.gc.ca
Information Service:
1 800 267-6315 or (613) 736-3799
Facsimile: (613) 736-3758

ISBN: 0-662-42337-2 (0-662-42338-0)
Catalogue number: H113-18/2006-5E (H113-18/2006-5E-PDF)

Executive Summary

Health Canada's Pest Management Regulatory Agency (PMRA) has completed a re-evaluation of the lawn and turf uses of the herbicide 4-chloro-2-methylphenoxy acetic acid, commonly known as MCPA. This re-evaluation is part of the PMRA's commitment to review the most common lawn and turf chemicals used in Canada under the *Action Plan for Urban Use Pesticides*[1].

The PMRA has carried out an assessment of available information and has found it sufficient to allow a determination of the safety, merit and value of the use of MCPA for application to lawns and turf. The PMRA has concluded that the use of MCPA and its end-use products to treat lawns and turf does not entail an unacceptable risk of harm to human health or the environment, provided that the mitigation measures recommended in this document are adopted. Standard precautionary statements and label improvements are also required.

These mitigation measures include the following:

- reducing the maximum application rate to 1.7 kg a.e./ha (affects MCPA Amine 500 Liquid Farm Weed Killer, Registration Number 9853, *Pest Control Products Act*);
- requiring a re-entry interval of one day for harvesting and transplanting treated turf following application on sod farms;
- phasing out the diethanolamine (DEA) form of MCPA, unless further data are provided; and
- adding buffer zones to commercial products applied by tractor-pulled sprayers to protect surrounding broad-leaved vegetation.

The PMRA will accept written comments on this proposal up to 60 days from the date of this document to allow interested parties an opportunity to provide input into the proposed re-evaluation decision for these products.

[1] More information on this program can be obtained at http://www.healthylawns.net.

Table of Contents

1.0 Purpose

This document describes the outcome of the re-evaluation of the herbicide 4-chloro-2-methylphenoxy acetic acid, commonly known as MCPA, and its end-use products for use on lawns and turf in Canada by Health Canada's Pest Management Regulatory Agency. The assessment considered the potential impact of MCPA on the health and safety of users and others incidentally exposed when these products are used on residential lawns, the potential environmental impact associated with using MCPA and its value as a herbicide in the maintenance of lawn and turf.

This re-evaluation was completed as part of the PMRA's commitment to review the most common lawn and turf chemicals used in Canada under the *Action Plan for Urban Use Pesticides*.

2.0 Background

2.1 Information Used in this Assessment

Information considered by the PMRA in the assessment of MCPA included proprietary data from individual registrants, the Industry Task Force III on MCPA, the Pesticide Handlers Exposure Database (PHED), the Outdoor Residential Exposure Task Force (ORETF) and the Broadleaf Turf Herbicide Transferable Foliar Residue Task Force. Regulatory documents from the United States Environmental Protection Agency (USEPA) and published studies were also reviewed.

2.2 Regulatory History of MCPA

MCPA was first registered in Canada in 1951 (Registration Number 3764, *Pest Control Products Act*). Unlike 2,4-D, mecoprop and dicamba, MCPA products that are registered for use on fine turf are formulated with MCPA as the sole active ingredient. As of 5 May 2005, one Domestic Class product and four Commercial Class products containing MCPA are registered for use on fine turf in Canada (Appendix I). No pesticide/fertilizer combination products containing MCPA are registered in Canada.

In September 2000, the PMRA published the *Action Plan on Urban Use Pesticides*, which gave priority to re-evaluating the lawn and turf uses of a number of pesticides. That month, the PMRA also formally announced the re-evaluation of the most commonly used lawn and turf pesticides, including MCPA, in Re-evaluation Note REV2000-04, *Re-evaluation of Lawn and Turf Uses of Pesticides*. In this document, the PMRA indicated that the review of the lawn and turf uses for MCPA would proceed in advance of the completion of the overall re-evaluation for MCPA, which will include all of its agricultural uses. The re-evaluation of the agricultural uses of MCPA is ongoing and will be the subject of a separate document.

2.3 Definitions of Turf and the Scope of this Review

The re-evaluation of the lawn and turf uses of MCPA has focussed on the assessment of risks resulting from the treatment of the following types of turf:

- sports and recreational turf such as turf in parks, playgrounds, golf courses[2], zoos, botanical garden and athletic playing fields;
- lawn turf such as turf planted in or around residences, public and commercial buildings including schools and cemeteries; and
- sod grown in sod farms and harvested for transplanting[2].

These types of turf are collectively known as fine turf, which may be maintained by homeowners or by professional applicators. Utility turf, also known as rough turf, is not included in this assessment. Utility turf is primarily intended for soil stabilization, requires less maintenance than fine turf and is usually maintained with commercial class products and equipment intended for large-scale application. Utility turf (i.e., roadsides; rights-of-way for railways, hydro installations, pipelines and highways; highway interchanges; airports; wasteland; and industrial parks) will be considered when agricultural uses of MCPA are re-evaluated.

2.4 Forms of MCPA

MCPA for turf is sold either as an amine salt (with dimethylamine [DMA] or diethanolamine [DEA]) or as a mixture of sodium and potassium salts, all based on MCPA acid. Different forms facilitate absorption of the MCPA into the plant differently. Compared to the free acid form, an amine salt formulation greatly increases the water solubility of the herbicide, which is desirable when effective use of the product depends on uptake by the plant via the roots.

The parent acid is the herbicidally active portion of the formulation. While the amine portion of the formulated product may allow for greater absorption into the plant, the parent acid is what binds to the herbicide target site within the plant and causes plant death. For example, when an amine salt of an herbicide penetrates the cuticle, the salt dissociates and forms the parent acid *in situ*. As a result, following absorption, the amine part of the formulation plays no direct role in herbicidal activity. Therefore, when assessing MCPA, the application rates were expressed in terms of the amount of acid equivalent per hectare (e.g., kg a.e./ha).

---

[2] Although excluded from the announcement of re-evaluation of turf uses (REV2000-04), the use of MCPA on golf courses and sod farms is addressed in the current assessment.

Other differences in the various forms of MCPA will be explained in the mammalian toxicology and environmental toxicology and fate sections of this review. The names of the various forms of MCPA for lawn and turf use are listed in Table 2.4.1.

Table 2.4.1 Forms of MCPA Included in this Assessment

Grouping	Form
Parent compound	MCPA
Salts	diethanolamine salt (DEA)[a]
	dimethylamine salt (DMA)
	sodium and potassium salt

[a] The MCPA Task Force III does not support the MCPA-DEA formulation.

3.0 Re-evaluation of the Turf Uses of MCPA

3.1 Identity of the Active Substance and the End-use Products Containing It

Active Substance: MCPA

Function: Herbicide

Chemical Names:

IUPAC: (4-chloro-2-methylphenoxy)acetic acid

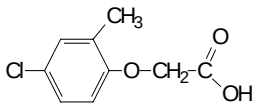
CAS: (4-chloro-2-methylphenoxy)acetic acid

CAS Number: 94-74-6

Molecular Formula: $C_9H_9ClO_3$

Molecular Weight: 200.6

Structural Formula:



3.1.1 Registration Number, Purity and Registrant of the Technical Grade Active Ingredient

Registration Number	Purity of Technical Grade Active Ingredient[a] (%)	Registrant
18783	94.0 (min.)	Nufarm Ltd.
19034	97.0 (94.1–99.9)	Nufarm Agriculture Inc.
19212	96.5 (93.5–99.5)	A.H. Marks and Company Ltd.
19262	96.5 (93.5–99.5)	A.H. Marks and Company Ltd.
20921	96.5 (93.5–99.5)	Nufarm Agriculture Inc.
21156	96.0 (min.)	Dow AgroSciences Canada Inc.
26229	93.0 (min.)	Dow AgroSciences Canada Inc.

[a] Nominal guarantee (upper and lower limits), unless otherwise specified.

3.2 Physicochemical Properties of MCPA Acid and Interpretation

Property	Result	Interpretation
Vapour pressure of the acid at 20°C	2.3×10^{-2} mPa	Low potential to volatilize
Henry's law constant	7.46×10^{-5} Pa m^3 mol^{-1}	Non-volatile from water or moist surfaces
Ultraviolet (UV) – visible spectrum	Not expected to show significant UV absorption at wave length > 300 nm.	Low potential for phototransformation
Solubility in water of the acid at 25°C	**pH** **Solubility (g/L)** 5 26.2 7 293 9 320.1	Very soluble at all pH levels
n-Octanol–water partition coefficient at 25°C	**pH** **Log K_{ow}** 5 0.59 7 -0.71	Unlikely to bioaccumulate
Dissociation constant	p*K*a = 3.07	Dissociates rapidly at environmental pHs

4.0 Effects Having Relevance to Human Health

4.1 Toxicology Summary

The toxicology database for the various forms of MCPA in products for lawn and turf use consisted of proprietary and published studies conducted in laboratory animals using MCPA acid and the dimethylamine (DMA) salt, as follows:

- metabolism studies;
- acute and short-term studies conducted in several mammalian species via various routes of exposure;
- chronic toxicity studies in rats, mice and dogs;
- a battery of mutagenicity/genotoxicity studies;
- reproductive and developmental toxicity studies; and
- acute and short-term neurotoxicity studies.

Although the 2-ethylhexyl ester (2-EHE) form of MCPA is used for agricultural products rather than for lawn and turf use, the toxicity data for MCPA-2-EHE was compared to that of the other forms of MCPA to assess toxicological equivalence. Regulatory documents from the United States Environmental Protection Agency were also considered.

A comparison of acute, short-term, developmental toxicity, neurotoxicity and genotoxicity studies indicated that the acid, DMA and 2-EHE forms of MCPA have similar toxicological profiles. However, certain quantitative differences were noted between MCPA acid, and the DMA and EHE forms of MCPA, as evidenced by different no-effect levels in short-term toxicity studies. These differences in no-effect levels were taken into consideration as part of the risk assessment. It is anticipated that the sodium and potassium salts of MCPA dissociate into MCPA acid and the relatively non-toxic sodium or potassium moiety.

There was no toxicological information on the DEA form of MCPA. However, concerns arise from published data showing that repeated dermal application of DEA on its own is carcinogenic in mice (National Toxicology Program 1997, 2001). No tumours were evident in a similar study conducted in rats, although the doses used were lower than those used in the mouse studies. Short-term oral and dermal studies also indicate that pure DEA causes brain and spinal cord demyelination in rats and is immunotoxic in rats and mice (NTP 1992a, 1992b, 1994). DEA is also identified as a List 2 formulant (potentially toxic formulants, with a high priority for testing). Based on apparent differences in the toxicological profile for DEA on its own relative to MCPA acid and other MCPA salts, further assessment for MCPA-DEA was not possible. As the MCPA Task Force III has indicated that it does not support this form of MCPA, the PMRA is proposing to phase out this form of MCPA (see Section 8.1).

4.1.1 Toxicology Profile of MCPA as Acid, 2-EHE, DMA, Sodium or Potassium Salt

Available data indicated that MCPA was readily absorbed and excreted by rats after oral administration, with an elimination half-life ($t_{1/2}$) of three to seven hours. In laboratory animals, the various forms of MCPA ranged from slight to moderate acute toxicity via the oral route of exposure, they were of low acute dermal and inhalation toxicity, slightly or non-irritating to skin and ranged from being minimally to severely irritating to eyes, depending on the formulation.

