Evaluation of the new active
QUINCLORAC
in the product
Drive Herbicide

Australian Pesticides and Veterinary Medicines Authority

August 2005

Canberra
Australia
FOREWORD

The Australian Pesticides and Veterinary Medicines Authority (APVMA) is an independent statutory authority with responsibility for assessing and approving agricultural and veterinary chemical products prior to their sale and use in Australia.

In undertaking this task, the APVMA works in close cooperation with advisory agencies, including the Department of Health and Ageing (Office of Chemical Safety), Department of Environment and Heritage (Risk Assessment and Policy Section), and State departments of agriculture and environment.

The APVMA has a policy of encouraging openness and transparency in its activities and of seeking community involvement in decision making. Part of that process is the publication of public release summaries for all products containing new active ingredients and for all proposed extensions of use for existing products.

The information and technical data required by the APVMA to assess the safety of new chemical products and the methods of assessment must be in accordance with accepted scientific principles. Details are outlined in the APVMA’s publications Manual of Requirements and Guidelines (MORAG)

This Public Release Summary is intended as a brief overview of the assessment that has been completed by the APVMA and its advisory agencies. It has been deliberately presented in a manner that is likely to be informative to the widest possible audience thereby encouraging public comment.

More detailed technical assessment reports on all aspects of the evaluation of this chemical can be obtained by completing the order form in the back of this publication and submitting with payment to the APVMA. Alternatively, the reports can be viewed at the APVMA Library First Floor, 22 Brisbane Avenue, Barton, ACT.

The APVMA welcomes comment on the usefulness of this publication and suggestions for further improvement. Comments should be submitted to the Program Manager Pesticides, Australian Pesticides and Veterinary Medicines Authority, PO Box E240, Kingston ACT 2604.
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LIST OF ABBREVIATIONS AND ACRONYMS

ac  active constituent
ADI  Acceptable Daily Intake (for humans)
AHMAC Australian Health Ministers Advisory Council
ai  active ingredient
BBA  Biologische Bundesanalstalt fur Land – und forstwirschaft
bw  bodyweight
CRP Chemistry and Residues Program
d  day
DAT Days After Treatment
DM Dry matter
DT₅₀ Time taken for 50% of the concentration to dissipate
E₅₀ concentration at which the biomass of 50% of the test population is impacted
EC₅₀ concentration at which 50% of the test population are immobilised
EEC Estimated Environmental Concentration
E₅₀ concentration at which the rate of growth of 50% of the test population is impacted
EUP End Use Product
Fo original parent generation
g  gram
GAP Good Agricultural Practice
GCP Good Clinical Practice
GLP Good Laboratory Practice
GVP Good Veterinary Practice
h  hour
ha  hectare
Hct  Heamatocrit
Hg  Haemoglobin
HPLC High Pressure Liquid Chromatography or High Performance Liquid Chromatography
id  intradermal
im  intramuscular
ip  intraperitoneal
IPM Integrated Pest Management
iv  intravenous
in vitro outside the living body and in an artificial environment
in vivo inside the living body of a plant or animal
kg  kilogram
Kₒc  Organic carbon partitioning coefficient
L  Litre
LC₅₀ concentration that kills 50% of the test population of organisms
LD₅₀ dosage of chemical that kills 50% of the test population of organisms
LOD Limit of Detection – level at which residues can be detected
LOQ Limit of Quantitation – level at which residues can be quantified
mg  milligram
mL  millilitre
MRL Maximum Residue Limit
MSDS Material Safety Data Sheet
NDPSC National Drugs and Poisons Schedule Committee
ng  nanogram
NHMRC National Health and Medical Research Council
NOEC/NOEL No Observable Effect Concentration Level
OC  Organic Carbon
OM  Organic Matter
po  oral
<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>POEM</td>
<td>Predictive Operator Exposure Model (UK)</td>
</tr>
<tr>
<td>ppb</td>
<td>parts per billion</td>
</tr>
<tr>
<td>PPE</td>
<td>Personal Protective Equipment</td>
</tr>
<tr>
<td>ppm</td>
<td>parts per million</td>
</tr>
<tr>
<td>Q-value</td>
<td>Quotient-value</td>
</tr>
<tr>
<td>RBC</td>
<td>Red Blood Cell Count</td>
</tr>
<tr>
<td>s</td>
<td>second</td>
</tr>
<tr>
<td>sc</td>
<td>subcutaneous</td>
</tr>
<tr>
<td>SC</td>
<td>Suspension Concentrate</td>
</tr>
<tr>
<td>SUSDP</td>
<td>Standard for the Uniform Scheduling of Drugs and Poisons</td>
</tr>
<tr>
<td>TGA</td>
<td>Therapeutic Goods Administration</td>
</tr>
<tr>
<td>TGAC</td>
<td>Technical grade active constituent</td>
</tr>
<tr>
<td>T-Value</td>
<td>A value used to determine the First Aid Instructions for chemical products that contain two or more poisons</td>
</tr>
<tr>
<td>µg</td>
<td>microgram</td>
</tr>
<tr>
<td>vmd</td>
<td>volume median diameter</td>
</tr>
<tr>
<td>WG</td>
<td>Water Dispersible Granule</td>
</tr>
<tr>
<td>WHP</td>
<td>Withholding Period</td>
</tr>
</tbody>
</table>
INTRODUCTION

This publication provides a summary of the data reviewed and an outline of the regulatory considerations for the proposed registration of the product DRIVE HERBICIDE, which contains the new active constituent quinclorac. The product is proposed to be used for the post-emergence control of summer grass and white clover and suppression of kikuyu in turf.

Responses to this Public Release Summary will be considered prior to registration of the product. They will be taken into account by the Australian Pesticides and Veterinary Medicines Authority (APVMA) in deciding whether the product should be registered and in determining appropriate conditions of registration and product labelling.

Copies of full technical evaluation reports on quinclorac, covering toxicology, occupational health and safety aspects, environmental aspects are available from the APVMA on request (see order form on last page). They can also be viewed at the APVMA library located at the APVMA offices, First Floor, 22 Brisbane Avenue, Barton ACT 2604.

Written comments should be received by the APVMA by 18 September 2005. They should be addressed to:

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Applicant
BASF Australia

Product Details
It is proposed to register DRIVE HERBICIDE containing 750g/kg of quinclorac as a water dispersible granule. The product will be imported fully formulated and packaged in 250 and 500g, as well as 1, 2 and 5kg HDPE bottles.

