## B25-1.0 IMAZAPYR

### B25-1.1 Background Information

**IMAZAPYR, ACID**

**IUPAC:** [2-(4-isopropyl-4-methyl-5-oxo-2-imidazolin-2-yl)-nicotinic acid]

**CAS:** 2-[4,5-Dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-pyridinecarboxylic acid.

**CASN:** 81334-34-1

**IMAZAPYR, ISOPROPYLAMMONIUM**

**IUPAC:** 2-Propanamine, 2-(4,5-dihydo-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-pyridinecarboxylate

**CAS:** 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-pyridinecarboxylic acid compound with 2-propanamine (1:1)

**CASN:** 81510-83-0

**IMAZAPYR USAGE**

Imazapyr is a systemic, non-selective, pre- and post-emergent herbicide used for the control of a broad range of terrestrial and aquatic weeds. Imazapyr is utilized for many applications in the forestry industry including noxious weed control and rights-of-way vegetation management. Imazapyr is applied as either the acid or the isopropylamine salt.

Imazapyr is the active ingredient of the herbicide Arsenal® that was applied in conjunction with Karmex DF® 2001 and 2002 at Gagetown. In 2001 imazapyr was present as the nicotinic salt and six locations were sprayed; whereas in 2002 imazapyr was applied in one location and was present as the isopropylamine salt.

Imazapyr was utilized for three years to treat the range and training area (RTA) on a yearly basis.

**Table B25-1 Imazapyr Usage at CFB Gagetown[a]**

| Year | Total Imazapyr Applied (kg) | Total Area Treated (ha) |
|---|---|---|
| 2000 | 1.5E+01 | 2.1E+01 |
| 2001 | 2.0E+01 | 2.8E+01 |
| 2002[c] | 1.2E+01 | 1.7E+01 |
| **Total** | **4.7E+01** | **6.5E+01** |

[a] Adapted from JW, 2006.
[b] Average maximum yearly application rate (kg/ha).
[c] Imazapyr was present as an isopropylamine salt in 2002, whereas in 2000 and 2001 it was present as a nicotinic acid.

## B25-2.0 CHEMICAL AND PHYSICAL PROPERTIES

**Formula:** $C_{13}H_{15}N_3O_3$ (acid)
$C_{13}H_{15}N_3O_3H_9N$ (salt)

**Activity:** Imidazolinone (chemical class)

**Notes:** Imazapyr is a systemic, non-selective, pre-and post-emergent herbicide used for the control of a broad range of terrestrial and aquatic weeds. It controls plant growth by preventing the synthesis of branched-chain amino acids. Imazapyr is applied as either the acid or the isopropylamine salt.

**Structure:**

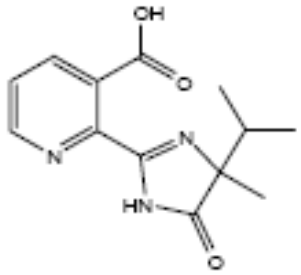


**Figure B25-1 Structure of Imazapyr [CAS 81334-34-1]**

**Table B25-2 Chemical and Physical Properties of Imazapyr**

| Chemical/Physical Property | Result | Reference |
|---|---|---|
| Dissociation Constant (pKa) | 1.81 | JW, 2006 |
| | $pk_1$= 1.9, $pk_2$=3.6 | U.S. EPA, 2005a |
| Henry's Law constant | $7.08x10^{-17}$ | JW, 2006 |
| Log $K_{ow}$ | 1.3 at 22°C | U.S. EPA, 2005a |
| | 1.56 | U.S. EPA, 2000 |
| Melting Point | 169-173°C | U.S. EPA, 2005a |
| Molecular Weight | 261.28 g/mol | JW, 2006; U.S. EPA, 2005a |
| Vapour Pressure | $<2x10^{-7}$ mm Hg at 25°C | U.S. EPA, 2005a |
| | $1.79x10^{-11}$ mm Hg | JW, 2006 |
| Water Solubility | $1.11x10^4$ mg/L | U.S. EPA, 2005a |
| | $1.13x10^4$ mg/L | JW, 2006 |

## B25-3.0 PMRA EVALUATION

Imazapyr is scheduled in the PMRA's Re-evaluation program work plan published on May, 2005. The program under which imazapyr's active ingredient will be re-evaluated will largely depend on the availability of suitable international (*e.g.*, U.S. EPA) reviews. PMRA will also consider other information such as monitoring data from provincial and territorial regulatory agencies and other governmental departments and stakeholders. Prior to a formal re-evaluation announcement, products containing active ingredients of imazapyr will not be subject to limitations in change of use pattern (PMRA, 2005).