Short-term dermal exposure resulted in decreased body-weight gain and renal tubule mineralization in rabbits treated with MCPA acid, and reduced hematology parameters in rats treated with MCPA-EHE, both at very high doses. Short- and long-term dietary studies in rats, mice and dogs indicated that the primary target organ for all species was the kidney, with dogs being the most sensitive (dog > rat > mouse). At higher doses, thyroid changes were also noted in dogs, with testicular and liver effects observed in all species. In acute and short-term neurotoxicity studies, clinical signs of neurotoxicity were noted at high doses. These neurotoxic effects were reversible, with no associated neuropathological findings.

The long-term mouse or rat studies using MCPA acid showed no evidence of oncogenicity. However, the long-term rat study did not achieve the maximum tolerated dose and, therefore, was not considered adequate for assessing the overall potential for carcinogenicity. Genotoxicity tests provided equivocal results for sister chromatid exchange induction and positive results were obtained for all three forms of MCPA in mammalian in vitro lymphocyte assays. However, in vivo mammalian cytogenetic assays were negative.

The maximum tolerated dose was not achieved in the multigeneration rat reproduction study and no adverse toxic effects were noted in the parental animals. However, both generations of pups had decreased body weight and body-weight gain on days 14 and 21 of lactation, demonstrating the potential for increased sensitivity of the young in the absence of maternal toxicity. In the developmental toxicity studies, developmental effects in rats occurred at maternally toxic doses. These included decreased fetal body weight, reduced viable litter size with a corresponding increase in postimplantation loss/early resorption, delayed ossification, and malformations (hydrocephaly, bent limbs, fused ribs). The magnitude of the developmental effects relative to maternal toxicity suggested increased qualitative sensitivity of the fetus.

4.1.2 Selection of Toxicological Endpoints for Risk Assessment

The toxicology endpoints used in the risk assessment of MCPA are based on studies in laboratory animals and are summarized in Table 4.1.2.1. Each endpoint is explained further in sections 4.2 to 4.5, as each scenario to which it is applied is discussed. Reference doses for various populations and subgroups have been set based on no observed adverse effect levels (NOAELs) for the most relevant endpoints, namely effects on body weight, renal toxicity (the primary target organ), neurotoxicity and

developmental effects occurring in the presence of maternal toxicity. These reference doses incorporate various safety/uncertainty factors to account for extrapolating between rats and humans, for variability within human populations, for data uncertainties, and for severity of effects. Additional safety factors have also been applied, where warranted, to protect children and pregnant females from the endpoints of concern indicated above.

Table 4.1.2.1 Toxicological Endpoints Used in the MCPA Lawn/Turf Risk Assessment

Endpoint	Population	MCPA Acid, DMA, 2-EHE		
		NOAEL (mg/kg bw/day)	Study	UF/SF or MOE
ARfD	Females 13–49 yrs old	40	Rat developmental	300
	GP/children	LOAEL: 146	Acute rat neurotoxicity	300
ADI[a]	All populations	0.22	One-year dog	1000
Short-term: dermal 1–7 day[a]	Females 13–49 yrs old	40	Rat developmental	300
	Children	100	Rabbit dermal	100
Short-term: dermal 1–7 day; 8–30 day	GP	100	Rabbit dermal	100
Short-term dermal: 8–30 day	Females 13–49 yrs old	30	Rabbit developmental	300
Short-term: 1–7 day incidental oral	Toddlers	30	Rabbit developmental	100
Short-term: 1–7 day inhalation	Females 13–49 yrs old	40	Rat developmental	300
	GP/children	30	Rabbit developmental	100
Short-term: 8–30 day inhalation	All populations	30	Rabbit developmental	300
Aggregate: 1–7 day	Females 13–49 yrs old	40[b] all routes	Rat developmental	300
	GP/children	100 dermal 30 oral/ inhalation[b]	Rabbit dermal Rabbit developmental	100

UF/SF (Uncertainty factor/Safety factor); ARfD (acute reference dose); GP (general population); ADI (acceptable daily intake); LOAEL (lowest observed adverse effect level) Females 13–49 years old (females of child-bearing age).

[a] The PMRA has recently received additional data from the MCPA Task Force III to address the applicability of the dog model as an indicator species for the human health assessment of MCPA as well as the potential for sensitivity of the young. These data will be fully assessed during the re-evaluation of MCPA for agricultural use. In the interim, a conservative ADI was established for the purpose of conducting an aggregate risk assessment.

[b] Where an oral endpoint is used in risk assessment, dermal absorption was considered to be 19% of the oral dose and inhalation absorption was considered to be 100% (default value) of the oral dose.

4.2 Residential Risk Assessment

The residential risk assessment for lawn and turf use of MCPA encompasses the exposures that adults may receive while applying MCPA to their lawn as well as those adults and children may receive through contact with treated turf.

Residential risk is estimated by calculating a margin of exposure (MOE) based on comparing the potential exposure to the most relevant endpoints from toxicology studies. The calculated MOE is then compared to a target MOE, which incorporates safety factors protective of the most sensitive subpopulations. If the MOE is less than this target MOE, it does not necessarily mean that exposure will result in adverse effects, rather that the absence of adverse effects is less certain. Mitigation measures are necessary to reduce exposure if MOEs are less than the target MOE.

4.2.1 Relevant Toxicological Endpoints and Target Margins of Exposure for Acute and Short-term Exposures of Homeowners and Children

For adults, the risk associated with a one-day (i.e., acute) exposure to MCPA was based on the most sensitive subgroup, which, in this case, was females of child bearing age (females 13 to 49 years) and the developing fetus. Protecting the most sensitive subpopulation inherently protects the general population. The most relevant endpoints for acute risk assessment were considered to be decreased viable litter size, increased postimplantation loss, resorptions, hydrocephaly and bent limb bones noted at maternally toxic doses in a rat developmental study. Any one of these endpoints could occur following a single exposure event. In this study, the NOAEL was 40 mg/kg bw/day a.e., with the above noted effects occurring at the lowest observed adverse effect level (LOAEL) of 120 mg/kg bw/day a.e. The target MOE was 300, based on standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation) as well as an extra 3-fold safety factor for the severity of the endpoints (qualitative sensitivity to the developing fetus) noted in the presence of maternal toxicity.

The effects identified in the rat developmental toxicity study above only pertain to the developing fetus and were not considered relevant to young children. Therefore, a separate acute exposure and risk assessment was conducted to account for the differences in physiological and behavioural parameters of children, which can result in unique exposures (e.g., hand-to-mouth exposures through touching treated turf). The endpoint of concern was ataxia in an acute rat neurotoxicity study, which was not associated with any neuropathological findings. The study LOAEL was 146 mg/kg bw/day (a.e.), the lowest dose tested. The target MOE was 300 based on application of the standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation) as well as an extra 3-fold to account for the use of a LOAEL instead of a NOAEL.

For short-term (1 to 7 days) dermal exposure to MCPA, a dermal endpoint was used in the risk assessment for the general population. A rabbit dermal study with a systemic NOAEL of 100 mg/kg bw/day was chosen, based on a decrease in body-weight gain and an increase in renal tubule mineralization at 1000 mg/kg bw/day. The target MOE was 100, based on the application of standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation). This same endpoint and target MOE was used to assess short-term (1 to 7 days) dermal exposure of children. Although the potential for increased sensitivity in the young was noted in the rat reproduction study, the exposure duration that resulted in effects in rat offspring was longer than the 1- to 7-day assessment period; thus, an additional safety factor was not required for this exposure scenario.

Effects resulting from the oral route of exposure were used in the short-term (1 to 7 days) dermal risk assessment for females of child-bearing age (females 13 to 49 years) to protect this sensitive subpopulation. In this study, the NOAEL was 40 mg/kg bw/day (a.e.), with decreased viable litter size, increased postimplantation loss, resorptions, hydrocephaly and bent limb bones occurring at the maternally toxic dose of 120 mg/kg bw/day (a.e.). The target MOE was 300, based on application of the standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation) as well as an extra 3-fold safety factor for the severity of the endpoints (qualitative sensitivity to the developing fetus) noted in the presence of maternal toxicity.

For the short-term inhalation exposure scenario (1 to 7 days), oral endpoints were chosen in the absence of inhalation studies of suitable duration. Assessment of short-term inhalation risk to the general population, including children, was based on a NOAEL of 30 mg/kg bw/day established in a rabbit developmental study. Decreased body weight and food consumption were observed at the LOAEL of 60 mg/kg bw/day. The target MOE was 100, based on application of the standard uncertainty factors (10-fold interspecies variation, 10-fold intraspecies variation). Although the potential for increased sensitivity in the young was noted in the rat reproduction study, the exposure duration that resulted in effects in rat offspring was longer than the 1- to 7-day assessment period; thus, an additional safety factor was not required for this exposure scenario.

For females of child-bearing age (13 to 49 years), effects resulting from the oral route of exposure were used for the 1 to 7 day short-term inhalation risk assessment, in order to protect this sensitive subpopulation. In this study, the NOAEL was 40 mg/kg bw/day (a.e.), with decreased viable litter size, increased postimplantation loss, resorptions, hydrocephaly and bent limb bones occurring at the maternally toxic dose of 120 mg/kg bw/day (a.e.). The target MOE was 300, based on application of the standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation) as well as an extra 3-fold safety factor for the severity of the endpoints (qualitative sensitivity to the developing fetus) noted in the presence of maternal toxicity.

To assess the risk to toddlers that could result from any potential non-dietary short-term oral exposure (1 to 7 days), an oral developmental study in rabbits was considered most

relevant with respect to route, duration of dosing and measurement of offspring sensitivity. The NOAEL was 30 mg/kg bw/day based on a decrease in body weight, body-weight gain and food consumption at the LOAEL of 60 mg/kg bw/day. The target MOE was 100, based on the application of the standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation). Although the potential for increased sensitivity in the young was noted in the rat reproduction study, the exposure duration that resulted in effects in rat offspring was longer than the 1- to 7-day assessment period; thus, an additional safety factor was not required for this exposure scenario.

For risk assessments based on animal toxicity studies that involve exposure via the oral route, estimations of risk to humans resulting from dermal exposure must include a correction for the differences between oral and dermal absorption. Based on the weight of evidence in consideration of dermal absorption studies submitted by the MCPA Task Force III as well as other available data on dermal absorption, a value of 19% was considered appropriate.

As stated in Section 4.1, no toxicological information on the diethanolamine (DEA) form of MCPA was available. As the MCPA Task Force III has indicated it does not support this form of MCPA, the PMRA is proposing to phase out this form (see Section 8.1). Therefore, a residential risk assessment for MCPA-DEA has not been included at this time.

4.2.2 Exposure and Risk Assessment for Homeowners Mixing, Loading and Applying MCPA to Residential Lawns

Homeowners typically apply MCPA to their lawns a maximum of twice a year. Therefore, residential applicators have the potential for short-term periods of exposure (less than seven days).

Dermal and inhalation exposure estimates for homeowner application on residential turf are based on PHED Version 1.1 data and ORETF studies.