DRIVE HERBICIDE is a member of the Carboxylic acid group of herbicides and has the disruptors of plant cell growth mode of action. For weed resistance management DRIVE HERBICIDE is a Group I herbicide.

The rate of product use is 1.1 kg/ha. DRIVE HERBICIDE is proposed for registration in all states.

Formulations containing quinclorac are currently registered overseas, mainly in rice and turf.
CHEMISTRY AND MANUFACTURE

Quinclorac is a member of carboxyl acid group of herbicides for post emergence control of summer grass and white clover in turf. The active constituent will not be imported into Australia as the product will be formulated in USA.

Active constituent

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common name (ISO)</td>
<td>quinclorac</td>
</tr>
<tr>
<td>IUPAC name</td>
<td>3,7-Dichloroquinoline-8-carboxylic acid</td>
</tr>
<tr>
<td>CAS Number</td>
<td>84087-01-4</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>242.07</td>
</tr>
<tr>
<td>Molecular formula</td>
<td>C_{10}H_{5}Cl_{2}NO_{2}</td>
</tr>
<tr>
<td>Physical form</td>
<td>white yellow powder</td>
</tr>
<tr>
<td>Odour</td>
<td>odourless</td>
</tr>
<tr>
<td>Melting point</td>
<td>271^0C</td>
</tr>
<tr>
<td>Density</td>
<td>1.630 g/cm^3</td>
</tr>
<tr>
<td>Vapour pressure at 25^0C</td>
<td>$4.10^{-12}$ mbar/hPa</td>
</tr>
<tr>
<td>Structural formula</td>
<td><img src="image" alt="Structural formula" /></td>
</tr>
</tbody>
</table>

The Chemistry and Residues Program (CRP) of the APVMA has evaluated the chemistry aspects of the quinclorac active constituent (manufacturing process, quality control procedure, batch analysis results and analytical methods) and found them to be acceptable.

Formulated product

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Product name</td>
<td>Drive Herbicide</td>
</tr>
<tr>
<td>Formulation type</td>
<td>Water Dispersible Granule</td>
</tr>
<tr>
<td>Active constituent concentration</td>
<td>750g/kg</td>
</tr>
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</table>

Physical and Chemical Properties

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical state</td>
<td>Granular</td>
</tr>
<tr>
<td>Colour</td>
<td>light tan</td>
</tr>
<tr>
<td>PH</td>
<td>4.0</td>
</tr>
<tr>
<td>Bulk density</td>
<td>595g/L</td>
</tr>
</tbody>
</table>
Storage and Stability

Stability data for 2 weeks at 54°C were provided for DRIVE HERBICIDE stored in polyethylene bottles. The results for the active content was within the expiry specifications, and there was a slight decrease (0.3%) at the completion of the trial. The results provided for water content, pH, wet sieve test, particle size distribution, suspensibility, dispersibility, flowability and dust content were only slightly changed and remain within the product specification limits. The accelerated storage stability results indicate that the product should remain within specifications for at least 2 years when stored under normal conditions.

Packaging

Drive Herbicide will be packaged in 250 and 500g as well as 1, 2 and 5kg HDPE bottles with screw caps. The packaging is not adversely affected by the product, nor is the product unstable in the packaging. The packaging details are acceptable.

Recommendation

The chemistry and residues program (CRP) has evaluated the chemistry and the manufacturing aspects of DRIVE HERBICIDE in data submitted by applicant to support their application for registration. The CRP is satisfied that the chemistry requirements of Section 14(5) Agricultural and Veterinary Chemicals Code have been met.
Evaluation of Toxicology
The toxicological database for quinclorac containing primarily of toxicological studies conducted in laboratory animals is quite extensive. In interpreting the data, it should be noted that toxicity tests generally use doses that are high compared with likely human exposures. The use of high doses increases the likelihood that potentially significant toxic effects will be identified. Findings of adverse effects in any one species do not necessarily indicate such effects may occur in humans. From a conservative risk assessment perspective, however, adverse findings in animal species are assumed to represent potential effects in humans, unless convincing evidence of species specificity is available. Where possible, considerations of the species-specific mechanisms of adverse reactions weigh heavily in the extrapolation of animal data to likely human hazard. Similarly, consideration of the risks to human health must take into account the likely human exposure levels compared with those, usually many times higher, which produce adverse effects in animal studies. Toxicity tests should also indicate dose levels at which the specific toxic effects are unlikely to occur. Such dose levels as the No-Observable-Effect-Level (NOEL) are used to develop acceptable limits for dietary or other intakes (ADI and ARfD) at which no adverse health effects in humans are expected.

Toxicokinetics and Metabolism Studies
Following oral dosing in rats, quinclorac was rapidly and almost completely absorbed. The maximum plasma levels were attained in about 30 minutes, which then declined rapidly. The maximum concentrations were observed in plasma followed by the kidneys and blood. The major route of elimination was via urine followed by faeces. Bile appeared to be a minor route of excretion after a small single oral dose (15 mg/kg bw), but was more significant following a single large dose (600 mg/kg bw). About 91-96% and 0.7-4% of the administered dose was excreted in the urine and faeces, respectively, in 120 h after dosing. Of these, approximately 80-98% was eliminated via urine, mostly unchanged in 24 h. Absorption, distribution and elimination of quinclorac was independent of the sex and dose administered, and was not affected by pre-treatment of the animals. Two minor metabolites were detected in the urine. One of the metabolites accounted for about 2-5% of the administered dose, and was identified as the glucuronic acid conjugate of quinclorac, whilst the second metabolite that represented about 2-4% of the administered dose, was not identified. Repeated dietary administration of quinclorac resulted in similar kinetics.

Acute Studies
Quinclorac has low acute oral (LD$_{50}$ >5000 mg/kg bw in mice and LD$_{50}$ of 2680 mg/kg bw in rats), dermal (LD$_{50}$ >2000 mg/kg bw in rats) and inhalational toxicity (LC$_{50}$ >5170 mg/m$^3$ in rats). It was a slight eye irritant, but not a skin irritant in rabbits. Quinclorac was a skin sensitisers in guinea pigs when tested under the conditions of the maximisation test. Clinical signs observed in rat acute oral toxicity studies with quinclorac included, dyspnoea, apathy, staggering, spastic gait, piloerection, dehydration, lacrimation or poor general health.