## B25-4.0 TOXICOLOGICAL SUMMARY

### B25-4.1 Human Health Effects

During 1993 to 1997, six cases of acute poisoning with Arsenal (active ingredient imazapyr) occurred (Lee *et al*., 1999). Of the six cases, five were suicide attempts and one was an act of violence inflicted on a child. Three of the patients had severe symptoms including impairment of consciousness and respiratory distress. Other symptoms included metabolic acidosis, hypotension, leukocytosis, fever, mild elevation of hepatic tranaminase and creatinine, unconjugated hyperbilirubinemia, oral ulceration, pharyngolaryngitis, and chemical burns of the cornea (Lee *et al*., 1999). Mortality did not occur in any of the six cases. In general, toxic syndrome from Arsenal ingestion occurs at doses >100 mL. Effects include hypotension, pulmonary dysfunction, oral mucosal and gastrointestinal irritation, and transient liver and renal dysfunction (Lee *et al*., 1999).

### B25-4.2 Health Effects by Route of Exposure

#### *B25-4.2.1 Oral Exposure*

Imazapyr has low acute toxicity *via* the oral route (Toxicity Category IV) (U.S. EPA, 2006).

U.S. EPA (2005a) concluded that there is not a concern for neurotoxicity resulting from exposure to imazapyr. Transient salivation observed in a rat developmental toxicity study was not considered to be evidence of neurotoxicity as transient salivation is commonly observed in other oral rat studies. Therefore, there is no evidence of neurotoxicity (U.S. EPA, 2005a).

**Table B25-3 Mammalian $LD_{50}$ Values Resulting from Oral Exposure to Imazapyr**

| Test Type | Test Organism (Species/Sex) | $LD_{50}$ (mg/kg) | Reference |
|---|---|---|---|
| Acute | Rat | >5,000 | U.S. EPA, 2005a |

**Table B25-4 Effects Resulting from Oral Exposure to Imazapyr**

| Test Organism (Species) | Dose (Duration) | Response | Reference |
|---|---|---|---|
| Rat | 1,000 (days 6-15 of gestation) | Salivation | Salamon *et al*., 1983a |

B25-4.2.1.1 No Observed Adverse Effect Levels

**Table B25-5 NOAELs and LOAELs for Oral Exposure to Imazapyr[a]**

| Test Organism (Species) | Effect | Value (mg/kg/day) | Endpoint | Reference |
|---|---|---|---|---|
| **Sub-Chronic** | | | | |
| Rat | NOAEL | 1,695 (M), 1,784 (F) | No effects at the highest dose tested | Hess, 1992 |
| **Chronic** | | | | |
| Mice | NOAEL | 1,301 (M) and 1,639 (F) | No effect at the highest dose level | Hess, 1992 |

**Table B25-5 NOAELs and LOAELs for Oral Exposure to Imazapyr[a]**

| Test Organism (Species) | Effect | Value (mg/kg/day) | Endpoint | Reference |
|---|---|---|---|---|
| Dog | NOAEL | 250 mg/kg/day | No effects at the highest dose tested | Shellenberger, 1987 |

[a] Obtained from U.S. EPA, 2005a.

**Table B25-6 Imazapyr Reproductive and Developmental NOAEL and LOAEL Values[a]**

| Test Organism (Species) | Effect | Value (mg/kg/day) | Endpoint | Reference |
|---|---|---|---|---|
| Rat | LOAEL | 1,000 | Maternal effects (salivation) | Salamon *et al.*, 1983a |
| Rat | NOAEL | 300 (days 6-15 of gestation) | Maternal effects (salivation) | Salamon *et al.*, 1983a |
| Rabbit | NOAEL | 400 (days 6-18 of gestation) | No maternal of developmental effects at the highest dose tested | Salamon *et al.*, 1983b |
| Rat | NOAEL | 738 (M); 933.3 (F) | No reproductive or parental/offspring systemic effects at the highest dose tested | Robinson, 1987 |
| Rat | NOAEL | 1,000 (days 6-15 of gestation) | No developmental effects at the highest dose tested | Salamon *et al.*, 1983a |

[a] Obtained from U.S. EPA, 2005a.