The PHED is a compilation of generic mixer/loader/applicator passive dosimetry exposure data that can be used to generate scenario-specific exposure estimates. The ORETF studies monitored exposure of workers and homeowners mixing/loading and applying pest control products to turf. Monitoring was conducted using passive dosimetry, including hand washes, face/neck wipes and personal air samplers.

Exposure is calculated as the product of the unit exposure for a given scenario, the application rate and the area treated per day divided by body weight. For broadcast applications, it was assumed that residential applicators treated an area of 2000 m^2 per day. This is considered an upper percentile estimate.

Exposure and risk estimates as well as details on the calculations are presented in Appendix II. Calculated MOEs for all residential applicators are above the target MOE of 300.

4.2.3 Exposure and Risk Assessment for Persons Entering a Treated Area

Postapplication exposure and risk were estimated for children and adults contacting treated residential lawns and golf courses, based on assumptions outlined in the USEPA's draft *Standard Operating Procedures (SOPs) for Residential Exposure Assessments* and the recommended revisions by the USEPA's Science Advisory Council (USEPA 1997, 2001).

Postapplication dermal exposures were estimated using generic transfer coefficients and MCPA turf transferable residue (TTR) data. Transfer coefficients are defined in the USEPA draft SOPs and measure the relationship between dermal exposure and TTR for individuals engaged in a specific activity on treated turf.

Acute and short-term risk assessments were conducted as there is potential for relatively higher exposures of children and adults on the day of application, and for repeated lower exposures over a short-term period (1 to 7 days), as residues of MCPA dissipate. Based on TTR data generated by the Broadleaf Turf Herbicide Transferable Foliar Residue Task Force, peak TTR levels were 7.3% of the applied rate, and the 7-day average TTR levels were 0.61% of the applied rate.

New postapplication exposure data relevant to estimating dermal exposure from contact with treated turf were received from the ORETF in 2004. The PMRA, the USEPA and California Department of Pesticide Regulation are currently evaluating these data. Preliminary calculations suggest that, while exposure estimates might increase slightly, target MOEs would still be met for all postapplication scenarios. If necessary, the PMRA will publish a revised risk assessment after a full review of the new data.

Non-dietary oral exposure was assessed for toddlers, as they could ingest residues through hand-to-mouth transfer from turf or other surfaces, by mouthing grass or by ingesting soil. As well, oral ingestion of granules was considered, although this is considered to be an acute, episodic exposure event rather than a typical exposure.

The contribution of inhalation exposure to the overall exposure in postapplication scenarios is considered to be negligible, due to the dilution effect of outdoor use and considering the study by Yeary and Leonard (1993) wherein MCPA was not detected in the breathing zone of 25 applicators during the application of MCPA to residential lawns, trees and shrubs (limit of detection of 0.001 mg/m^3).

Calculated acute and short-term MOEs for adults and toddlers exceeded the target MOEs. This indicates that the potential exposures are below levels that would be of concern. Further details on calculations as well as exposure and risk estimates are presented in Appendix II.

4.3 Dietary Assessment

A dietary exposure assessment was conducted so that aggregate exposure and risk could be estimated (see Section 4.4.2). An aggregate risk assessment considers the risk resulting from combined exposures from all sources and routes, including food, drinking water and residential (lawn and turf) exposures.

4.3.1 Dietary Exposure

The dietary exposure assessment estimated how much MCPA residues, including residues in milk and meat, may be ingested with the daily diet. The assessment was age-specific and incorporated the different eating habits of the population at various stages of life. For example, the assessment took into account the greater consumption of fruits, vegetables and juices by children, relative to their body weight, as compared to adults.

The assessment is based on the residue of concern being defined as MCPA (parent compound only). The definition of the residue of concern will be considered when the overall re-evaluation for MCPA is performed. All Canadian and foreign food commodities for which MCPA has a registered use were considered in the dietary risk assessment. There are no registered aquatic uses of MCPA in Canada. The assessed domestic and foreign uses on food and feeds include the following.

Domestic	**Foreign**
Rye (winter and spring)	Asparagus
Barley (malting, winter and others)	Sorghum
Blueberry	Cotton (seeds, meal)
Cereal crops	Corn
Corn	Oats
Sweet corn	Millet
Pasture and rangeland grasses	Proso
Oats	Barley (grain, straw)
Wheat (spring, winter and durum)	Grass (forage, hay)
Strawberry	Soybean (seeds, hull)
	Wheat grain
	Sugarcane (forage and molasses)

The dietary risk assessments were conducted using monitoring data from Canada, the United States Department of Agriculture Pesticide Data Program, the United States Food and Drug Administration and processing studies. Where no data were available, potentially treated commodities were assessed using the default maximum residue limit of 0.1 ppm under general regulation B.15.002(1) of the Food and Drug Regulations. The dietary exposure scenarios were assessed for all human populations and for population subgroups. The dietary exposure and risk estimates were generated using the Dietary Exposure Evaluation Model (DEEM) and consumption data from the United States Department of Agriculture's Continuing Survey of Food Intakes by Individuals (1994–1996, 1998).

4.3.2 Dietary Risk

An acute dietary exposure assessment considers the highest ingestion of MCPA likely on any one day. A probabilistic statistical analysis allows all possible combinations of food consumption and residue levels to be combined to generate a distribution of the amount of MCPA residue that might be eaten in one day. A value representing the high end (99.9^{th} percentile) of this distribution, which is referred to as the potential daily intake (PDI), is compared to the acute reference dose (ARfD). The ARfD is the dose at which an individual could be exposed on any given day and expect no adverse health effects. When the expected PDI from residues is less than the ARfD, this intake is not considered to be of concern.

To protect expectant mothers and unborn children, an ARfD was set at 0.13 mg/kg bw/day. This was based on the oral rat developmental NOAEL of 40 mg/kg bw/day (a.e.) and application of a 300-fold uncertainty/safety factor (10-fold for interspecies variation, 10-fold for intraspecies variation and an additional 3-fold to account for severity of effects [qualitative sensitivity to the developing fetus] noted in the presence of maternal toxicity). In this study, decreased viable litter size, increased postimplantation loss, resorptions, hydrocephaly and bent limb bones were noted at maternally toxic doses. The acute PDI (99.9^{th} percentile) for females of child bearing age accounted for 3.5% of the ARfD. Protecting the most sensitive subpopulation inherently protects the general population. The acute PDI for all subpopulations was < 4.4% of the ARfD (Table 4.3.2.1).

Chronic dietary exposure is calculated using the average consumption of different foods and average residue values on those foods over a 70-year lifetime. This expected intake of residues is compared to the acceptable daily intake (ADI), which is the dose at which an individual could be exposed over the course of a lifetime and expect no adverse health effects. When the expected intake from residues is less than the ADI, this intake is not considered to be of concern.

The ADI was set at 0.00022 mg/kg bw/day. This ADI is based on a NOAEL of 0.22 mg/kg bw/day from a 1-year dietary study in dogs and applying a 1000-fold uncertainty/safety factor. At the next highest dose level, kidney effects were noted. In addition to the standard uncertainty factors (10-fold for interspecies variation, 10-fold for

intraspecies variation), an additional factor of 10-fold was applied for potential sensitivity to the young noted in the rat reproduction study and for the lack of an acceptable rat oncogenicity study. The rat oncogenicity study did not reach the maximum tolerated dose; therefore, the oncogenic potential of MCPA could not be fully assessed. This ADI provides a margin of safety of 182 000 to the NOAEL of 40 mg/kg bw/day for developmental effects noted in the rat developmental study. The chronic PDI accounted for 92.3% of the ADI in children ages 1 to 6 years old, with the chronic PDI in the remaining subgroups accounting for < 58% of the ADI.

These chronic and acute dietary risk assessments demonstrated that there were no health concerns for any population subgroup in Canada, including infants, children, teenagers, adults and seniors. The dietary exposure estimates are presented in Table 4.3.2.1.

Table 4.3.2.1 Chronic and Acute Dietary Exposure and Risk Summary for MCPA

Population Subgroup	Chronic Dietary Exposure		Acute Dietary Exposure	
	mg/kg bw/day	% ADI	mg/kg bw	% ARfD
General population	0.000 084	38.2	0.013 326	2.74
Non-nursing infants	0.000 114	51.9	0.021 229	4.37
Children 1–6 years	0.000 203	92.3	0.019 634	4.03
Children/Youth 7–12 years	0.000 127	57.6	0.009 186	1.89
Females 13–49 years	0.000 077	35.1	0.004 597	3.46

4.3.3 Drinking Water

As indicated above, residues in drinking water can be a potential source of exposure to MCPA. To evaluate this source's contribution to overall exposure, drinking water quality monitoring data from several sources, ranging from provincial reports to scientific studies, were considered. The combined Canadian data set incorporated monitoring results from ambient surface and groundwater, as well as treated municipal drinking water. These data were supplemented by relevant monitoring information from the United States. Based on these data, the locations of high MCPA concentrations are generally randomized and do not persist. When detected, residues of MCPA in surface waters were generally ≤ 0.26 µg/L. The maximum estimates of acute and chronic residues of MCPA in drinking water were 4.2 and 0.26 µg/L, respectively, based on the 95th percentile of observed absolute maximum concentrations.

Canadian drinking water levels of comparison (DWLOCs) were calculated to assess whether these concentrations posed any risk. The DWLOC is the maximum concentration in drinking water that, when considered together with all other sources of exposure, does not exceed a level of concern. The acute and chronic DWLOCs were > 3900 and 0.26 µg/L, respectively. As the acute and chronic anticipated residues of MCPA in drinking water do not exceed the respective DWLOCs indicated in Table 4.3.3.1, they are below the PMRA's level of concern.

Table 4.3.3.1 Chronic and Acute Drinking Water Levels of Comparison for MCPA

Population Subgroup	Chronic Drinking Water Exposure	Acute Drinking Dater Exposure
	DWLOC[a] (µg/L)	DWLOC[a] (µg/L)
General population	4.76	16 600
Non-nursing infants	1.06	4670
Children 1–6 years	0.26	7010
Children/Youth 7–12 years	4.09	21 000
Females 13–49 years	4.43	3970

[a] Where DWLOC = (reference dose − dietary exposure) × (body weight) / (water consumption). Body weight is considered to be 70, 62, 44, 15 and 10 kg for adults, adult females, children/youth (7–12 years), children and infants, respectively. Water consumption is 2 L/day, except for children; for children 1–6 years and infants, water consumption is 1 L/day.

4.4 Aggregate Risk Assessment

The purpose of aggregating exposure is to estimate the risk resulting from total exposure to MCPA from all sources and routes of exposure, including food, drinking water and residential exposures.

4.4.1 Acute Aggregate Risk Assessment

Acute aggregate risk is estimated as the risk that would result from the highest likely single day exposures to MCPA. Acute aggregate exposure to MCPA combines dietary and drinking water exposures only. The acute aggregate risk assessment did not incorporate residential exposure as it is improbable that an individual would be exposed to high-end dietary and residential exposures on the same day. Average (chronic) dietary exposure is a very small fraction of the highest one-day residential exposure and would not have an impact on the total risk.

The acute PDI for all subpopulations was < 4.4% of the ARfD (Table 4.3.2.1).