Drive Herbicide, containing 750 g/L quinclorac is of low acute oral (LD$_{50}$ >2000 mg/kg bw in rats), dermal (LD$_{50}$ >2000 mg/kg bw) and inhalational (LC$_{50}$ >5000 mg/m$^3$) toxicity in rats. It was a slight eye and skin irritant in rabbits, but was not a skin sensitisers in guinea pigs when tested using the Buehler method.
**Short-term Studies**

Quinclorac in the diet was administered to mice at 0, 1000, 4000, 8000 and 16000 ppm for 4 weeks. A slight reduction in food consumption was seen in females at 16000 ppm during the final study week. Treatment-related alterations in several study parameters were observed at 16000 ppm. These included depressions in bodyweight in both sexes, an increase in ALP activity in both sexes, an increase in ALT activity and, an increase in liver weight in males and decreases in kidney weight in both sexes, and histopathological abnormalities in the liver. The NOEL was 8000 ppm (equal to approximately 2048 mg/kg bw/d).

Quinclorac in the diet was administered to rats at concentrations of 0, 100, 400, 1600 and 6400 ppm for 4 weeks. There were no treatment-related effects on any of the study parameters tested in the study.

In another short-term study, rats received quinclorac technical 15000 or 30000 ppm in the diet for 4 weeks. One male animal at 30000 ppm died after 26 days on study. Clinical signs such as impaired general health, dehydration and ruffled fur were noted at and above 15000 ppm. Food consumption was decreased at 15000 and 30000 ppm. Body weight was depressed at 30000 ppm, reaching levels below the initial values. At 15000 ppm, there were body weight deficits at termination. Perturbations noted in some haematological and clinical chemistry parameters were restricted to the 30000 ppm group. Pathology revealed depressions in liver and testes weights in males at 30000 ppm, together with histological findings of renal tubular atrophy, testicular tubular atrophy, splenic lymphocyte depletion, cloudy swelling of hepatocytes, and vacuolisation of the adrenal cortex. Renal tubular atrophy was also seen in 2 males and a female at 15000 ppm. A NOEL was not achieved in this study.

In a 4-week study, dogs received quinclorac in the diet at concentrations of 0, 1000, 3000, 9000 or 27000 ppm. The mean intake of quinclorac was equal to approximately 30, 95, 278 and 912, and 36, 108, 314 and 905 mg/kg bw/d in males and females, respectively. Vomiting was seen in all animals at 27000 ppm on the first day after feeding, and occasionally on other days thereafter. Treatment-related depressions in food consumption occurred at 27000 ppm. At termination, the group mean body weights of both sexes at this dose level were depressed relative to controls. AP activity was depressed at and above 9000 ppm. Testes weight was decreased at 27000 ppm. Histopathology showed chronic inflammatory foci in the interstitial tissue in the kidney at 27000 ppm, with no degenerative or regenerative changes in the tubular epithelium. The NOEL was 3000 ppm (95 mg/kg bw/d), based on decreased ALP activity at and above 9000 ppm (278 mg/kg bw/d).

In a dermal toxicity study, rabbits received quinclorac at 0, 40, 200 or 1000 mg/kg bw on a clipped, intact skin area on the back for 6 h/d, 7 days per week for at least 21 days under semi-occlusive conditions. Body weight gain was slightly reduced in females at 1000 mg/kg bw/d. Organ weight data showed a significant increase in kidney weight in males at 1000 mg/kg bw/d. Based on increased kidney weight in males at 1000 mg/kg bw/d, the dermal NOEL was 200 mg/kg bw/d.

**Subchronic Studies**

In a 92 –93-day study, mice received quinclorac in the diet at 0, 4000, 8000 or 16000 ppm. Calculated daily intake of the test substance for M/F was equal to 0, 1000/1465, 2048/2734 and 4555/5952 mg/kg bw/d, respectively. No treatment-related mortalities or clinical signs were observed. Water consumption was increased at and above 8000 ppm. Body weight gain was significantly depressed in both sexes at 8000 and 16000 ppm. Both sexes at 4000 ppm showed a reduction in body weight gain, being statistically significant in females. Treated males showed decreases in monocyte and eosinophil counts. In males, an elevation in ALT activity at 16000 ppm, and significant increases in plasma urea levels at and above 8000 ppm were noted. Based on statistically significant reduction in body weight gain at and above 4000 ppm, no NOEL could be established for this study.
Because no NOEL could be established in the preceding study, a further study was conducted in mice with the dietary levels of 0 or 500 ppm, administered for 92-93 days. The calculated daily intake values of the test substance for M/F were equal to 0 and 85/130 mg/kg bw/d, respectively. No treatment-related effects were observed in any of the parameters tested in the study. The NOEL was 500 ppm (85 and 135 mg/kg bw/d for males and females, respectively).

In a 3-month study, rats received quinclorac in the diet at 0, 1000, 4000 or 12000 ppm. Calculated daily intake of the test substance for M/F was equal to 0, 78/86, 302/358 and 929/1035 mg/kg bw/d, respectively. There were no treatment-related mortalities or clinical signs. Food consumption was depressed, whilst the water consumption was elevated in both sexes at 12000 ppm. Both sexes at 12000 ppm showed decreased body weight gain. Statistically significant differences were noted in some RBC parameters at 12000 ppm. Females at 12000 ppm showed increases in monocyte and neutrophil counts, together with a reduction in lymphocyte counts. AST activity was elevated in males at 12000 ppm. At necropsy, small cortical scars or fine granular areas on the renal cortex were noted in 3 males at 12000 ppm. Histopathology of these animals and two additional rats at this dose group (1/sex) showed an increased incidence of minimal to slight, focal, chronic interstitial nephritis. Based on decreased body weight gain, increased AST activity, and an elevated incidence of focal chronic interstitial nephritis in males at 12000 ppm, the NOEL was 4000 ppm (302 and 358 mg/kg bw/d for males and females, respectively).

**Chronic/Carcinogenicity Studies**

Quinclorac in the diet was administered to mice at concentrations of 0, 1000, 4000 or 8000 ppm for 78 weeks, including a satellite group that was maintained over a 6-month feeding period. Mean intake of quinclorac during the study was equal to 0, 170, 710, 1443 and 0, 220, 898, 1890 mg/kg bw/d for males and females, respectively. There were no treatment-related effects on the survival of the test animals. However, statistically significant body weight depressions were observed in both sexes at all dietary levels throughout the study. Histopathology did not reveal any treatment-related effect on tumour incidence. Based on significant decreases in body weights in all treated groups, a NOEL could not be established.