### *B25-4.2.2 Dermal Exposure*

Imazapyr has low acute toxicity (Toxicity Category III) *via* dermal exposure (U.S. EPA, 2005a). It is not irritating to the skin, and is negative for dermal sensitization.

Imazapyr results in irreversible eye damage (Toxicity Category I) (U.S. EPA, 2005a). In a primary eye irritation study with New Zealand White rabbits, two of the six rabbits showed cornel opacity after 21 days. Discharge and vascularization of the cornea were each observed in one of the six rabbits at the end of the study (U.S. EPA, 2005a).

**Table B25-7 Mammalian acute $LD_{50}$ Value Resulting from Dermal Exposure to Imazapyr**

| Test Type | Test Organism (Species/Sex) | $LD_{50}$ (mg/kg) | Reference |
|---|---|---|---|
| Acute | Rabbit | >2,000 | U.S. EPA, 2005a |

**Table B25-8 NOAELs and LOAELs for Dermal Exposure to Imazapyr**

| Test Type | Test Organism (Species) | Effect | Value | Endpoint | Reference |
|---|---|---|---|---|---|
| Subchronic | Rabbit | NOAEL | >400 | No effects at the highest dose | Larson and Kelly, 1983 |

### *B25-4.2.3 Inhalation Exposure*

For the inhalation route imazapyr has been placed in Toxicity Category II (U.S. EPA, 2005a).

**Table B25-9 Mammalian $LD_{50}$ Value Resulting from Inhalation Exposure to Imazapyr**

| Test Type | Test Organism (Species/Sex) | $LC_{50}$ (mg/L) | Reference |
|---|---|---|---|
| Acute | Rat | >1,300 (gravimetric)<br>>5,100 (nominal) | U.S. EPA, 2005a |

## B25-4.3 Carcinogenicity

In the two year chronic exposure in the rat there was an increase in an uncommon tumour type of the brain (benign/malignant astrocytomas) in male rats (Daly, 1988). The increase was statistically significant for a positive trend, but not statistically significant in a pair-wise comparison. The majority of the carcinogenicity peer review committee (CPRC) in 1995 considered the astrocytomas not related to the imazapyr treatment based on the absence of a statistically significant pairwise increase (U.S. EPA, 2005a). In addition, herbicides structurally related to imazapyr did not indicate concern for carcinogenicity, and imazapyr is not mutagenic. Despite this, a review conducted by a subcommittee of the CPRC in 2003, re-evaluated the carcinogenic potential of imazapyr. Both meetings of the CPRC have resulted in imazapyr being classified by the U.S. EPA as a Group E chemical, with no evidence of carcinogenicity in at least two adequate studies in the rat and mouse (U.S. EPA, 2005a).

**Table B25-10 Animal Carcinogenicity Data[a]**

| Test Subjects | Exposure | Dose (mg/kg/day) | Response | Reference |
|---|---|---|---|---|
| Mice | Oral | 1,301 (M); 1,639 (F)<br>(18 months) | No evidence of carcinogenicity at any dose level | Hess, 1992 |
| Rat (Sprague Dawley) | Diet | 503 (M); 638.6 (F)<br>(2 years) | Nominal increase (not statistically pairwise) in brain astrocytomas in females | Daly, 1988 |

[a] Obtained from U.S. EPA, 2005a.

## B25-4.4 Populations at Special Risk

Special risk for specific populations were not identified for imazapyr. Imazapyr did not exhibit developmental toxicity, therefore, no extra safety factor is required for women or children (U.S. EPA, 2006).

## B25-4.5 Toxicokinetics

### *B25-4.5.1 Absorption*

Imazapyr is readily absorbed when exposure occurs through the oral route (Mallipudi and Wu, 1994).

### *B25-4.5.2 Distribution*

Administration of imazapyr to rats did not show any specific organs or tissues that sequestered imazapyr (Mallipudi and Wu, 1994).

### *B25-4.5.3 Metabolism*

After the administration of imazapyr to rats, almost all of the test material was excreted unchanged (Mallipudi and Wu, 1994). Two minor metabolites were detected in the urine or feces of treated rats; however, these metabolites only contributed <0.5% of the dose. Up to 12 additional unidentified metabolites were isolated that consisted of <3% of the original dose. Imazapyr is broken down by hydrolysis to form 2-carbonyl derivatives (Mallipudi and Wu, 1994).

