



Pesticide
Fact Sheet

Name of Chemical: Tebufenpyrad
Reason for Issuance: Unconditional Registration
Date Issued: August 26, 2002

1. DESCRIPTION OF CHEMICAL

Generic Name: 4-chloro-N-[[4-(1,1-dimethylethyl)phenyl]methyl]-3-ethyl-1-methyl-1H-pyrazole-5-carboxamide

Common Name: Tebufenpyrad

Trade Name: AC 801, 757 3EC Miticide-Insecticide

EPA PC Code: 090102

Chemical Abstracts
Service (CAS) Number: 119168-77-3

Year of Initial
Registration: 2002

Pesticide Type: Insecticide/Miticide

Chemical Family: Pyrazole

U.S. Producer: BASF Corporation

2. USE PATTERNS AND FORMULATIONS

Application Sites: Tebufenpyrad is registered for use on ornamental plants grown in commercial greenhouses.

Types of Formulations: 98.9% technical product
34.6% EC end-use product

Types and Methods: Hydraulic, backpack, compressed air, low volume

of Application: electrostatic and other types of sprayers for greenhouse applications

Application Rates: An application rate of 0.8 to 3.4 fluid ounces of product (0.02 to 0.08 pounds active ingredient) per 100 gallons sprayed to obtain uniform and complete coverage of foliage. Applications may be repeated at 5 to 7 day intervals to maintain control. 3 to 4 treatments may be needed.

Carrier: Water

3. SCIENCE FINDINGS

Tebufenpyrad is a member of the pyrazole class of insecticides. The review of available product chemistry, toxicology, ecological effects and environmental fate data for tebufenpyrad has been completed. The data and estimated risks to human health and the environment from its use on ornamental crops grown in commercial greenhouses are summarized below:

Chemical Characteristics

PROPERTY	TECHNICAL	END-USE
Physical State	crystalline solid	liquid
Color	white	N/A
Odor	weak halide	N/A
Melting Point	64 -66 C	N/A
Density	0.5 g/mL @ 24.1 C	8.71 lbs./gal.
Solubility (Water)	2.61 ppm at pH 5.9 3.21 ppm at pH 4 2.39 ppm at pH 7 2.32 ppm at pH 10	N/A
Vapor Pressure	$<7.3 \times 10^{-8}$ mm Hg @ 20C	N/A
Octanol/Water Partition Coefficient	$K_{ow} = 84,850$	N/A
pH	5.9 in water	5.0

Toxicology Characteristics

Acute Toxicity Data on Technical Tebufenpyrad

Guideline No.	Study Type	Results	Toxicity Category
81-1; OPPTS 870.1100	Acute Oral - Mouse	LD ₅₀ in males = 224 mg/kg; LD ₅₀ in females = 210 mg/kg Combined LD ₅₀ = 217 mg/kg	II
81-1 OPPTS 870.1100	Acute Oral - Rat	LD ₅₀ in males = 595 mg/kg; LD ₅₀ in females = 997 mg/kg Combined LD ₅₀ = 786 mg/kg	III
81-2; OPPTS 870.1200	Acute Dermal - Rat	LD ₅₀ > 2000 mg/kg in males and females	III
81-3; OPPTS 870.1300	Acute Inhalation - Rat	LD ₅₀ in males = 2.66 mg/L; LD ₅₀ in females = could not be calculated ^a Combined LD ₅₀ = 3.01 mg/L	IV
81-4; OPPTS 870.2400	Primary Eye Irritation - Rabbit	minimal irritant	III
81-5; OPPTS 870.2500	Primary Skin Irritation - Rabbit	not a dermal irritant	IV
81-6; OPPTS 870.2600	Dermal Sensitization - guinea pig	not a dermal sensitizer	
81-6; OPPTS 870.2600	Dermal Sensitization - guinea pig	mild dermal sensitizer	

^a In females treated at 0.38, 1.28, 2.14, 2.70 and 3.09 mg/L, there were 0/5, 2/5, 1/5, 2/5 and 2/5 deaths, respectively. The LD₅₀ could not be calculated but it is > 2 mg/L (Toxicity Category IV)