To aggregate the acute drinking water and dietary exposure, acute DWLOCs of > 3900 µg/L were calculated and assessed against the acute drinking water estimate of 4.2 µg/L. The acute exposure from drinking water sources is below the DWLOC. As both the dietary and drinking water exposures are acceptable, the acute aggregate exposure is not of concern.

4.4.2 Short-term Aggregate Risk Assessment

Short-term aggregate exposure (1 to 7 days) to MCPA was estimated based on contributions from food, drinking water and residential exposures (dermal, inhalation and oral components).

The most relevant data with respect to route and duration for this risk assessment were from a 3-week dermal study in rabbits and from the 2-week oral developmental studies in rats and rabbits. These studies confirmed that a decrease in body-weight gain was the endpoint of concern in the oral and dermal routes of exposure. Despite the absence of repeat-dose inhalation data, it is assumed that body weight would also be a critical endpoint by this route of exposure. Therefore, the endpoints selected for short-term aggregate risk assessment included the dermal systemic NOAEL of 100 mg/kg bw/day from the 3-week dermal study and the oral NOAEL of 30 mg/kg bw/day from the rabbit developmental toxicity study. In lieu of suitable inhalation data, the oral NOAEL for body-weight effects was also used for the inhalation exposure component of the aggregate risk assessment. Standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation) were used to establish a target MOE of 100 for the general population including children. Although the potential for increased sensitivity in the young was noted in the rat reproduction study, the exposure duration that resulted in effects in rat offspring was longer than the 1- to 7-day assessment period; thus, an additional safety factor was not required.

For females of child bearing age (females 13 years to 49 years), an additional endpoint of concern for short-term aggregate exposure included developmental effects (increased malformations and variations) noted at maternally toxic doses in rat developmental studies. The NOAEL for these effects is 40 mg/kg bw/day (a.e.). It was assumed that these developmental effects could result from exposure by each of the various routes

(oral, dermal or inhalation); therefore, this endpoint served as the default for all three exposure components of the aggregate risk assessment for this subpopulation. The target MOE was 300 and included the standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation) as well as an additional safety factor of 3-fold to account for the severity of the endpoint (qualitative sensitivity to the developing fetus) in the presence of maternal toxicity.

The chronic dietary exposure was considered representative of a typical exposure because it represents the average daily exposure over an individual's lifetime. Ingestion of granules is not aggregated in the short-term oral scenario as this is considered to be episodic rather than a typical exposure event.

Dermal exposure was extrapolated to a systemic exposure by considering a default 19% dermal absorption factor. Inhalation exposure and oral ingestion through dietary and non-dietary pathways are considered to be 100% absorbed. However, the contribution from inhalation exposure in postapplication scenarios is considered to be negligible.

Short-term aggregate exposure estimates of food, residential exposure (dermal, inhalation and incidental oral components) and drinking water did not indicate any unacceptable risk. The calculated DWLOCs ranged from 1500 to 6600 µg/L. These were compared to the chronic estimate of MCPA residues in drinking water, which is 0.26 µg/L. This is lower than the calculated DWLOCs for all populations and below the PMRA's level of concern.

Further details on the exposure calculations as well as estimates of short-term aggregate exposure and risk are summarized in Appendix II.

4.4.3 Chronic Aggregate Risk Assessment

Chronic aggregate exposure to MCPA is considered to arise from dietary and drinking water exposures only. Residential exposure is not included, as all the relevant time frames and exposure routes are considered in the short-term aggregate risk assessment. The derivation of the dietary and drinking water exposure estimates is described in tables 4.3.2.1 and 4.3.3.1.

The chronic PDI accounted for < 93% of the ADI for all population subgroups, with children 1 to 6 years being the most highly exposed subpopulation.

Chronic DWLOCs of ≥ 0.26 µg/L were calculated and assessed against the 95th percentile chronic drinking water estimate of 0.26 µg/L. The chronic exposure from drinking water sources does not exceed the DWLOC. As both the dietary and drinking water exposures are acceptable, the chronic aggregate exposure is not of concern.

4.5 Occupational Risk Assessment

Occupational risk is estimated by comparing the potential exposure of persons mixing, loading and applying pesticides or re-entering treated areas, to the no-effect level for an endpoint from the most relevant toxicology study with respect to route and duration. This generates the MOE. The MOE is compared to a target MOE that incorporates safety factors protective of the most sensitive population. If the MOE is less than this target MOE, it does not necessarily mean that exposure will result in adverse effects, rather that the absence of adverse effects is less certain. Mitigation measures will be necessary to reduce exposure if MOEs are less than the target MOE.

4.5.1 Relevant Toxicological Endpoints and Target Margins of Exposure for Acute and Short-term Exposures of Commercial Applicators and Re-entry Workers

To protect the most sensitive subpopulation, the unborn child of pregnant workers (females 13 to 49 years), the most relevant endpoints for acute worker risk assessments were considered to be decreased viable litter size, increased postimplantation loss, resorptions, hydrocephaly and bent limb bones noted at maternally toxic doses in a developmental rat study. These endpoints could occur following a single exposure event. Protecting the most sensitive subpopulation inherently protects the general population. In this study, the NOAEL was 40 mg/kg bw/day (a.e.), with the above noted effects occurring at a LOAEL of 120 mg/kg bw/day (a.e.). The target MOE was 300, based on application of the standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation) and an extra 3-fold safety factor to account for the severity of the endpoints (qualitative sensitivity to the developing fetus) noted in the presence of maternal toxicity.

For short-term (1 to 7 days) dermal and inhalation exposures to MCPA, an oral study was selected to account for the potential increase in sensitivity to the unborn child of pregnant workers (females 13 to 49 years). Again, protecting the most sensitive subpopulation inherently protects the general population. For females 13 to 49 years, a NOAEL of 40 mg/kg bw/day (a.e.) established in the rat developmental study was used. This was based on decreased viable litter size, increased postimplantation loss, resorptions, hydrocephaly and bent limb bones noted at maternally toxic doses. The target MOE was 300, based on the standard uncertainty factors (10-fold for interspecies variation, 10-fold for intraspecies variation), with an additional 3-fold to account for the severity of endpoint (qualitative sensitivity to the developing fetus) at maternally toxic doses.

To assess 8- to 30-day exposures via dermal (females 13 to 49 years) and inhalation (all populations) routes, a NOAEL of 30 mg/kg bw/day was selected for risk assessment because of effects on maternal body weight at 60 mg/kg bw/day in the rabbit developmental study. The target MOE was 300-fold (10-fold for interspecies variation, 10-fold for intraspecies variation and an additional 3-fold to account for potential

sensitivity in the young noted in the rat reproduction study), which is inherently protective of any potential effects to the young, including the unborn child of a pregnant worker.

For any oral toxicity endpoints that were used, a dermal absorption value of 19% was incorporated into the dermal estimates of exposure (see Section 4.2.1).

As stated in Section 4.1, no toxicological information on the diethanolamine (DEA) form of MCPA was available. As the MCPA Task Force III has indicated it does not support this form of MCPA, the PMRA is proposing to phase out this form (see Section 8.1). Therefore, an occupational risk assessment for MCPA-DEA has not been included at this time.

4.5.2 Exposure and Risk Assessment for Commercial Applicators Mixing, Loading and Applying MCPA to Residential Lawns, Golf Courses and Sod Farm Turf

Commercial applicators have potential for short-term exposure (up to 1 month) to MCPA during use on residential, recreational, golf course and sod farm turf.

Exposure estimates for mixer/loader/applicators were based on data from the PHED Version 1.1 and the ORETF studies.

Exposure is calculated as the product of the unit exposure for a given scenario, the application rate and the area treated per day divided by the body weight. All calculated MOEs for commercial lawn care operators and for golf course and sod farm commercial mixer/loader/applicators wearing long pants, a long-sleeved shirt and gloves are above the target MOE of 300. Further details on the calculations as well as exposure and risk estimates are presented in Appendix II.

4.5.3 Postapplication Exposure and Risk Assessment

Golf course and sod farm workers who re-enter treated sites to conduct turf maintenance activities may have acute and short-term (< 1 week) exposure to MCPA. Potential exposure was estimated using generic agricultural transfer coefficients for workers aerating, fertilizing, mowing, harvesting and transplanting treated turf, coupled with TTR data. A peak residue level of 7.3% of the applied rate was used for the acute risk assessment, and a 7-day average of 0.61% was calculated for the short-term risk assessment (1 to 7 days).

The MOEs for all golf course and sod farm postapplication activities are above the target MOEs for acute (1 day) and short-term (1 to 7 days) exposure. Details are presented in Appendix II.

5.0 Environmental Assessment

In characterizing the environmental risk of MCPA, the PMRA used a deterministic approach which characterizes the risk by the quotient method, in which a risk quotient (RQ) is calculated as the ratio of the expected environmental concentration (EEC) to the toxicity endpoint of concern. RQs less than one are considered as a low risk to non-target organisms, whereas RQs greater than one indicate some degree of risk.

In this assessment, EECs for aquatic and terrestrial ecosystems were based on the maximum label rates in turfgrass. Toxicity endpoints (acute or chronic) were chosen for the most sensitive species and used as surrogates for the range of species that can potentially be exposed following treatment with MCPA.

The results of this deterministic assessment identified various levels of risk to non-target organisms that could be exposed to MCPA.

5.1 Environmental Fate

MCPA exists as several chemical forms. This review addresses the activity of MCPA alone and/or when applied as sodium, potassium or dimethylamine salt (MCPA-Na^+, K^+, or DMA, respectively). These derivatives are considered equivalent to MCPA as they all transform readily to the acid form; this risk assessment is based on the most sensitive endpoint regardless of the derivative.

MCPA, MCPA-DMA and MCPA-Na^+ were very soluble in water at environmental pH levels. At pH 7, the range of aqueous solubilities were 270 to 766 g/L. MCPA-DMA, MCPA-Na^+ as well as MCPA-K^+ transformed readily in aqueous solution to MCPA. MCPA was non-volatile from water and moist soil as indicated by its Henry's law constant (7.46×10^{-5} Pa m^3 mol^{-1}). Neither hydrolysis nor phototransformation were important routes in the transformation of MCPA in soil or in water. Biotransformation in aerobic soil, however, was an important route in the transformation of MCPA (dissipation time 50% [DT_{50}] = 11 to 24 days). Similarly, aerobic biotransformation in aquatic systems (water/sediment) was an important route in the transformation of MCPA (DT_{50} = 8 to 13 days). By contrast, in anaerobic soil and anaerobic aquatic systems (sediment/water), the biotransformation of MCPA was negligible.

Under field conditions, MCPA was non-persistent to slightly persistent in soil (DT_{50} = 3.3 to 22.6 days). MCPA had a low potential for leaching as it was not detected at soil depths greater than 15 cm. MCPA was subject, however, to surface runoff.

5.2 Environmental Toxicology

In terrestrial invertebrates (honeybees and earthworms), MCPA-Na^+ and MCPA-DMA were relatively non-toxic on an acute basis. In the honeybee exposed to MCPA-Na^+, the acute oral median lethal dose (LD_{50}) was 94.4 µg a.e./bee. Similarly, with exposure to

MCPA-DMA, the acute contact no observed effect level (NOEL) was 13 µg a.e./bee. In earthworms, the no observed effect concentration (NOEC) was 100 mg a.e./kg soil with MCPA-DMA.