Because, no NOEL was established in the preceding study, another 78-week feeding study was conducted subsequently in mice with the dose levels of 0 and 250 ppm, administered for the same treatment duration. No treatment-related effects were observed in any of the parameters studied and the findings showed no evidence for test substance-related carcinogenicity. The NOEL was 250 ppm (41 mg/kg bw/d).

In a 2-year rat study, quinclorac in the diet was administered to rats at 0, 1000, 4000, 8000 or 12000 ppm, including 2 satellite groups maintained at the above dietary levels for 12 or 24 months. Calculated mean intake of the test substance was equal to 0, 57, 215, 443, 675 and 0, 66, 262, 528, 831 mg/kg bw/d for males and females at 0, 1000, 4000, 8000 and 12000 ppm, respectively. There were no treatment-related mortalities or clinical signs. Females at 12000 ppm showed depressions in body weights relative to controls during the second year. Histopathology did not reveal any treatment-related effect on tumour incidence. Based on significant decreases in body weights in 12000 ppm females, the NOEL was 8000 ppm (443 mg/kg bw/d).

Dogs received quinclorac at 0, 1000, 4000 or 12000 ppm in the diet for 12 months (calculated mean intake of the test substance was 0, 35, 139, 489 and 0, 35, 141, 472 mg/kg bw/d for males and females respectively). No treatment-related mortalities or clinical signs were observed in the study. Food conversion efficiency was markedly depressed in both sexes at 12000 ppm, and in males at 4000 ppm. Treatment-related body weight depressions were recorded at 12000 ppm reaching statistical significance.
in males from study week 70 onwards, and leading to body weight deficits at termination in males and females. A slight anaemia was seen at 12000 ppm. Clinical chemistry showed a dose-related decrease in plasma creatinine level in both sexes at and above 4000 ppm. Liver weights were increased in both sexes at 12000 ppm. Kidney weights showed increases at and above 4000 ppm. Histopathology revealed an increase in focal mononuclear infiltrates in the liver, and hydropic degeneration in the kidney of both sexes at 12000 ppm. Based on decreased plasma creatinine concentrations, and increased absolute and/or relative kidney weight in both sexes at and above 4000 ppm, the NOEL was 1000 ppm (35 mg/kg bw/d).

Reproduction Studies
In a 2-generation reproductive toxicity study, rats received quinclorac in the diet at 0, 1000, 4000 or 12000 ppm commencing from at least 70 days pre-mating throughout all phases. Mean daily intake of the test substance was equal to 87, 343 and 1026 mg/kg bw/d, respectively. Food consumption was depressed in females at 12000 ppm. Body weight was depressed in parents of both generations at 12000 ppm. There were no treatment-related effects on any of the reproductive parameters investigated. An increased incidence of focal or multi-focal chronic non-purulent interstitial nephritis was observed in 12000 ppm females of both generations. Neonatal toxicity characterised by reduced pup survival, weight gain, and retarded growth was observed at 12000 ppm. The NOEL for reproductive toxicity was >12000 ppm (1026 mg/kg bw/d). The NOEL for maternal and neonatal toxicity was 4000 ppm (343 mg/kg bw/d), based on decreased food consumption and body weight gains and chronic nephritis in parents, and decreased survival, body weight gain and retarded growth in pups at 12000 ppm (1026 mg/kg bw/d).

Developmental Studies
Quinclorac was administered once daily to pregnant rats at 0, 24.4, 146 or 438 mg/kg bw/d by po gavage on days 6 through 15 post-coitum. Three mortalities occurred at 438 mg/kg bw/d. There were no treatment-related clinical signs. Food consumption was significantly depressed, but water consumption was elevated at 438 mg/kg bw/d. Dams at 438 mg/kg bw/d lost body weight during the first 2 days of treatment. Animals that died or were sacrificed at this dose level showed severe ulcerations in the glandular stomach. Statistically significant increase in foetal body weight was observed in all treated groups. There were no external, soft tissue or skeletal alterations that could be attributed to treatment. The NOEL for maternotoxicity was 146 mg/kg bw/d, based on mortality, decreased food consumption, increased water consumption and necropsy findings at 438 mg/kg bw/d. Because there were no treatment-related effects on any of the developmental toxicity parameters tested, the NOEL for developmental toxicity was >438 mg/kg bw/d.

Quinclorac was administered once daily to pregnant rabbits at 0, 70, 200 or 600 mg/kg bw/d by po gavage on days 7 through day 19 post insemination. There were 6 mortalities at 600 mg/kg bw. Treatment-related clinical signs were observed at 600 mg/kg bw/d, and included reduced or no defecation, diarrhoea, apathy and/or poor general health. Food consumption was depressed at 200 and 600 mg/kg bw/d. Body weights were depressed at 600 mg/kg bw/d during treatment and post-treatment periods. Body weight gain data showed deficits at 200 and 600 mg/kg bw/d. Necropsy showed watery faeces in the large intestine, thickened content and ulceration in the stomach, implantations that were not consistent with the stage of pregnancy, pale coloured renal cortex and liver, and a statistically significant decrease in uterine weight at 600 mg/kg bw/d. There was an increase in the number of dead implantations at 600 mg/kg bw/d. Foetal weight was significantly reduced at 600 mg/kg bw/d. There were no external, soft tissue or skeletal alterations attributable to treatment. Embryo/foeto-toxicity characterised by reduced foetal weight, increased post-implantation loss and foetal growth retardation were noted at the maternotoxic dose level of 600 mg/kg bw/d. Therefore, the NOEL for developmental toxicity was 200 mg/kg bw/d. Based on reduced food consumption and weight gain in dams at 200 mg/kg bw/d, the NOEL for maternotoxicity was 70 mg/kg bw/d.
Genotoxicity Studies

The genotoxicity of quinclorac has been examined in a battery of in vitro genotoxicity studies including the Ames test, HGPRT mutation test and unscheduled DNA synthesis assay in rat hepatocytes, and in vivo micronucleus and unscheduled DNA synthesis assays. An in vitro chromosome aberration assay conducted with human lymphocytes was the only assay, which yielded positive results, as reflected by an increase in the number of aberrant metaphases at concentrations showing clear cytotoxicity. Quinclorac is unlikely to be genotoxic in vivo.

PUBLIC HEALTH STANDARDS

Poisons Scheduling

The National Drugs and Poisons Schedule Committee (NDPSC) considered the toxicity of the product and its active ingredients.