Acute Toxicity Data on AC 801,757 3EC Formulation

Guideline No.	Study Type	Results	Toxicity Category
81-1 OPPTS 870.1100	Acute Oral - Rat	LD ₅₀ in males = 371 mg/kg; LD ₅₀ in females = 206 mg/kg Combined LD ₅₀ = 279 mg/kg	II
81-2; OPPTS 870.1200	Acute Dermal - Rat	LD ₅₀ > 2000 mg/kg in males and females	III
81-3; OPPTS 870.1300	Acute Inhalation - Rat	LD ₅₀ in males = 1.4 mg/L; LD ₅₀ in females = 1.8 mg/L Combined LD ₅₀ = 1.6 mg/L	III
81-4; OPPTS 870.2400	Primary Eye Irritation - Rabbit	moderate eye irritant	III
81-5; OPPTS 870.2500	Primary Skin Irritation - Rabbit	moderate dermal irritant	III
81-6; OPPTS 870.2600	Dermal Sensitization - guinea pig	not a dermal sensitizer	

Subchronic/Chronic Toxicity Data on Technical Tebufenpyrad

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
870.3100 90-Day oral toxicity in rat	43309311, 43309312 (1991) Acceptable/guideline 0, 10, 100, 400 ppm (M: 0.7, 6.8 and 29 mg/kg/day; F: 0.7, 7.3 and 32 mg/kg/day, respectively)	NOAEL for males and females = 100 ppm (M/F: 6.8/7.3 mg/kg/day) LOAEL = 400 ppm (M/F: 29/32 mg/kg/day) based on decreases in body weight gain and changes in relative weights of multiple organs
870.3150 90-day oral toxicity in dog	43309313 (1992) acceptable/guideline 0, 2, 10, 20 mg/kg/day	NOAEL for males and females = 2 mg/kg/day LOAEL = 10 mg/kg/day based on an increased incidence of diarrhea and/or vomiting ^a
870.3200 21/28-Day dermal toxicity in rabbit	43309314 (1992) acceptable/guideline 0, 40, 200, 1000 mg/kg/day	systemic NOAEL = 200 mg/kg/day. systemic LOAEL = 1000 mg/kg/day based on decreased body weight gain and food consumption dermal irritation NOAEL > 1000 mg/kg/day dermal irritation LOAEL = not established
870.3700a Prenatal developmental in rats	43309317 (1992) acceptable/guideline 0, 15, 50, 90 mg/kg/day	maternal toxicity NOAEL = 15 mg/kg/day. maternal toxicity LOAEL = 50 mg/kg/day based on decreased body weight gain and increased water consumption developmental toxicity NOAEL = 50 mg/kg/day developmental toxicity LOAEL = 90 mg/kg/day based on an increase in fetal and litter incidence of additional ribs
870.3700b Prenatal developmental in rabbits	43309318(1991) acceptable/guideline 0, 5, 15, 40 mg/kg/day	maternal toxicity NOAEL = 15 mg/kg/day. maternal toxicity LOAEL = 40 mg/kg/day based on abortions, reduced body weight gain and food consumption developmental toxicity NOAEL = 15 mg/kg/day developmental toxicity LOAEL = 40 mg/kg/day based on abortions.
870.3800 Reproduction and fertility effects in rats	43309319 (1992) acceptable/guideline 0, 20, 100, or 200 ppm (1.7, 8.3 and 16.7 mg/kg/day for F ₀ males; 1.9, 9.6 and 19.4 mg/kg/day for F ₀ females; 1.7, 8.4 and 16.8 mg/kg/day for F ₁ males; 1.9, 9.6 and 19.3 mg/kg/day for F ₁ females)	Parental toxicity NOAEL >200 ppm. Parental toxicity LOAEL was not established Reproduction toxicity NOAEL >200 ppm Reproduction toxicity LOAEL was not established Offspring NOAEL = 100 ppm (8.3-9.6 mg/kg/day). Offspring LOAEL = 200 ppm (16.7-19.4 mg/kg/day) based on reduced body weights/ body weight gains in male and female offspring and delayed vaginal opening in females