In birds, MCPA and MCPA-DMA were moderately toxic on an acute oral basis (LD_{50} = 377 to 390 mg a.e./kg bw), and MCPA-DMA was practically non-toxic (median lethal concentration [LC_{50}] > 4589 mg a.e./kg diet) on an acute dietary basis.

In mammals, MCPA and MCPA-DMA were classified as slightly toxic on an acute oral basis (LD_{50} = 653 to 1470 mg a.e./kg bw).

In terrestrial plants, dicots were more sensitive to MCPA and MCPA-DMA than the monocots. Onion was the most sensitive species to seedling emergence (effect concentration at 25% [EC_{25}] = 0.005 kg a.e./ha), and tomato was the most sensitive species in vegetative vigour tests (EC_{25} = 0.006 kg a.e./ha).

The most sensitive aquatic organism was the freshwater floating macrophyte, *Lemna gibba*, with a NOEC and median concentration (EC_{50}) of 13.2 and 124 µg a.e./L, respectively, when exposed to MCPA-DMA. In freshwater fish, MCPA-DMA was practically non-toxic (LC_{50} = 250 mg a.e./L) to slightly toxic (LC_{50} = 96 mg a.e./L), and MCPA-Na^+ was practically non-toxic (LC_{50} = 132 mg a.e./L). In freshwater invertebrates, the most sensitive NOEC was 31 mg a.e./L with MCPA-DMA. The most sensitive endpoint for freshwater algae was the NOEC of 4.7 mg a.e./L with exposure to MCPA-DMA. MCPA was practically non-toxic to marine invertebrates (LC_{50} = 231 mg a.e./L) and marine fish (LC_{50} = 180 mg a.e./L). In marine algae, the most sensitive NOEC was 9.5 mg a.e./L.

5.3 Concentrations in Drinking Water

When assessing residues in drinking water, it is essential to consider all potential sources; thus, the following data regarding MCPA concentrations includes sources from both urban and agricultural areas.

Data from Canadian water monitoring studies in which MCPA was quantified are summarized in Table 5.3.1. The acute exposure value was estimated from monitoring data by determining the 95th percentile of the maximum concentration detected at each site in the individual monitoring studies. 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 for which samples were from potential drinking water sources. The samples with values less than the limit of detection (LOD) were given a value of ½ LOD.

Table 5.3.1 Drinking Water Exposure Concentrations for MCPA

Compound	Groundwater (µg/L)	Surface Water (µg/L)	
		Acute[a]	Chronic[b]
MCPA	N/A	4.225	0.257

[a] 95th percentile of the maximum detected concentrations from surface water or drinking water monitoring studies

[b] 95th percentile of the arithmetic means of all the surface water or drinking water monitoring studies (includes detects and non-detects)

N/A Not available

For this assessment, information was extracted from the available sources, tabulated and sorted into two categories, as follows:

- residues in known drinking water sources; and
- residues in ambient water that may serve as a drinking water source.

Samples were included in the first category if sources were known to describe standard drinking water resources including both ground and surface water. The second category describes water sources (surface and ground) that may potentially be used as drinking water. The data presented here, in many cases, were not accompanied with MCPA use data or the frequency and timing of monitoring in relation to pesticide application and runoff events. Thus, it is likely that higher concentrations of MCPA may be detected.

5.4 Terrestrial Assessment

Terrestrial invertebrates such as earthworms may be exposed to MCPA in the soil, and bees and other beneficial insects may be exposed to spray deposits. Using the EECs based on the maximum application rate (1.7 kg a.e./ha) and the NOEC, it was determined that there would be no acute risk to earthworms as the RQ is 0.008. Similarly, MCPA was shown to be relatively non-toxic to honeybees on an acute exposure basis.

Birds and mammals could be exposed to MCPA by ingesting contaminated food (e.g., seeds, insects or grasses). The assessments for birds and mammals were based on the assumption that animals would be feeding exclusively on contaminated food. In addition, the assessment did not consider avoidance behaviour toward contaminated food as these data were not available.

Based on the acute oral toxicity in the bobwhite quail as well as using standard exposure scenarios that take into account feeding preferences, food consumption rates and body-weight index, it was determined that birds would have to consume contaminated food sources for 14.2 days for their population to be reduced by 50%. For no observable effects on a population, birds would have to consume contaminated food for 1.4 days. As the number of feeding days required for an adverse effect is greater than one, there is a

negligible risk to the bobwhite quail consuming contaminated food sources. In smaller species (American robin and field sparrow), it would require 4 to 5 days of consumption on contaminated food sources for their population to be reduced by 50% and 0.4 to 0.5 days to reach a NOEL. As the number of feeding days required for an adverse effect is less than one, there would be some level of risk to small avian species that consume only MCPA-contaminated food. On an acute dietary basis, the risk quotients (RQ = EEC/NOEC) in bobwhite quail and mallard duck are 0.2 and 0.13, respectively, which indicate a low risk.

Similarly, based on the acute oral toxicity in small mammals (LD_{50} = 665 mg a.e./kg; NOEL = 508 mg a.e./kg) and using standard PMRA exposure scenarios, it was determined that animals would have to consume contaminated food sources for 15.5 days for their population to be reduced by 50% (LD_{50}). For no-observable effects on a population, animals would have to consume contaminated food for up to 11.8 days (NOEL). As the number of feeding days required for an adverse effect is greater than one, the risk to small mammals consuming MCPA-contaminated food sources is negligible. On a chronic dietary basis, the risk quotient (RQ = EEC/NOEC) in the rat is 5.7 and, thus, indicates a moderate risk.

For terrestrial vascular plants, an EC_{25} was used as the endpoint of concern based on the assumption that plants will recover at later stages of growth from an initial 25% inhibition that occurs at earlier stages of growth. As MCPA is applied as a postemergent control of broadleaf weeds in turf, the potential exposure occurs in established non-target plants. Thus, the vegetative vigour endpoint is used as the endpoint of concern. The most sensitive species in vegetative vigour tests was the tomato, where the EC_{25} was 0.006 kg a.e./ha. The EEC is the maximum application rate of 1.7 kg a.e./ha. The RQ (EEC/EC_{25}) is equivalent to 283, which indicates a very high risk to terrestrial plants.

5.5 Aquatic Assessment

For the aquatic risk assessment, the exposure scenarios (EECs) were based on both non-residential uses (e.g., golf courses and sod farms) and residential uses (lawns). For non-residential uses, the exposure scenario for aquatic systems was based on the maximum label rate of MCPA (1.7 kg a.e./ha) applied to a 1-ha pond at water depths of 0.3 m, 1 m or 3 m. For residential uses, the exposure scenario for aquatic systems was based on monitoring data from urban areas (EEC = 0.02 to 8.49 µg/L).

For non-residential and residential uses, MCPA poses no risk to a low risk to aquatic organisms, with the exception of aquatic vascular plants.

In freshwater fish, the RQ is $\leq$ 0.06. Similarly, in marine fish, the RQ is $\leq$ 0.03. Both of these RQs indicate no risk. In freshwater and marine invertebrates, the RQs are $\leq$ 0.02 and $\leq$ 0.07, which indicate no risk. For freshwater algae, there is no risk to a low risk as the RQs are 0.01 to 0.12. In marine algae, there is no risk as the RQ is $\leq$ 0.06. In freshwater vascular plants, the risk is moderate to high as the RQs are 4.4 to 44.

5.6 Environmental Assessment Conclusions

MCPA poses the greatest risk to terrestrial and aquatic plants. There is a high to a very high risk to terrestrial plants exposed to MCPA that enters non-target areas through spray drift. Similarly, with MCPA entering aquatic habitats through spray drift, there is a moderate to high risk to aquatic vascular plants.

A risk was identified in small birds ingesting MCPA-contaminated food. However, to determine the magnitude of this risk, the development of more realistic exposure scenarios is required. The assessment was based on the assumption that birds would be feeding exclusively on MCPA-contaminated food and feeding preference or avoidance behaviour towards contaminated food was not considered.

5.7 Risk Mitigation

MCPA can enter terrestrial habitats and aquatic ecosystems through spray drift. Buffer zones, however, can effectively mitigate the risk to terrestrial and aquatic organisms. Pesticide spray drift from groundboom sprayers was predicted using the data of Nordby and Skuterud (1975). Based on these spray drift predictions and the most sensitive terrestrial species (tomato; EC_{25} = 0.006 kg a.e./ha) and aquatic species (*Lemna gibba*; NOEC = 0.013 mg a.e./L), buffer zones were calculated for mitigating the entry of MCPA into terrestrial and aquatic habitats. This estimation of buffer zones was based on the maximum application rate of 1.7 kg a.e./ha, as indicated in Table 5.7.1.

Table 5.7.1 Buffer Zones to Protect Non-target Terrestrial and Aquatic Environments from a Single Commercial Application of MCPA

Maximum Application Rate (kg a.e./ha)	Terrestrial Buffer Zones (m)[a]	Aquatic Buffer Zones (m)[a]		
		Water Depth (m)		
		≤ 1.0 m	1–3 m	> 3 m
1.7	30	15	5	0

[a] For field sprayers, buffer zones can be reduced by 70% when shrouds are used and by 30% when cones are used.

6.0 Value

As indicated in Section 2.4, the re-evaluation of the lawn and turf uses of MCPA has focussed on the assessment of fine turf (i.e., sports and recreational turf, lawn turf and sod). Sports and recreational turf—including parks, playgrounds, golf courses, zoos, botanical gardens and athletic playing fields—provide enjoyment for users and spectators. Lawn turf is designed principally to serve a decorative function. Lawns include turf planted in or around residences, public and commercial buildings and cemeteries. Utility turf, also known as rough turf, is primarily intended for soil

stabilization and is planted on roadsides, railway rights-of-way, highway rights-of-ways, highway interchanges, airports, hydro, pipeline, wasteland and industrial parks. Turf can also be divided into fine turf and rough turf. Fine turf requires high maintenance and rough turf requires low maintenance. Sports and recreational turf, lawn turf and sod are considered to be fine turf. Utility turf is not included in this assessment.

Hundreds of species of broadleaf weeds can infest turf in Canada, although weed pressure and the types of weeds most likely to become problematic vary from region to region. Experience has shown that most of the broadleaf weed problems in Canadian turf can be attributed to a few weed species. These broadleaf weeds include dandelion, plantain, black medick, chickweed, prostrate knotweed, round-leaved mallow, henbit, ground-ivy (creeping Charlie), wild carrot and white clover. MCPA controls a broad range of broadleaf weeds including plantain, dandelion, chickweed and wild carrot. The application rates of MCPA on turf range from 0.5 to 2.38 kg a.e./ha depending on the turf types and weeds controlled, but the maximum rate of 4 of the 5 end-use products does not exceed 1.5 kg a.e./ha. Products containing MCPA are applied postemergence with groundboom, backpack and handheld sprayers, low-pressure lawn spray guns and hose sprayer (container attached at the end of the hose to spray when watering turf).