On the basis of its toxicity, the NDPSC has included quinclorac in Schedule 5 of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP). There are provisions for appropriate First-Aid Instructions and Safety Directions on the product label.

NOEL/ADI

The Acceptable Daily Intake (ADI) is that quantity of an agricultural compound, which can safely be consumed on a daily basis for a lifetime and is based on the lowest NOEL obtained in the most sensitive species. This NOEL is then divided by a safety factor, which reflects the quality of the toxicological database and takes into account the variability in responses between species and individuals.

The ADI for quinclorac was established at 0.3 mg/kg bw/d based on a NOEL of 35 mg/kg bw/d in the 1-year dog study. A 100-fold safety factor was used to derive this ADI in recognition of the extensive toxicological database available for quinclorac.

Acute Reference Dose (ARfD)

The acute reference dose (ARfD) is the maximum quantity of an agricultural or veterinary chemical that can safely be consumed as a single, isolated, event. The ARfD is derived from the lowest single or short term dose which causes no effect in the most sensitive species of experimental animal tested, together with a safety factor which reflects the quality of the toxicological database and takes into account the variability in responses between species and individuals.

The ARfD is 2.0 mg/kg bw based on a NOEL of 200 mg/kg bw in an acute toxicity study in mice, using a 100-fold safety factor.
RESIDUES ASSESSMENT

At this point in time, the applicant only seeks use on turf. As such, no assessment of quinclorac residues in food was considered necessary at this time.
ASSESSMENT OF OVERSEAS TRADE ASPECTS OF RESIDUES IN FOOD

At this point in time, the applicant only seeks use on turf. As such, no assessment of trade aspects of quinclorac residues in food was considered necessary.
OCCUPATIONAL HEALTH AND SAFETY ASSESSMENT

Health hazards
Quinclorac has very low oral, low dermal and very low inhalational toxicity in rats. It was a slight eye irritant, but not a skin irritant in rabbits. Quinclorac was a skin sensitiser in guinea pigs when tested in the maximisation test. Quinclorac is listed on the NOHSC List of Designated Hazardous Substances, with a risk phrase R43.

Drive Herbicide is a dry flowable formulation. Drive Herbicide is of low oral and dermal and very low inhalational toxicity in rats. It was a slight eye and skin irritant in rabbits, but was not a skin sensitiser in guinea pigs when tested using the Buehler method. Drive Herbicide has been classified as a hazardous substance, according to NOHSC Approved Criteria for Classifying Hazardous Substances, with risk phrases R38 and R43.

Formulation, packaging, transport, storage and retailing
BASF Australia Ltd intends to import the product into Australia fully formulated. Drive Herbicide will be packaged in 250 g, 500 g, 1 kg, 2 kg, and 5 kg high density polyethylene containers. Transport workers, store persons and retailers will handle the packaged product and could become contaminated if the packaging were breached.

Use and exposure
Drive Herbicide is indicated for the post-emergence control of summer grass and white clover and the suppression of Kikuyu in turf. The draft label specifies that Drive Herbicide will be applied by boom spray and knapsack applications. The recommended maximum application rate is 1.1 kg/ha, in a minimum spray volume of 400 L/ha (0.21% quinclorac, 0.28% EUP).

Workers may be contaminated with the product during mixing/loading and application, cleaning up spills, cleaning and maintaining equipment, and at re-entry. The main routes of exposure to the product are dermal, ocular and inhalation.

Drive Herbicide is a skin irritant and possibly a skin sensitiser. It is expected that spray concentration (0.28% EUP) would not cause any skin irritation/sensitisation. Therefore, cotton overalls buttoned to the neck and wrist (or equivalent clothing) and a washable hat and elbow-length PVC gloves, are recommended when opening the container and preparing spray to protect against skin irritation/sensitisation.

There were no worker exposure studies on quinclorac or Drive Herbicide available for assessment. Therefore, an exposure model (the Pesticide Handler Exposure Database Surrogate Exposure Guide) was used to estimate repeated worker exposure to Drive Herbicide during mixing/loading and ground application.

These estimates in conjunction with toxicology data demonstrated that the use of clothing and gloves during mixing/loading/application is required to protect workers during acute and repeated exposure.

Entry into treated areas
Workers entering treated areas can be exposed to product residues, photodegradates and degradation products during crop management activities.
Using the US Occupational Post-Application Risk Assessment Calculator (US Policy 003.1) and based on the toxicity profile and use pattern of Drive Herbicide, OCS (OHS) concluded that workers re-entering treated areas will not be at risk. Therefore, OCS (OHS) does not recommend a re-entry statement.

**Recommendations for safe use**

Users should follow the instructions and Safety Directions on the product label. Safety Directions include the use of cotton overalls buttoned to the neck and wrist (or equivalent clothing) and a washable hat, and elbow-length PVC gloves when opening the container, preparing spray and using the prepared spray.

**Information provision**

*Material Safety Data Sheet (MSDS)*

BASF Australia Pty Ltd has produced a MSDS for Drive Herbicide. This should contain information relevant to Australian workers, as outlined in the NOHSC National Code of Practice for the Preparation of MSDS. Employers should obtain the MSDS from the supplier and ensure that their employees have ready access to it.

**Conclusion**

The registration of quinclorac in Drive Herbicide at 750 g/kg as a dry flowable formulation, for use on turf, is supported. Drive Herbicide can be used safely if handled in accordance with the instructions on the product label and any other control measures described above. Additional information is available in the product MSDS.
ENVIRONMENTAL ASSESSMENT

Environmental exposure
Drive Herbicide is to be applied to established turf (but not to golf course collars and greens) of various species (common couch, hybrid couch, Japanese lawngrass, marine couch, perennial ryegrass and Kentucky bluegrass) at 1.1 kg/ha in 400 L/ha water when weeds are actively growing. The target weeds are summer grass, kikuyu (suppression only) and white clover. No more than four sprays will be made each year. The product is intended for professional boom spray application by turf managers to high quality turf areas such as sporting fields, golf courses and bowling greens.

Drive must not be applied if heavy rain is expected within 48 hours. Treated turf must not be irrigated until at least 8 hours after application. Following application, residues are expected to enter soil, where they are likely to be mobile. Hydrolysis and photolysis are not expected to be significant routes of degradation for quinclorac. Laboratory testing indicates that quinclorac is stable in aquatic systems under aerobic and anaerobic conditions, with half-lives of more than a year. Similarly, quinclorac persists in laboratory soils, with half-lives in excess of 6 months for microbial metabolism and photodegradation. Two slowly formed metabolites, 2-hydroxyquinclorac and quinclorac methyl ester, were identified at low levels (<20% of applied) in soil metabolism studies.