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
870.4100b Chronic toxicity in dogs	43309315 (1992) acceptable/guideline 0, 1, 6, 20 mg/kg/day	NOAEL in males and females = 1 mg/kg/day LOAEL = 6 mg/kg/day based on increased incidence of vomiting and diarrhea/loose stools and thickened gastric mucosa and chronic gastritis in the pyloric region
870.4300 Chronic toxicity/ carcinogenicity in rats	43309320 (1992) acceptable/guideline 0, 5, 20, 150, or 300 ppm (M; 0, 0.21, 0.82, 6.52, 13.43 mg/kg/day; F: 0, 0.26, 1.01, 8.13, 16.95 mg/kg/day)	NOAEL in males and females = 20 ppm (M/F: 0.82/1.01 mg/kg/day) LOAEL = 150 ppm (M/F: 6.52/8.13 mg/kg/day) based on decreased body weights/ body weight gains and liver toxicity in both sexes and slight microcytic anemia and effects on ovary in females There was an increased incidence of hepatocellular adenomas in male rats at 150 (7%, N.S.) and 300 ppm (18%, p<0.01) compared with the incidence in concurrent controls (0%). The incidence of hepatocellular adenomas in the historical control animals was 0 - 8%.
870.4300 Carcinogenicity in mice	43309316 (1994) acceptable/guideline 0, 30, 500, or 1000 ppm. (M: 0, 3.6, 64.4, and 132.1 mg/kg/day; F: 0, 4.2, 71.3, and 162.0)	NOAEL = 30 ppm (M/F: 3.6/4.2 mg/kg/day) LOAEL = 500 ppm (M/F: 64.4/71.3 mg/kg/day) based on decreased body weight gain no evidence of carcinogenicity at doses which were adequate
Gene Mutation 870.5100 - bacterial reverse mutation	43309321 (1990) acceptable/guideline 50, 158, 500, 1580 and 5000 : g/plate, with and without metabolic activation	There was no evidence of induced revertant colonies over solvent control values in any strain at any dose tested, either with or without S9 mix.
Gene Mutation 870.5300 - HGPRT locus in Chinese hamster V79 cells	43309322 (1991) acceptable/guideline non-activated conditions at concentrations of 1.25, 2.5, 5, 10, 20, 30 : g/ml in the first assay and 2.5, 5, 10, 20, 30, 40 : g/ml in a second assay and under activated conditions at concentrations of 10, 20, 40, 60, 100, 150 : g/ml in the first assay and to 40, 60, 100, 150, 175, 200 : g/ml in a second assay	No reproducible dose-related increase in mutation frequency was seen at the HGPRT locus in Chinese hamster V79 cells in this study, either with or without S9 mix.
Cytogenetics 870.5395 - micronucleus assay in Chinese hamsters	43309323 (1991) acceptable/guideline 75, 150 or 300 mg/kg	There was no significant increase in the frequency of micronucleated PCEs in bone marrow after any dose of MK-239 tested in this study.

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
Cytogenetics 870.5395 - <i>in vitro</i> cytogenetics assay with human lymphocytes	43309324 (1990, 1994) acceptable/guideline a) 6, 8, 20, 40, 60, 80 : g/ml for 21 hours without activation and 8.25, 11, 27.5, 55, 82.5, 110 : g/ml for 4 hours with activation b) 6.25, 12.5, 25 : g/ml for 2 hours without activation and 12.5, 25 and 50 : g/ml for 3 hours with activation.	Overall, the combined data from both studies indicate that without S9 activation, the compound induced variable but nevertheless reproducible significant increases in the percentage of aberrant cells in two of three experiments using treatment times of 20-24 hours. In general, levels causing a 40% decrease in the MI were negative, whereas concentrations causing a 42% decrease in the MI induced significant effects with reproducibly flat dose response curves. Furthermore, the same type of aberrations (chromatid breaks) was induced in both studies. Based on these considerations, it is concluded that MK-239 exhibited reproducible but weak evidence of a clastogenic response but only after prolonged exposure to cytotoxic doses and only in the absence of S9 activation.
Other Effects 870.5500 - Bacterial DNA Damage or Repair Assay	43320001 (1991) acceptable/guideline 200, 500, 1000, 2000, 5000 and 10,000 : g/filter paper disk/plate with and without exogenous metabolic activation.	MK-239 did not induce preferential killing of the repair defective M45(re-) strain tested, either in the presence or absence of S9 mix, indicating no ability to damage DNA as tested in this study.
Other Effects 870.5550 - Unscheduled DNA Synthesis	43309325 (1994) acceptable/guideline concentrations of 0.0977, 0.309, 0.977, 3.09 and 9.77 : g/ml	MK-239 did not induce DNA damage detectable by the UDS assay.
870.7485 Metabolism and pharmacokinetics	43309326 (1993) Acceptable/guideline single gavage dose of 10 or 50 mg/kg single gavage dose of 10 mg/kg or 50 mg/kg (bile cannulation study) pretreatment with unlabeled test material for 14 days before single dose of 10 mg/kg radiolabeled MK-239	The results show >80% of the MK-239 was absorbed from the digestive system within 24 hours. The compound appeared to undergo rapid and extensive first-pass metabolism to primarily hydroxylated or carboxylated products with little of the parent compound appearing in the urine or feces. As a result, the test material was found within the stomach and intestinal tract, associated lymphatics, and the liver with lesser amounts found within the kidney. The test material was excreted primarily in the feces which accounted for 60% of the elimination; however, a significant portion was found in the urine (16-24%). More than 70% of the test material or its metabolites were eliminated within 72 hours of treatment and >90% was eliminated by 7 days. No accumulation of the parent compound or its metabolites was noted. A slight sex-specific difference in the metabolic disposition of the test material was found with male rats excreting more of the carboxylic acid metabolite on a relative basis while females tended to excrete more of the sulfate conjugate.