### *B25-4.5.4 Elimination and Excretion*

Imazapyr is rapidly excreted from the body, as >90% of imazapyr administered to rats was recovered in the excreta unchanged within 48 hours (Mallipudi and Wu, 1994). Seven days after treatment essentially all of the labeled imazapyr had been eliminated.

Rats that were intravenously injected with imazapyr excreted 87 to 95% of the dose in the urine, and approximately 6% into the feces (Mallipudi and Wu, 1994).

## B25-4.6 Exposure Limits

### Table B25-11 Existing RfD Values for Imazapyr Exposures

| Reference Dose (mg/kg/day) | Reference | Endpoint | Study | Reference | NOEL (mg/kg/day) | UF | Study Classification |
|---|---|---|---|---|---|---|---|
| **Acute/Short-term (1-7 days)** | | | | | | | |
| 2.5 | USDA Forest Service, 1999; 2004 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | 1 year dog feeding study | Shellenberger, 1987 | 250 | 100 | -- |
| **Intermediate-term (7 days- Several months)** | | | | | | | |
| No information found | -- | -- | -- | -- | -- | -- | -- |
| **Long-term (6 months to lifetime)** | | | | | | | |
| 2.5 | U.S. EPA, 2006 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | 1 year dog feeding study | Shellenberger, 1987 | 250 | 100 | -- |

An acute dietary endpoint was not established by the U.S. EPA (2006) as there were no appropriate endpoints attributable to a single dose of imazapyr. The salivation seen in rat dams during gestation days 8 to 15 at 1,000 mg/kg (limit dose) in the rat developmental study was not considered to be an appropriate endpoint for risk assessment because it was a transient effect seen only at the highest dose level. However, a short-term incidental oral risk value that covers exposures ranging from one to 30 days was derived by the U.S. EPA (2005a). A NOAEL of 250 mg/kg/day is specified, with a margin of exposure of 100, resulting in a value 2.5 mg/kg/day. USDA Forest Service (1999; 2004) established an acute RfD based on the short-term incidental oral exposure provided by the U.S. EPA (2005a).

The one year dog feeding study (NOAEL= 250 mg/kg/day) was selected for the chronic RfD as it was the lowest NOAEL in the imazapyr database. However, the NOAEL of 250 mg/kg/day was also the highest dose tested. An uncertainty factor of 100 was applied to account for interspecies extrapolation and intraspecies variation. Therefore a chronic RfD of 2.5 mg/kg/day was calculated. However, because there were no adverse effects seen in any dog study on in any imazapyr toxicity studies, U.S. EPA relied on a structural analog, the pesticide imazapic, to choose an endpoint. Imazapic causes skeletal muscle effects in dogs at 137 and 180 mg/kg/day for males and females, respectively (U.S. EPA, 2005a).

**Table B25-12 Summary of the Toxicological dose and Endpoints for the used in Imazapyr Human Risk Assessment by the U.S. EPA (2006)**

| Exposure Scenario | Reference Dose | Endpoint | Study | NOEL (mg/kg/day) | LOEL (mg/kg/day) | MOE | Reference |
|---|---|---|---|---|---|---|---|
| Short- and Intermediate- Term Incidental Oral (1-30 days and 1-6 months) | 2.5 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | Oral study, 1 year dog feeding study | 250 | -- | 100 | Shellenberger, 1987 |
| Short, Intermediate- and Long-Term Dermal (1-30 days, 1 to 6 months, > 6 months)[a] | 2.5 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | 1 year dog feeding study | 250 | -- | 100 | Shellenberger, 1987 |
| Short, Intermediate- and Long-Term Inhalation (1-30 days, 1 to 6 months, >6 months)[a] | 2.5 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | 1 year dog feeding study | 250 | -- | 100 | Shellenberger, 1987 |

[a] Dermal and inhalation absorption rate of 100% were utilized.

Based on the general population (Table B25-11) and occupational (Table 25-12) reference values, the following exposure limits were selected for the risk assessment purposes of this report (Table B25-13).