Quinclorac sorbs weakly to laboratory soils and is likely to leach. Volatilisation is not expected to be a significant route of dissipation from soil and water for quinclorac, as it has high water solubility and low volatility.

Field observations differ from laboratory expectations. For example, there are reports of atmospheric transport from rice growing areas in the US, with consequent damage to sensitive tomato plants. It appears that, under some circumstances, significant quantities of quinclorac’s methyl ester could be volatilised from soils and/or water. Atmospheric transport can be expected, as quinclorac is relatively resistant to degradation by tropospheric hydroxyl radicals, with an estimated half-life of about a week.

Field dissipation proceeds more rapidly than in the laboratory, with typical first half-lives (DT50s) of a week to a month and DT90s often less than 6 months. Even so, quinclorac can be persistent in some soils, with DT50s of 6 months or more and DT90s well in excess of a year. Dissipation is strongly retarded in cold winter soils. The rapid initial dissipation of quinclorac appears to largely reflect metabolism, with both metabolites identified in the field, but the extent to which the methyl ester may volatilise has not been investigated. Quinclorac residues have been detected to 75 cm beneath turf and to 120 cm in bare ground plots. Quinclorac is not expected to bioconcentrate in fish because of its water solubility. This has been confirmed experimentally.

Environmental effects
Testing indicates that quinclorac is a typical water-soluble herbicide in being practically non-toxic to birds and aquatic organisms but highly toxic to some terrestrial plants. Testing in bobwhite quail and mallard ducks returned acute NOELs of at least 1000 mg/kg body weight, and chronic NOELs of at least 500 mg/kg diet. The nominal NOECs in rainbow trout, bluegill sunfish and sheepshead minnow were 100 mg/L (the highest concentration tested). The most sensitive aquatic invertebrate tested was mysid shrimp with an EC50 of 67 mg/L. The EC50s in green and blue-green algae and in duckweed were above 100 mg/L. Quinclorac is practically non-toxic to terrestrial invertebrates such as bees and lacewings, but may be moderately toxic to earthworms. Reproduction of predatory mites was impaired by quinclorac.
residues on glass plates. Some crop plants were affected by quinclorac in laboratory assays, with tomatoes generally most sensitive. The NOEC in tomatoes was 1.4 g/ha for phytotoxicity ratings, and 5.6 g/ha for plant dry weight.

**Environmental risk assessment**

Application of quinclorac to shallow water (15 cm) at 825 g/ha would leave residues of 0.55 mg/L. Even in this artificial situation of direct overspray, the estimated environmental concentration is two orders of magnitude below the most sensitive endpoint (LC50 of 67 mg/L to mysid shrimp) for aquatic fauna.

Quinclorac may be expected to have greater effects on algae and aquatic plants because it is a herbicide. However, the estimated environmental concentration is well below the most sensitive E<sub>B</sub>C10 of 2.41 mg/L for duckweed. Given that surface waters are extremely unlikely to be directly oversprayed, the risk that quinclorac will affect aquatic life when used on turf is minimal.

Estimated residues of quinclorac on vegetation and in soil after application of Drive Herbicide are well below levels that could be toxic to birds, bees or earthworms.

The application rate for Drive Herbicide is 1.1 kg/ha (825 g/ha quinclorac). The main risk arising from its use in turf is its potential to cause phytotoxicity in nontarget plants. For example, the NOEC for phytotoxic symptoms in tomatoes during their early growth is 1.4 g/ha, or 0.17% of the application rate. The basic drift values developed for field crops indicate that spray drift (90<sup>th</sup> percentile) will fall below 0.17% at a distance of 15-20 m from the sprayed area. Assessment indicates that a buffer of less than 5 m would be protective based on the NOEC of 5.6 g/ha (0.68% of application rate) for plant dry weight. Given that the phytotoxic effects reported in tomato seedlings did not lead to death of the plants, that native flora will likely be protected by a thick or waxy cuticle, and that native vegetation does not generally abut high-value turf areas, it does not appear likely that use of Drive Herbicide on turf will lead to significant off-target damage to native vegetation.

The risk that spray drift may damage nontarget plants is further reduced by the prohibition on the draft label of application under weather conditions, or from spraying equipment, that may cause spray to drift onto nearby susceptible plants/crops, cropping lands or pastures.

The basic drift values reflect droplet rather than vapour transport. The likelihood of damage to native vegetation arising from vapour transport of quinclorac’s methyl ester does not appear high as Drive Herbicide will be used sporadically in small areas of turf, rather than extensively across broad agricultural landscapes. Aerobic soil metabolism studies indicate that the methyl ester remains below 10% of applied in soil, and is therefore unlikely to be emitted in large quantities from treated turf.

Runoff could also expose nontarget plants to quinclorac. The draft label prohibits application if heavy rain is expected within 48 hours, or within 8 hours prior to irrigation.

Another potential route for off-target transport of quinclorac is the management of clippings when turf is mown. The draft label advises that clippings from the first three mowings should be left on the treated area.

There is a risk that quinclorac will contaminate groundwater, given its hydrophilicity and identification as a probable leacher. The magnitude of any such contamination is difficult to predict, but likely to be limited given that quinclorac will only be applied to small areas of high-value turf. The season of use (summer/autumn) will limit any groundwater contamination, as warmer soil temperatures will favour
degradation of quinclorac. Users of Drive Herbicide need to be alerted to the likelihood that quinclorac may contaminate groundwater.
EFFICACY AND SAFETY ASSESSMENT

Adequacy of efficacy data

The proposed uses of the product are postemergence control of summer grass and white clover and the suppression of kikuyu in turf. Twelve efficacy trials were conducted across five Australian states and over three seasons. Two phytotoxicity studies were also conducted.

Trial design (controls, treatments, replicates)
All experiments appear appropriately designed (Randomised Complete Block) with adequate replication and controls.

Analysis of trial data, interpretation
All trial data is appropriately recorded and statistically analysed. Interpretation of the data provided is consistent for most of the studies but inconsistent for some.

Trial validation, location, date
The trials were conducted over a three year period. Trials were conducted throughout Queensland, New South Wales, Western Australia and Victoria under a range of different environmental/climatic conditions.