MCPA is a chemical that mimics the natural plant hormone indole-3-acetic acid (IAA, also known as auxin). Despite decades of examination and research, the exact mechanism or mechanisms of most auxin and auxinic herbicide-mediated physiological responses in susceptible plants are not fully known. Their primary effects include altered gene expression and enhanced ethylene production. These two effects are likely the beginning of a cascade of events that lead to the biochemical and physiological responses observed. MCPA's modes of action in susceptible plants are likely to produce severe and uncontrolled cell growth that lead to the disintegration of phloem, cortical cells and xylem tissues. When applied at appropriate doses, these herbicides produce an "auxin overload", thereby causing susceptible plants to be injured/controlled. In general, dicot species are much more sensitive to auxinic herbicides (e.g., MCPA) than grasses; therefore, these herbicides have been widely used on turf to give selective of control unwanted broadleaf weeds.

MCPA, although it has certain value on turf, is not a major turf herbicide in Canada. MCPA and 2,4-D have similar efficacy on several important weeds including wild carrot, chickweed, dandelion, ground-ivy, round-leaved mallow, black medick and plantain and, in this sense may be considered equivalent. The actual amount of MCPA products sold for use on turf in Canada is estimated to be less than 5% of the amount of 2,4-D and mecoprop sold.

The MCPA Task Force III supports a reduction of the maximum application rate to 1.7 kg a.e./ha and PMRA accepts this proposal. This new rate will not significantly affect the efficacy of MCPA products. It will also contribute to a reduction in potential exposure and risk to those persons directly and incidentally exposed and will reduce the amount of MCPA in the environment.

7.0 Other Assessment Considerations

7.1 Toxic Substances Management Policy

During this review of the lawn and turf uses of MCPA, the PMRA took into account the federal Toxic Substances Management Policy[3] and followed its Regulatory Directive DIR99-03[4]. Technical grade MCPA does not meet the criteria for TSMP Track 1 substances for the following reasons.

- MCPA is not bioaccumulative. The *n*-octanol–water partition coefficient (K_{ow}) is 0.09–3.9, which is below the TSMP Track 1 cut-off criterion of log $K_{ow} \geq 5.0$.

- MCPA does not meet the criteria for persistence as its half-life values in water (8–13 days) and soil (11–24 days) are below the TSMP Track 1 cut-off criteria for water (≥ 182 days) and soil (≥ 182 days).

7.2 Formulant Issues

Products containing MCPA are subject to all the requirements of Regulatory Directive DIR2004-01, *Formulants Program*, published on 9 January 2004.

Based on the considerations outlined in Section 4.1, the PMRA is proposing that the DEA form of MCPA be phased out (see Section 8.1).

DMA formulations may contain trace levels of N-nitrosodimethylamine (NDMA). Typically, NDMA, if present as a microcontaminant, is at a concentration of less than 1 ppm. Toxicology studies done with these pesticide formulations do not exhibit any of the toxicological findings that are characteristic of NDMA. Also, NDMA is rapidly decomposed by sunlight and, therefore, does not persist in the environment under use conditions. As such, it is unlikely that trace levels of NDMA from pesticide sources would pose a health risk to humans. However, the PMRA will monitor the level of NDMA in certain formulations by requiring registrants to specify NDMA levels in the DMA used for manufacturing purposes (see Section 9.1.2).

---

3 The federal Toxic Substances Management Policy is available through Environment Canada's website at www.ec.gc.ca/toxics

4 Regulatory Directive DIR99-03, *The Pest Management Regulatory Agency's Strategy for Implementing the Toxic Substances Management Policy*, is available through the Pest Management Information Service. Phone: 1 800 267-6315 within Canada or (613) 736-3799 outside Canada (long distance charges apply); Fax: (613) 736-3798; E-mail: pmra_infoserv@hc-sc.gc.ca; or through our website at www.pmra-arla.gc.ca

8.0 Proposed Regulatory Actions

The use of MCPA on residential, recreational and commercial turf is acceptable for continuing registration provided that the mitigation measures described in Section 8.1 are implemented. Standard label precautionary statements and improvements are also required, as presented in Section 8.2.

8.1 Mitigation Measures

1. As the MCPA Task Force III has indicated it does not support the DEA form of MCPA, the PMRA is proposing that MCPA formulations containing DEA be phased out.

2. The maximum application rate of MCPA Amine 500 Liquid Farm Weed Killer, Registration Number 9853, *Pest Control Products Act*, will be reduced to 1.7 kg a.e./ha.

3. A re-entry interval of 1 day is required for harvesting and transplanting treated turf following application on sod farms.

4. Buffer zones are required to protect sensitive terrestrial and aquatic areas, as shown in Section 8.2.2.

8.2 Label Recommendations and Improvements

8.2.1 General

The statement “Keep out of reach of children” must appear on the primary panel of all labels.

The following statement must appear under the “DIRECTIONS FOR USE” section of the label of commercial class products only:

- Do not apply by air.

The following statement must appear under the “DIRECTIONS FOR USE” section of the label of products intended for broadcast application:

- Do not apply more than two broadcast applications per season. This does not include spot treatments.

8.2.2 Label Statements Relating to Health

The label text of Commercial Class products containing MCPA must include the following text.

- **Toxicological Information**

  High concentrations of MCPA may cause severe irritation to the eyes. Symptoms of very high acute exposure to MCPA could include slurred speech, twitching, jerking and spasms, drooling, low blood pressure and unconsciousness. Treat symptomatically.

8.2.3 Label Statements Relating to the Environment

The labels of all products must be amended to include the following statements.

ENVIRONMENTAL HAZARDS

- Toxic to terrestrial plants. This product will harm other broad-leaved plants in the vicinity of the treatment area. If applying this product using a handheld sprayer, do not directly spray or allow the spray to drift onto ornamentals or gardens.
- Do not spray exposed roots of trees and ornamentals.
- Do not contaminate irrigation/drinking water supplies or aquatic habitats by cleaning of equipment or disposal of wastes.
- Do not apply this product if rain is forecasted in the 8 hours following application.
- To reduce runoff from treated areas into aquatic habitats, consider the characteristics/conditions of the site before treatment. Site characteristics/conditions that may lead to runoff include, but are not limited to, heavy rainfall, moderate to steep slope, bare soil, poorly draining soil (e.g., soils that are compacted, fine textured or low in organic matter). Potential for contamination of aquatic areas as a result of runoff may be reduced by including a vegetative strip between the treated area and the edge of the water body. To prevent runoff, avoid spraying on driveways, sidewalks or any other hard surface. Do not irrigate within 24 hours after application.
- The use of this chemical may result in contamination of groundwater particularly in areas where soils are permeable (eg. sandy soil) and/or the depth to the water table is shallow.

In addition, the labels of liquid commercial class products that may be applied by tractor-pulled field sprayers (e.g., to golf courses or sod farms) must include the following statements:

- Buffer Zones

  The buffer zones specified in the table below are required between the point of direct application and the closest downwind edge of sensitive terrestrial habitats (such as grasslands, forested areas, shelter belts, woodlots, hedgerows, pastures, rangelands and shrublands).

Method of Application	Buffer Zones (m) Required for Protection of:			
	Terrestrial Habitats	Aquatic Habitats at Water Depths of:		
Field sprayer	30	< 1 m	1–3 m	> 3 m
		15	5	0

Buffer zones can be reduced by 70% when using shrouds or 30% when using cones.

- Do not apply during periods of dead calm or when winds are gusty.
- When a tank mixture is used, consult the labels of the tank-mix partners and observe the largest (most restrictive) buffer zone of the products involved in the tank mixture.

8.2.4 Label Statements Related to Value

For the product with registration number 9853, *Pest Control Products Act*, a reduction of the maximum rate to 1.7 kg a.e./ha must be reflected in the label, as supported by the MCPA Task Force III.

9.0 Additional Data Requirements

9.1 Data Requirements Relating to Chemistry

9.1.1 Technical Grade Active Ingredient

The PMRA is currently requiring that all label guarantees be expressed as nominal guarantees. As a result of this re-evaluation, technical product labels must be revised to indicate the nominal guarantee value. In addition, the following information is required, if not previously submitted:

- A copy of Statement of Product Specification Form (SPSF) that includes the nominal concentration, lower and upper limits for the active ingredient, the

nominal concentration and upper limits for all impurities present in the product at levels > 0.1%.

- Analytical data for the active and all impurities from 5 recent batches of the technical grade active ingredient to 0.1% to support the SPSF.

- Label revised at the printing time to the nominal guarantee if the data on the SPSF are approved.

9.1.2 All Products to which DMA is Added During the Manufacturing or Formulation Process

An updated Statement Product Specification Form is required for all products to which DMA is added during manufacturing/formulation process. The form must identify the levels of NDMA present in the DMA that is used. This requirement pertains only to products where DMA is added as part of the manufacturing/formulating process; it does not apply to products that use the already manufactured DMA form of MCPA in the formulation process.

9.2 Data Requirements Relating to Toxicology

The PMRA has accounted for uncertainties associated with some studies considered in the risk assessment through uncertainty/safety factors. During this re-evaluation, the following confirmatory data were identified as requirements to refine the risk assessment:

- a multigeneration rat reproduction study (Wistar), including developmental neurotoxicity endpoints (DACO 4.5.1); and

- long-term oncogenicity study with rats (Wistar) (DACO 4.4.2).

More recently, additional data were submitted by the MCPA Task Force III to address the potential for sensitivity of the young. These data will be fully assessed during the re-evaluation of MCPA for agricultural use, at which time a determination will be made on whether a new multigeneration rat reproduction is still required.

9.3 Data Requirements Relating to Occupational, Residential and Bystander Exposure

ORETF data were used in this assessment. All registrants must either gain access to the data of the ORETF or provide equivalent data.

10.0 Proposed Re-evaluation Decision

The PMRA has assessed the available information and has concluded that the use of MCPA and associated end-use products to treat lawns and turf does not entail an unacceptable risk of harm to human health or the environment, provided the mitigation measures in this document are adopted. Standard precautionary statements and label improvements are also required.

The PMRA will accept written comments on this proposal up to 60 days from the date of publication of this document to allow interested parties an opportunity to provide input into the proposed decision.