* The actual dosages in the 10 and 20 mg/kg/day animals are uncertain due to the vomiting.

Toxicological Endpoints

The dose at which no adverse effects are observed (the NOAEL) from the toxicology study identified as appropriate for use in risk assessment is used to estimate the toxicological level of concern (LOC). The lowest dose at which adverse effects of concern are identified (the LOAEL) is sometimes used for risk assessment if no NOAEL was achieved in the toxicology study selected. An uncertainty factor (UF) is applied to reflect uncertainties inherent in the extrapolation from laboratory animal data to humans and in the variations in sensitivity among members of the human population as well as

other unknowns. An UF of 100 is routinely used, 10X to account for interspecies differences and 10X for intra species differences.

The acute, sub-chronic and chronic (non-cancer) toxicological endpoints that have been established for tebufenpyrad are summarized in the following table.

Exposure Scenario	Dose (mg/kg/day) UF /MOE	Hazard Based Special FQPA Safety Factor	Endpoint for Risk Assessment	Study
Dietary Risk Assessments				
Acute Dietary <u>females 13-50 years of age</u>	N/A	N/A	N/A - no food uses	
Acute Dietary <u>general population including infants and children</u>	N/A	N/A	N/A - no food uses	
Chronic Dietary <u>all populations</u>	N/A	N/A	N/A - no food uses	
Incidental Oral Short-Term (1 - 30 Days) Residential Only	N/A	N/A	N/A - no residential uses	
Incidental Oral Intermediate-Term (1 - 6 Months) Residential Only	N/A	N/A	N/A - no residential uses	
Non-Dietary Risk Assessments				

Exposure Scenario	Dose (mg/kg/day) UF /MOE	Hazard Based Special FQPA Safety Factor	Endpoint for Risk Assessment	Study
Dermal Short- and Intermediate - Term (1 - 30 days and 1-6 Months)	Dermal NOAEL= 200 mg/kg/day		LOAEL = 1000 mg/kg/day based on decreased bodyweight gain and food consumption in male and female New Zealand White rabbits.	21-day dermal study in the rabbit
Residential	MOE = N/A	N/A		
Occupational	MOE = 100	N/A		
Dermal Long-Term (> 6 Months)	Oral NOAEL ¹ = 0.82 mg/kg/day		LOAEL = 6.52 mg/kg/day based on decreased body weights and body weight gain and liver toxicity in both sexes and a slight microcytic anemia and effects on the ovary in females.	Combined chronic toxicity/carcinogenicity in rats
Residential	MOE = N/A	N/A		
Occupational	MOE = 100	N/A		
Inhalation Short-Term (1 - 30 days)	Oral NOAEL ² = 15 mg/kg/day		LOAEL = 50 mg/kg/day based on decreased body weight gain and increased water consumption.	Prenatal developmental toxicity study in rats
Residential	MOE = N/A	N/A		
Occupational	MOE = 100	N/A		
Inhalation Intermediate-Term (1 - 6 Months)	Oral NOAEL ² = 2 mg/kg/day		LOAEL = 10 mg/kg/day based on an increased incidence of diarrhea and/or vomiting in males and females.	90-Day Capsule Study in the Dog
Residential	MOE = N/A	N/A		
Occupational	MOE = 100	N/A		

Exposure Scenario	Dose (mg/kg/day) UF /MOE	Hazard Based Special FQPA Safety Factor	Endpoint for Risk Assessment	Study
Inhalation Long-Term (>6 Months)	Oral NOAEL ² = 1 mg/kg/day		LOAEL = 6 mg/kg/day based on vomiting and diarrhea/loose stools and thickened gastric mucosa and chronic gastritis in the pyloric region.	1-Year Capsule Study in the Dog
Residential	MOE = N/A	N/A		
Occupational	MOE = 100	N/A		
Cancer	Classification: Suggestive Evidence of Carcinogenicity, but Not Sufficient to Assess Human Carcinogenic Potential			

¹ An oral NOAEL was used for an endpoint for the dermal assessment. An estimated 4% dermal absorption value will be used with the oral NOAEL.

² An oral NOAEL was used for an endpoint for the inhalation assessment. Inhalation absorption is assumed to be equivalent to oral absorption.