Phytotoxicity
Two phytotoxicity studies were undertaken on 26 warm season turf grasses and results recorded across 2 rates of herbicide. These were summarised and are accurately reflected in the label recommendations.

Safety to non-target species
Relevant information provided. Appropriate warnings in relation to susceptible turf species and inappropriate uses eg ornamental beds

Conclusion
The data as presented were adequate to demonstrate the efficacy and crop safety aspects of the product Drive Herbicide. It is recommended that on the basis of efficacy and crop safety, the product Drive Herbicide be considered for registration.
LABELLING REQUIREMENTS

CAUTION
KEEP OUT OF REACH OF CHILDREN
READ SAFETY DIRECTIONS BEFORE OPENING OR USING

DRIVE®
HERBICIDE

ACTIVE CONSTITUENT: 750 g/kg QUINCLORAC

For the post-emergence control of summer grass and white clover and the suppression of Kikuyu in turf as specified in the DIRECTIONS FOR USE table.

CONTENTS: 250 g, 500 g, 1 kg, 2 kg, 5 kg

• ® = Registered trademark of BASF
**DIRECTIONS FOR USE:**

**RESTRAINTS:**

- Clippings from the first three mowings should be left on the treated area.
- Do not apply into any ornamental bed.
- Do not apply when weeds are not actively growing.
- Do not apply if heavy rain is expected within 48 hours, or within 8 hours prior to irrigation.
- Do not apply this product through any type of irrigation system.
- Do not apply to golf course collars or greens.
- Do not apply within 4 weeks after seedling emergence of Kentucky bluegrass, creeping bentgrass and perennial ryegrass.

<table>
<thead>
<tr>
<th>SITUATION</th>
<th>WEEDS CONTROLLED</th>
<th>RATE</th>
<th>CRITICAL COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Established turf of: Common couch (<em>Cynodon dactylon</em>), hybrid couch (<em>Cynodon dactylon x C. transvaalensis</em>), Japanese lawngrass (<em>Zoysia japonica</em>), marine couch (<em>Sporobolus virginicus</em>), perennial ryegrass (<em>Lolium perenne</em>) bent (<em>Agrostis stolonifera</em>), Kentucky Bluegrass (<em>Poa pratensis</em>)</td>
<td>Summer grass (<em>Digitaria spp.</em>)</td>
<td>1.1 kg/ha in 400 L water/ha</td>
<td>One application: Weeds at cotyledon stage up to prior to second tiller. OR Weeds at 5 tillers and greater. Two applications, 21 to 28 days apart: Weeds from 2 to 4 tillers. The addition of a crop oil adjuvant may improve weed control in unfavourable weather conditions.</td>
</tr>
<tr>
<td></td>
<td>Kikuyu (<em>Pennisetum clandestinum</em>) (Suppression only)</td>
<td></td>
<td>For Kikuyu suppression, apply twice, 14 to 21 days apart. Best results are gained from Autumn applications. The addition of a crop oil adjuvant may improve weed control in unfavourable weather conditions.</td>
</tr>
<tr>
<td></td>
<td>White clover (<em>Trifolium repens</em>)</td>
<td></td>
<td>Apply to actively growing weeds.</td>
</tr>
</tbody>
</table>

**NOT TO BE USED FOR ANY PURPOSE, OR IN ANY MANNER, CONTRARY TO THIS LABEL UNLESS AUTHORISED UNDER APPROPRIATE LEGISLATION.**
GENERAL INSTRUCTIONS
DRIVE will provide post-emergence control of summer grass and clover in turf. Quinclorac is rapidly absorbed through the foliage and translocated throughout the plant. Death of sensitive weeds usually occurs between 1 and 2 weeks after treatment. Kikuyu can be suppressed by applying two post-emergence treatments. Best results will be achieved if kikuyu is treated in Autumn.

HERBICIDE RESISTANCE WARNING

DRIVE Herbicide is a member of the Carboxylic acid group of herbicides and has the Disruptors of Plant Cell Growth mode of action. For weed resistance management this product is a Group I herbicide. Some naturally occurring weed biotypes resistant to DRIVE and other disruptors of plant cell growth herbicides may exist through normal genetic variability in any weed population. The resistant individuals can eventually dominate the weed population if these herbicides are used repeatedly. These resistant weeds will not be controlled by DRIVE or other Group I herbicides. Since the occurrence of resistant weeds is difficult to detect prior to use, BASF Ltd accepts no liability for any losses that may result from the failure of DRIVE to control resistant weeds.

• MIXING
To ensure even mixing, half-fill the spray tank with clean water and add the required amount of product. Agitate thoroughly, then add the remainder of the water. Agitate again before spraying commences.

The addition of a crop oil adjuvant may improve weed control in unfavourable weather conditions

• APPLICATION
Apply DRIVE to actively growing weeds at the growth stage indicated in the Directions for Use table. Apply the product in 400 L of water/ha. Do NOT apply through any type of irrigation system. Avoid overlaps during application. Separate applications should be made if all target weeds are not at the correct growth stage for treatment at the same time. It is recommended not to mow 2 days before or after applying DRIVE to maximize weed control and minimize turf injury. Refer to the Restraints listed above the Directions for Use table for further application advice.

• COMPATIBILITY
The product is compatible with DSMA, Primo* 250 EC and Primo Maxx.

• PROTECTION OF CROPS, NATIVE AND OTHER NON-TARGET PLANTS
DO NOT apply under weather conditions, or from spraying equipment, that may cause spray to drift onto nearby susceptible plants / crops, cropping lands, pastures or native vegetation. Quinclorac has properties and characteristics associated with chemicals detected in groundwater. The use of this product where soils are permeable, particularly where the water table is shallow, may result in groundwater contamination.

DO NOT apply to Bahia grass (Paspalum notatum), centipedegrass (Eremochloa ophiuroides), buffalo grass (Stenotaphrum secundatum), Kikuyu (Pennisetum clandestinum), Qld blue Couch (Digitaria didactyla), carpet grass (Axonopus spp.), or lawns or turf where desirable clovers are present. DO NOT apply to ornamental beds or to exposed feeder roots of trees and ornamentals. Be particularly careful within the drip line of these plants. DO NOT apply to golf course collars or greens. Clippings from the first three mowings should be left on the treated area.

• PROTECTION OF WILDLIFE, FISH, CRUSTACEANS AND ENVIRONMENT
DO NOT contaminate streams, rivers or waterways with the chemical or used containers.