List of Abbreviations

2-EHE	2-ethylhexyl ester
ADI	acceptable daily intake
a.e.	acid equivalent
a.i.	active ingredient
ARfD	acute reference dose
ARI	aggregate risk index
bw	body weight
CAS	Chemical Abstracts Service
DACO	data code
DEA	diethanolamine
DEEM	Dietary Exposure Evaluation Model
DMA	dimethylamine
DT_{50}	time required for 50% dissipation
DWLOC	drinking water level of comparison
EC_{25}	effect concentration 25%
EC_{50}	median effect concentration
EEC	expected environmental concentration
IAA	indole acetic acid
IUPAC	International Union of Pure and Applied Chemistry
kg	kilogram
K_{ow}	*n*-octanol–water partition coefficient
LC_{50}	median lethal concentration
LD_{50}	median lethal dose
LOAEL	lowest observed adverse effect level
LOD	limit of detection
LOQ	level of quantitation
m	metre
MCPA	(4-chloro-2-methylphenoxy) acetic acid
MCPP	mecoprop
mg	milligram
MOE	margin of exposure
N/A	not available
NDMA	N-nitrosodimethylamine
NOAEL	no observed adverse effect level
NOEC	no observed effect concentration
NOEL	no observed effect level
ORETF	Occupational and Residential Exposure Task Force
Pa	Pascal
PDI	potential daily intake
p*K*a	dissociation constant
PHED	Pesticide Handlers Exposure Database
PMRA	Pest Management Regulatory Agency
RI	risk index
RQ	risk quotient

SF	safety factor
$T_{1/2}$	half-life
TC	transfer coefficient
TTR	turf transferable residue
TWA	time-weighted average
TSMP	Toxic Substances Management Policy
UF	uncertainty factor
USEPA	United States Environmental Protection Agency

Appendix I List of Products Containing MCPA Registered for Use on Turf as of 5 May 2005

Registration Number	Product Name	Registrant	Class
9858	MCPA Sodium 300 Herbicide	United Agri Products Canada Inc.	Commercial
9516	MCPA Amine 500 Herbicide	United Agri Products Canada Inc.	Commercial
9853	MCPA Amine 500 Liquid Farm Weed Killer	Dow AgroSciences Canada Inc.	Commercial
15729	Wilson MCPA Amine 500 Liquid Weed Killer	Nu-Gro IP Inc.	Commercial
13570	Later's Creeping Buttercup Weed Killer	Nu-Gro IP Inc.	Domestic

Appendix II Risk Assessment of Human Exposure: Details of Calculations

Table 1 Homeowner Mixer/Loader/Applicator: Short-term Exposure Estimates and Margins of Exposure

Application Equipment	Data Source[a]	Formulation/ Rate	Area treated (ha/day)	Dermal Unit Exposure (µg/kg handled)	Dermal Exposure (µg/kg/day)[b]	Inhalation Unit Exposure (µg/kg handled)	Inhalation Exposure (µg/kg/day)[c]	Dermal MOE [d]	Inhalation MOE [d]	Combined MOE
Residential Lawns: Homeowner wearing short-sleeved shirt, short pants, no gloves									**Target MOE = 300**	
Low-pressure handwand/ handpump	ORETF	Liquid (1.7 kg a.e./ha)	0.2	82 741	76.4	24	0.12	524	343 137	523
			0.01		3.8		0.01	10 477	6 862 745	10 461
Ready-to-use hose-end sprayer	ORETF		0.2	6875	6.3	32.2	0.16	6305	255 754	6153
			0.01		0.3		0.01	126 091	5 115 090	123 057
Dial-type hose-end sprayer	ORETF		0.2	21 525	19.9	35.6	0.17	2014	231 328	1996
			0.01		1		0.01	40 273	4 626 570	39 925
Backpack[e]	PHED		0.2	10 149	9.4	62.1	0.3	4271	132 613	4137
			0.01		0.5		0.02	85 415	2 652 269	82 750

a Median unit exposures are used from ORETF. Best-fit unit exposures are used from PHED.

b A dermal penetration factor of 19% was used. Where dermal exposure µg/kg/day = (unit exposure × area treated × use rate [expressed as acid equivalents]) / 70 kg bw. 70 kg bw and corresponding body surface area (18 440 cm^2) used for both males and females, as calculated exposure is similar to estimates for females alone (62 kg bw and surface area of 16 597 cm^2).

c Where inhalation exposure µg/kg/day = (unit exposure × area treated × use rate [expressed as acid equivalents]) / 70 kg bw. 70 kg bw and corresponding body surface area (18 440 cm^2) used for both males and females, as calculated exposure is similar to estimates for females alone (62 kg bw and surface area of 16 597 cm^2).

d Based on an oral NOAEL of 40 mg/kg/day; target MOE is 300 for the acid equivalent forms, based on severity of endpoint.

e The backpack application clothing scenario is short pants, short-sleeved shirt and gloves (no non-gloved data); The USEPA SOPs state that this PHED data is not completely applicable for application to lawns.

Table 2 Postapplication Exposure Estimates and Margins of Exposure for Adults and Toddlers

Scenario		Data Source	Dermal Exposure (µg/kg/d)[a]	Oral Exposure (µg/kg/d)			Dermal MOE	Oral MOE
				Hand-to-Mouth[b]	Turf Mouthing[c]	Ingestion of Soil[d]		
Adult Target MOE: Acute and short-term dermal = 300								
1.7 kg a.e./ha	Acute	USEPA SOP 1997, 2001a (shorts/ T-shirt)	97.68	N/A	N/A	N/A	410	N/A
	Short-term		8.2	N/A	N/A	N/A	4884	N/A
Toddler Target MOE: Acute and short-term dermal = 100; acute and short-term oral = 300								
1.7 kg a.e./ha	Acute	USEPA SOP 1997, 2001a (shorts/ T-shirt)	163.4	45.3	1.4	0.08	893[f]	3118[f]
	Short-term		72.1	4.53	0.14	0.08	1386[g]	6315[h]

[a] Based on a dermal penetration factor of 19% where applicable. Dermal exposure = % TTR × rate in µg/cm^2 × TC × duration / bw (70 kg for adults, 15 kg for toddlers). 70 kg bw and corresponding body surface area (18 440 cm^2) used for both males and females, as calculated exposure is similar to estimates for females alone (62 kg bw and surface area of 16 597 cm^2). TTR values are based on the TTR study and normalized for Canadian rates. TCs are 14 500 and 5200 cm^2/hour for adults and children, respectively. Exposure duration is 2 hours. TTR values = 7.3% for acute and 0.612% for short-term scenarios.

[b] Based on 20 hand-to-mouth events per hour, a surface area of 20 cm$^{2.}$, TTR 10% acute and 1% short-term, saliva extraction factor (SEF) of 50%. Exposure = DFR × SA × hand-to-mouth events × SEF × duration/15 kg bw.

[c] Based on an ingestion of 25 cm^2 turf/day and SEF of 50%. Exposure = DFR × 25 × SEF/15 kg bw.

[d] Based on an ingestion of 0.1 g soil/day, depth of 1 cm, 100% available/cm soil, 0.67 cm^3/g soil weight to volume conversion factor. Exposure = rate × 0.1 × 0.67 × 1/15 kg bw.

[e] Based on an oral NOAEL of 40 mg/kg/day (target MOE is 300 for acid equivalent forms, based on severity of effects).

[f] Based on an acute oral LOAEL of 146 mg/kg/day (target MOE is 300 for acid equivalent forms, since a NOAEL was not available).

[g] Based on a dermal NOAEL of 100 mg/kg/day (target MOE is 100 for acid equivalent forms).

[h] Based on an oral NOAEL of 30 mg/kg/day (target MOE is 100 for acid equivalent forms).

Table 3 Postapplication Exposure Estimates and Margins of Exposure for Golfers

Scenario			Dermal Exposure (µg/kg/d)[a]	Dermal MOE
1.7 kg a.e./ha	**Adults**			
	Acute	Liquid	6.7	5938 [b]
	Short-term	Liquid	0.56	70 823 [b]
	Adolescents			
	Acute	Liquid	10.7	13 622 [c]
	Short-term	Liquid	4.73 [d]	21 146 [d]

[a] Based on a dermal penetration factor of 19% where applicable. Dermal exposure = % TTR × rate of 17 µg/cm^2 × TC × duration / bw (70 kg for adults, 44 kg for adolescents). The acute TTR value is 7.3%; the short-term TTR value is 0.612% based on the TTR study. TC is 500 cm^2/hr based on generic transfer coefficients for turf. Duration is 4 hours.

[b] Based on an oral NOAEL of 40 mg/kg/day (target MOE is 300 for acid equivalent forms based on severity of effects).

[c] Based on a oral LOAEL of 146 mg/kg/day (target MOE is 300 for acid equivalent forms).

[d] The dermal exposure was calculated without considering dermal absorption and the MOE is based on a dermal NOAEL of 100 mg/kg/day (target MOE is 100 for acid equivalent forms).

Table 4 Short-term Aggregate Exposure Estimates and Risk Index Values

Age Group	Scenario	Food (µg/kg/day)[a] (risk index)	Application on Turf (µg/kg/day) (risk index)		Postapplication on Turf (µg/kg/day) (risk index)		Aggregate Risk Index[d] (excluding drinking water)	DWLOC (µg/L)[e]
			Dermal[b]	Inhalation[c]	Dermal[b]	Oral[c]		
Adults 62 kg	Low-pressure sprayer—broadcast turf	0.077 (1732)	76.36 (2)	0.12 (1140)	8.19 (16)	N/A	2	1506
	Low-pressure sprayer—spot turf	0.077 (1732)	3.82 (35)	0.01 (22 222)	N/A	N/A	34	4012
	Dial-type sprayer—broadcast turf	0.077 (1732)	19.86 (7)	0.17 (771)	8.19 (16)	N/A	5	3256
	Dial-type sprayer—spot turf	0.077 (1732)	0.99 (135)	0.01 (14 815)	N/A	N/A	124	4100
	Backpack—broadcast turf	0.077 (1732)	9.37 (14)	0.30 (442)	8.19 (16)	N/A	7	3577
	Backpack—spot turf	0.077 (1732)	0.47 (284)	0.02 (8889)	N/A	N/A	237	4116
	Golfing (liquid)	0.077 (1732)	N/A	N/A	0.56 (238)	N/A	209	4114
Youth 44 kg	Golfing (liquid)	0.127 (2362)	N/A	N/A	4.73 (211)	N/A	194	6566
Toddler 15 kg	Broadcast turf (liquid)	0.203 (1478)	N/A	N/A	72.13 (14)	4.75 (63)	11	4101

[a] Based on chronic dietary exposure estimates generated using DEEM.

[b] A use rate of 1.7 kg a.e./ha was used for all application scenarios. Risk index (RI) values were calculated using a dermal NOAEL of 100 mg/kg/day and a UF/SF of 100 for children and all adults, except for females 13–49 years, and an oral NOAEL of 40 mg/kg/day for females 13–49 years with a dermal absorption factor of 19% and a UF/SF of 300 for acid equivalent forms.

[c] Risk index values were calculated using an oral NOAEL of 30 mg/kg/day and a UF/SF of 100 for children and all adults, except for females 13–49 years, and an oral NOAEL of 40 mg/kg/day is used for females 13–49 years with a UF/SF of 300 for acid equivalent forms.

[d] As MOEs could not be calculated for combined food, oral, inhalation and dermal (application and postapplication) exposure (different NOAELs and target MOEs), an aggregate risk index (ARI) was calculated using the following equation: $ARI = 1/ (1/RI_{food} + 1/RI_{oral} + 1/RI_{inhalation} + 1/RI_{dermal(app)} + 1/RI_{dermal [postapp]})$. If the ARI exceeds 1, the risk is below the level of concern.

[e] DWLOC = maximum allowable exposure from drinking water × body weight ÷ drinking water consumption rate, where the maximum exposure from water is $(1 - 1/ARI_{(excluding\ drinking\ water)})$ and the drinking water consumption rate is 2 L/day for adults and 44-kg children and 1 L/day for 15-kg toddlers (USEPA 2001).