**Table B23-15 Summary of Selected TRVs for Imazapyr**

| Chemical of Concern | TRV Type[a] | Route | TRV value (mg/kg/day) | Major Health Effects | Route of Exposure in Primary Study | Reference |
|---|---|---|---|---|---|---|
| Imazapyr | Acute/Short-term RfD (1-30 days) | Oral | 2.5 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | Oral | U.S. EPA, 2006 |
| | | Dermal | | | | |
| | | Inhalation | | | | |
| | Intermediate-term RfD (1 to 6 mths) | Oral | 2.5 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | Oral | U.S. EPA, 2006 |
| | | Dermal | | | | |
| | | Inhalation | | | | |
| | Long-term RfD (>6 months) | Oral | 2.5 | No adverse effects at the highest dose tested. Skeletal muscle effects caused by structural analog imazapic. | Oral | U.S. EPA, 2006 |
| | | Dermal | | | | |
| | | Inhalation | | | | |

## B25-5.0 ENVIRONMENTAL FATE AND EXPOSURE

Imazapyr is an anionic, organic acid that is non-volatile but persistent and mobile in the soil (U.S. EPA, 2006). Commercial formulations contain either imazapyr acid or the imazapyr isopropylamine salt. As imazapyr is mainly in anionic form at environmental pH levels, both the acid and salt forms are expected to have similar behaviour (U.S. EPA, 2006).

### B25-5.1 Air

Imazapyr is expected to exist solely in the particulate phase of the ambient environment. Particulate phase imazapyr may be removed from the air by wet and dry deposition (HSDB, 2002).

### B25-5.2 Water

Imazapyr is mobile in runoff and surface water, and has the potential to leach to groundwater (U.S. EPA, 2006).

Laboratory studies show that imazapyr is essentially stable to hydrolysis and aerobic and anaerobic aquatic metabolism (U.S. EPA, 2006). Upon direct or indirect application to surface water imazapyr may be rapidly degraded through photolysis (U.S. EPA, 2006) (Table 5-1).

**Table B25-13 Half-life of Imazapyr in Water**

| Conditions | Half-life | Reference |
|---|---|---|
| Photodegradation | ~2-5 days | JW, 2005 |
| Photolysis; surface water | 3-5 days | U.S. EPA, 2006 |
| Aerobic and anaerobic aquatic metabolism | > 120 days | U.S. EPA, 2005b |

### B25-5.3 Sediment and Soil

In general, imazapyr does not absorb to soil, and can be mobile in the environment. Heavy rainfall results in high mobilization and leaching (JW, 2005).

Imazapyr is essentially stable to aerobic and anaerobic soil degradation (U.S. EPA, 2006). The half-life of imazapyr in soil varies greatly depending on the soil type (JW, 2006) (Table 5-2).

**Table B25-14 Half-life of Imazapyr in Soil**

| Conditions | Half-life | Reference |
|---|---|---|
| Anaerobic soil metabolism; loamy sand soil | >60 days | U.S. EPA, 2005b |
| Field dissipation; sandy loam soil | 64 days | U.S. EPA, 2005b |
| Field dissipation; silt loam soil | 143 days | U.S. EPA, 2005b |
| Photolysis; loamy sand soil | 149 days | U.S. EPA, 2005b |
| Dry soil | ~ 1 year | JW, 2005 |
| Loam and clay loam soil | >4 year | JW, 2005 |
| Aerobic soil metabolism; loamy sand soil | ~5.9 years | U.S. EPA, 2005b |

### B25-5.4 Other Environmental Media

Imazapyr is not expected to bioaccumulate in aquatic organisms because it exists as an anion at typical environmental pHs (U.S. EPA, 2006). Imazapyr has a bioconcentration factor of 3.162 (JW, 2006).

## B25-6.0 SUMMARY

Imazapyr is a systemic, non-selective, pre/post-emergent herbicide used for the control of a broad range of terrestrial and aquatic weeds. Imazapyr was utilized for 3 years to treat the range and training area (RTA) at CFB Gagetown. Approximately 47 kg of imazapyr was applied over an area of 65 ha (JW, 2006).

Imazapyr shows very low inherent toxicity, with no evidence of acute or chronic neurotoxicity and no signs of developmental toxicity. However, maternal toxicity, based on increased salivation, was observed in rats. Chronic imazapyr feeding studies in the rat, mouse and the dog have shown no compound related effects. The U.S. EPA (2006) has classified imazapyr as a Group E carcinogen (no evidence of carcinogenicity in humans) with at least two adequate studies in rodents.

## B25-7.0 REFERENCES

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