• STORAGE AND DISPOSAL
Store in the closed, original container in a cool, well-ventilated area. Do not store for prolonged periods in direct sunlight. Triple or preferably pressure rinse containers before disposal. Add rinsings to spray tank. Do NOT dispose of undiluted chemicals on-site. If recycling, replace cap and return clean container to recycler or designated collection point. If not recycling, break, crush, or puncture and bury empty containers in a local authority landfill. If not available, bury the containers below 500 mm in a disposal pit specifically
marked and set up for this purpose clear of waterways, desirable vegetation and tree roots. Empty containers and product should NOT be burnt.

- **FIRST AID**
  If poisoning occurs contact a doctor or Poisons Information Centre (131126). Additional information is listed in the Material Safety Data Sheet.

- **SAFETY DIRECTIONS**
  Will irritate the eyes and skin. Avoid contact with eyes and skin. When opening the container, preparing spray, and using the prepared spray, wear cotton overalls buttoned to the neck and wrist (or equivalent clothing) and a washable hat, and elbow length PVC gloves. Wash hands after use. After each day’s use wash gloves and contaminated clothing.

- **MSDS**
  Additional information is listed in the Material Safety Data Sheet.

- **CONDITIONS OF SALE**
  All conditions and warranties rights and remedies implied by law or arising in contract or tort whether due to the negligence of BASF Australia Ltd or otherwise are hereby expressly excluded so far as the same may legally be done provided however that any rights of the Buyer pursuant to non excludable conditions or warranties of the Trade Practices Act 1974 or any relevant legislation of any State are expressly preserved but the liability of BASF Australia Ltd or any intermediate Seller pursuant thereto shall be limited if so permitted by the said legislation to the replacement of the goods sold or the supply of equivalent goods and all liability for indirect or consequential loss or damage of whatsoever nature is expressly excluded. This product must be used or applied strictly in accordance with the instructions appearing hereon. This product is solely sold for use in Australia and must not be exported without the prior written consent of BASF Australia Ltd.

FOR SPECIALIST ADVICE IN AN EMERGENCY ONLY
PHONE 1 800 803 440
TOLL FREE - ALL HOURS - AUSTRALIA WIDE

APVMA Approval No.: 57935 /
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® = Registered trademark of BASF
* = Other trademarks
Product number

Batch Number:
Date of Manufacture:
Website: www.agro.basf.com.au
Fax on Demand: 0500 544 044
Label version: v110805

Drum muster logo (2.5, 5, 10, 20 and 200 L packs only)

BASF Australia Ltd
ABN 62 008 437 867
500 Princes Hwy
Noble Park Vic 3174
# GLOSSARY

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td><strong>Active constituent</strong></td>
<td>The substance that is primarily responsible for the effect produced by a chemical product.</td>
</tr>
<tr>
<td><strong>Acute</strong></td>
<td>Having rapid onset and of short duration.</td>
</tr>
<tr>
<td><strong>Carcinogenicity</strong></td>
<td>The ability to cause cancer.</td>
</tr>
<tr>
<td><strong>Chronic</strong></td>
<td>Of long duration.</td>
</tr>
<tr>
<td><strong>Codex MRL</strong></td>
<td>Internationally published standard maximum residue limit.</td>
</tr>
<tr>
<td><strong>Desorption</strong></td>
<td>Removal of an absorbed material from a surface.</td>
</tr>
<tr>
<td><strong>Efficacy</strong></td>
<td>Production of the desired effect.</td>
</tr>
<tr>
<td><strong>Formulation</strong></td>
<td>A combination of both active and inactive constituents to form the end use product.</td>
</tr>
<tr>
<td><strong>Genotoxicity</strong></td>
<td>The ability to damage genetic material</td>
</tr>
<tr>
<td><strong>Hydrophobic</strong></td>
<td>Water repelling</td>
</tr>
<tr>
<td><strong>Leaching</strong></td>
<td>Removal of a compound by use of a solvent.</td>
</tr>
<tr>
<td><strong>Log P&lt;sub&gt;ow&lt;/sub&gt;</strong></td>
<td>Log to base 10 of octanol water partitioning co-efficient.</td>
</tr>
<tr>
<td><strong>Metabolism</strong></td>
<td>The conversion of food into energy</td>
</tr>
<tr>
<td><strong>Photodegradation</strong></td>
<td>Breakdown of chemicals due to the action of light.</td>
</tr>
<tr>
<td><strong>Photolysis</strong></td>
<td>Breakdown of chemicals due to the action of light.</td>
</tr>
<tr>
<td><strong>Subcutaneous</strong></td>
<td>Under the skin</td>
</tr>
<tr>
<td><strong>Toxicokinetics</strong></td>
<td>The study of the movement of toxins through the body.</td>
</tr>
<tr>
<td><strong>Toxicology</strong></td>
<td>The study of the nature and effects of poisons.</td>
</tr>
</tbody>
</table>
References


Australian Pesticides and Veterinary Medicines Authority 1997, *Ag Requirements Series: Guidelines for Registering Agricultural Chemicals*, APVMA, Canberra. (See footnote below)

Australian Pesticides and Veterinary Medicines Authority 1996, *MRL Standard: Maximum Residue Limits in Food and Animal Feedstuffs*, APVMA, Canberra. (See footnote below)

Australian Pesticides and Veterinary Medicines Authority 2001, *Ag Labelling Code—Code of Practice for Labelling Agricultural Chemical Products*, APVMA, Canberra. (See footnote below)

Footnote:
Updated versions of these documents are available on the APVMA website http://www.apvma.gov.au.
APVMA PUBLICATIONS ORDER FORM

To receive a copy of the full technical report for the evaluation of quinclorac in the product Drive Herbicide please fill in this form and send it, along with payment of $30 to:
David Hutchison
Pesticides Division
Australian Pesticides and Veterinary Medicines Authority
PO Box E240
Kingston ACT 2604

Alternatively, fax this form, along with your credit card details, to:
David Hutchison at 02 6272 3218.

Name (Mr, Mrs, Ms, Dr)_________________________________________
Position ______________________________________________________
Company/organisation __________________________________________
Address ______________________________________________________
Contact phone number (___) _____________________________________

I enclose payment by cheque, money order or credit card for $__________

Make cheques payable to ‘Australian Pesticides and Veterinary Medicines Authority’.

___ Bankcard    ___ Visa       ___ Mastercard
Card number _____/_____/_____/_____   Expiry date ...../...../......

Signature__________________________________  Date ______________