Table 5 Commercial Mixer/Loader/Applicator: Short-term Exposure Estimates and Margins of Exposure

Application Equipment	Data Source[a]	Application Rate (kg a.e./ha)	Area Treated (ha/day)	Dermal Unit Exposure (µg/kg handled)	Dermal Exposure (µg/kg/ day)[b]	Inhalation Unit Exposure (µg/kg handled)	Inhalation Exposure (µg/kg/ day)[c]	Short-term Exposure[d]		
								Dermal MOE	Inhalation MOE	Combined MOE
Commercial lawn care operator wearing long pants, long-sleeved shrit, gloves										
Low-pressure turf gun	ORETF	1.7	2	785	7.24	4	0.19	4141	154 412	4033
Backpack[e]	PHED		0.4	5446	10.05	62.1	0.6	2985	49 730	2816
Low-pressure handwand	PHED		2	943	8.7	45.2	2.2	3447	13 665	2753
			0.4	943	1.74	45.2	0.44	22 084	68 324	16690
Groundboom	PHED		30	83.63	11.58	2.6	1.89	2591	15 837	2227

a Median unit exposures are used from ORETF, Best-fit unit exposures are used from PHED.

b Where dermal exposure µg/kg/day = (unit exposure × area treated × use rate [expressed as acid equivalents])/70 kg bw.

c Where inhalation exposure µg/kg/day = (unit exposure × area treated × use rate [expressed as acid equivalents])/70 kg bw.

d Based on an oral NOAEL of 30 mg/kg/day; target MOE is 300 based on severity of effects for all acid equivalent forms.

e The USEPA SOPs state that the PHED backpack data is not completely applicable for application to lawns.

Table 6 Postapplication Exposure Estimates and Margins of Exposure for Golf Course and Sod Farm Workers

Scenario		Transfer Coefficient	TTR Data[a] % TTR	Dermal Exposure Absorbed (µg/kg/d)[b]	Dermal MOE[c]	Re-entry Interval (day)
1.7 kg a.e./ha	**Golf Courses/Sod Farms**: Aerating, fertilizing, pruning, scouting, mowing					
	Acute	500	7.3	13.47	2969	0
	Short-term	500	0.61	1.13	35 412	0
	Sod Farms: harvesting, transplanting					
	Acute [d]	16500	7.3	444.63	90	0
			0.89	54.46	734	1
	Short- term	16500	0.61	37.28	1073	0

[a] Chemical-specific data from turf transferable residue study; acute TTR = 7.30%, 7 day TWA = 0.612%; Day 1 TTR = 0.89%.

[b] Incorporate a dermal penetration factor of 19% based on available data. Dermal exposure = % TTR × rate of 17 µg/cm^2 × TC × 8-hour duration / 70 kg bw. 70 kg bw and corresponding body surface area (18 440 cm^2) used for both males and females, as calculated exposure is similar to estimates for females alone (62 kg bw and surface area of 16 597 cm^2).

[c] Based on an acute and short-term oral NOAEL of 40 mg/kg/day; target MOE is 300 for the acid equivalent forms.

[d] For re-entry exposure on the day of application (day 0) the calculated MOE was less than the target MOE. Based on a 1-day re-entry interval, MOEs were above the target.

Appendix III Bibliography

A list of additional information regarding MCPA is included below for those interested in further information. However, this is limited to a selection of published studies and does not include references to all proprietary data used in this assessment.

Barney, W.P. 1998. Determination of Transferable Turf Residues on Turf Treated with 2,4-D, 2,4-DP-p, MCPA, MCPP-p and Dicamba. Broadleaf Turf Herbicide Transferable Foliar Residue Task Force LLC. Study Number BTH TFR TF 001. Unpublished.

Barney, W.P. 1998. Determination of Transferable Turf Residues on Turf Treated with 2,4-D DMA + MCPP-p DMA + Dicamba DMA in Various Spray Volumes. Broadleaf Turf Herbicide Transferable Foliar Residue Task Force LLC. Study Number BTH TFR TF 200. Unpublished.

Camann, D.E., T.K. Majumadar, H. Harding, W.D. Ellenson and R.G. Lewis. 1996. Transfer Efficiency of Pesticides from Carpet to Saliva-Moistened Hands, in Measurement of Toxic and Related Air Pollutants: Proceeding of an International Specialty Conference. Publication VIP-64, Air & Waste Management Association, Pittsburgh, PA, 1996, p.532–540.

Camann, D.E., T.K. Majumadar and P. Geno. 1995. Determination of Pesticide Removal Efficiency from Human Hands Wiped with Gauze Moistened with Three Salivary Fluids, Final Report to the USEPA by ManTech under Contract 68-D5-0049.

Clothier, J. 2000. Dermal Transfer Efficiency of Pesticides from Turf Grass to Dry and Wetted Palms. Prepared for the USEPA, Research Triangle Park, NC, under Contract 68-D5-0049.

Hughes, D.L. and D.D. Bomkamp. 2000. Determination of Transferable Turf Residues on Turf Treated with 2,4-D DMA, MCPA DMA, 2,4-D DMA + MEPP-p DMA + Dicamba DMA and MCPA DMA + MEPP-p DMA + 2,4-DP-p DMA. Covance Laboratories Inc. Study Number BTH TFR TF 003/6926-105. Unpublished.

Johnson, D., R. Thompson and B. Butterfield. (ORETF). 1999. Outdoor Residential Pesticide Use and Usage Survey and National Gardening Association Survey. Doane Marketing Research Inc., St. Louis, MO. Gallup Organization Inc: Burlington, VT.

Klonne, D., S. Artz and D.L. Merricks. 1999. A Generic Evaluation of Homeowner Exposure Associated with Liquid Pesticide Handling and Hose-End Application (Ready-to-Use and Ortho DIAL'n SPRAY) to Residential Lawns ORETF Study No. OMA004, Extracted from D.R. Klonne, 10 October 1999. *Integrated Report for Evaluation of Potential Exposure to Homeowners and Professional Lawn Care Operators Mixing, Loading, and Applying Granular and Liquid Pesticides to Residential Lawns,* Study Number OM005, Vols, 1 and 6 of 6, p. 1736–2213. OMA004.

Klonne, D.R. and R. Honeycutt. 1999. A Generic Evaluation of Homeowner Exposure Associated with Granular Turf Pesticide Handling and Application to Residential Lawns.

H.E.R.A.C. Inc., and Outdoor Residential Exposure Task Force. Study Number OMA003. Unpublished.

Leibold, E., H.D. Hoffman, and B. Holdebrand. 1998. 14C-Dicamba-DMA Salt - Study of the Dermal Absorption in Rats. Reg. Doc. #98-10140.

Lu, C., and R. Fenske. 1999. Dermal Transfer of Chlorpyrifos Residues from Residential Surfaces: Comparison of Hand Press, Hand Drag, Wipe and Polyurethane Foam Roller Measurements after Broadcast and Aerosol Pesticide Applications. *Environmental Health Perspectives*. 107(6): 463–7.

Makary, M.H., J.C. Street and R.P. Sharma. 1986. Parmacokinetics of dicamba isomers applied dermally to rats. *Pesticide Biochemistry and Physiology*. 25: 258–263.

Merricks, D.L. 1997. Carbaryl Mixer/Loader/Applicator Exposure Study during Application of RP-2 Liquid (21%), Sevin® Ready to Use Insect Spray or Sevin® 10 Dust to Home Garden Vegetables. Agrisearch Incorporated (Frederick, MD) and Rhone-Poulenc Ag Company (Sponsor), Research Triangle Park, NC, and Morse Laboratories, Inc., Sacramento, CA. Agrisearch Study No. 1519; Rhone-Poulenc Ag No. 10564. Unpublished.

Merricks, L., D. Klonne and S. Artz. 1999. Exposure of Professional Lawn Care Workers During the Mixing, Loading, and Application of Granular Turf Pesticides Utilizing a Surrogate Compound, Agrisearch Inc. (Frederick, MD) and Ricerca, Inc. (Painesville, OH). Laboratory report numbers 3701 (Agrisearch) and 69331-96-0182-CR-001 (Ricerca). OMA001. Unpublished.

Merricks, L., D. Klonne and L. Smith. 1999. Exposure of Professional Lawn Care Workers During the Mixing and Loading of Dry and Liquid Formulations and the Liquid Application of Turf Pesticides Utilizing a Surrogate Compound, Agrisearch Inc. (Frederick, MD) and Morse Laboratories Inc. (Sacramento, CA). Study numbers 3702 (Agrisearch) and ML96-0656-ORE (Morse). OMA002.Unpublished.

National Toxicology Program. 1992. The immunotoxicity of diethanolamine (CAS No. 111-42-2) in female Fischer 344 rats. NTP Report No. IMM20303. United States Public Health Service, Department of Health and Human Services, National Institutes of Health, Research Triangle Park, NC.

National Toxicology Program. 1992. The immunotoxicity of diethanolamine (CAS No. 111-42-2) in female B6C3F1 mice. NTP Report No. IMM98011. United States Public Health Service, Department of Health and Human Services, National Institutes of Health, Research Triangle Park, NC.

National Toxicology Program. 1994. Technical report on toxicity studies of diethanolamine (CAS No. 111-42-2) administered topically and in drinking water to F344/N rats and B6C3F1 mice. (NIH Publication No. 92-3343). *Journal of Applied Toxicology*. 14(1):1–19.

National Toxicology Program. 1997. Technical report on the toxicology and carcinogenesis studies of diethanolamine (CAS No. 111-42-2) in F344/N rats and B6C3F1 mice. NTP TR 478, NIH Publ. No. 97-3968. United States Public Health Service, Department of Health and Human Services, National Institutes of Health, Research Triangle Park, NC.

National Toxicology Program. 2001. Technical report on the toxicology and carcinogenesis studies of coconut oil acid diethanolamine condensate (CAS No. 68603-42-9). NTP TR 479 Pub. No 01-3969. United States Public Health Service, United States Department of Health and Human Services, National Institutes of Health, Research Triangle Park, NC.

Nordby, A. and R. Skuterud. 1974. The effects of boom height, working pressure and wind speed on spray drift. *Weed Research*. Volume 14, p. 385–395.

United States Environmental Protection Agency. 1997. Draft Standard Operating Procedures (SOPs) for Residential Exposure Assessments. Contract No. 68-W6-0030. Work Assignment No 3385.102. Residential Exposure Assessment Work Group. 18 December 1997. Washington, DC

United States Environmental Protection Agency. 2000. Agricultural Transfer Coefficients. Science Advisory Council for Exposure. Policy No. 003.1. Unpublished.

United States Environmental Protection Agency. 2001. Recommended Revisions to the Standard Operating Procedures (SOPs) for Residential Exposure Assessments. Policy No. 12. 22 February 2001.

United States Environmental Protection Agency. 2001. *Pesticide Science Policy. Standard Operating Procedure (SOP) for Incorporating Screening-Level Estimates of Drinking Water Exposure into Aggregate Risk Assessments*. Public Comment Draft. Office of Pesticide Programs. 1 September 2001.

United States Environmental Protection Agency. 2004. *MCPA; Availability of Risk Assessments*. Federal Register. Docket ID OPP-2004-015. Available online at http://www.epa.gov/EPA-PEST/2004/June/Day-23/p14093.htm

United States Environmental Protection Agency. 2004. *Reregistration Eligibility Decision for MCPA*. List A, Case 0017. Available online at http://www.epa.gov/oppsrrd1/REDs/mcpa_red.pdf

Yeary, R.A. and J.A. Leonard. 1993. Measurement of Pesticides in Air During Application to Lawns, Trees and Shrubs in Urban Environments. In: K. Racke and A. Leslie (eds.). American Chemical Society Symposium Series 552: *Pesticides in Urban Environments: Fate and Significance*. Chapter 23, p. 275–281.