Carcinogenicity

In accordance with the EPA *Draft Guidelines for Carcinogen Risk Assessment* (July 1999), the Cancer Assessment Review Committee classified tebufenpyrad into the category “**Suggestive Evidence of Carcinogenicity, but Not Sufficient to Assess Human Carcinogenic Potential**”, based on the following findings:

- < Male rats had a significant increasing trend, and a significant difference in the pair-wise comparison of the 300 ppm dose group with the controls, for hepatocellular adenomas, both at $p < 0.01$. The incidence at the high dose (300 ppm) exceeded the historical control range. The CARC considered the increase in benign liver tumors to be treatment-related in males. No hepatocellular carcinomas were observed in any group, including controls.
- < Female rats had a significant increasing trend, and a significant difference in the pair-wise comparison of the 150 ppm dose group with the controls, for hepatocellular adenomas, both at $p < 0.05$. However, no dose-related increase in these tumors was noted at the high dose (300 ppm). The incidence at 150 ppm was just outside the historical control range; and the incidence at 300 ppm was within the historical control range. No hepatocellular carcinomas were observed in any group, including controls.
- < In rats, dosing at the highest level was considered by the CARC to be adequate, but not excessive, in both sexes based on decreased body weight gains in males (21%) and females (33%), clinical chemistry changes, increased liver weights, and liver hypertrophy.

- < There was no treatment-related increase in any tumors in male and female mice.
- < In mice, dosing at the highest level (1000 ppm) was considered by the CARC to be adequate, but not excessive, in both sexes based on decreased body weight and body weight gain in males and females (61-75% of controls) after 52 and 78 weeks of treatment, decreased food efficiency, and a dose-related increased incidence of stomach dysplasia in both sexes.
- < Tebufenpyrad was not mutagenic in bacterial or mammalian cell gene mutation assays. However, tebufenpyrad induced weak but reproducible clastogenic effects in human lymphocytes after prolonged exposure to cytotoxic concentrations. There was no increase in the frequency of micronucleated PCEs in bone marrow and no induced DNA damage in mammalian cells or in bacteria. Based on the findings the level of concern for in vitro clastogenesis is low because it was only seen after prolonged exposure to cytotoxic concentrations and was not manifested in the in vivo cytogenetic assay up to lethal doses. The submitted studies were acceptable and satisfy the guideline requirements for mutagenicity data. No further testing is required at this time. The Committee has no concern for mutagenicity.
- < No appropriate structural analogues were located for comparison purposes.
- < There are no mode of action studies available at this time.

Metabolism

The metabolism study in the rat showed that >80% of tebufenpyrad was absorbed from the digestive system within 24 hours. The compound appeared to undergo rapid and extensive first-pass metabolism to primarily hydroxylated or carboxylated products with little of the parent compound appearing in the urine or feces. It was excreted primarily in the feces which accounted for 60% of the elimination; however, a significant portion was found in the urine (16-24%). More than 70% of the test material or its metabolites were eliminated within 72 hours of treatment and >90% was eliminated by 7 days. No accumulation of the parent compound or its metabolites was noted. A slight sex-specific difference in the metabolic disposition of the test material was found with male rats excreting more of the carboxylic acid metabolite on a relative basis while females tended to excrete more of the sulfate conjugate.

Human Exposures and Risks

Acute and Chronic Dietary Risk - Not applicable, since there are no food uses of tebufenpyrad.

Occupational Risk - Estimated Margins of Exposure (MOEs) for all workers (including handler and postapplication exposures) exceed the target Margins of Exposure (MOEs) of 100 in all cases, provided handlers wear gloves in addition to the baseline Personal Protective Equipment (PPE) and a 12-hour Restricted-Entry Interval is observed. The product's label includes both requirements. Details of the occupational risk assessment are provided below.

Acute toxicity categories for the technical grade tebufenpyrad are toxicity category II for oral, toxicity category III for dermal, toxicity category IV for inhalation, and toxicity category III for primary eye irritation. Assessment of risk was based on the toxicologic endpoints selected by HIARC. For estimating short- and intermediate-term dermal risk, a 21-day rabbit study reflecting dermal application of the pesticide was used. Both short-term (1-30 days) and intermediate-term (30-180 days) dermal exposures were compared to a NOAEL of 200 mg/kg/day based on decreased body weight gain and food consumption at the LOAEL of 1000 mg/kg/day. Long-term (more than 180 days) dermal exposures were compared to an oral NOAEL of 0.82 mg/kg/day with 4 % absorption factor. This endpoint was also based on body weight effects as well as anemia and effects on the liver and ovaries.

For assessing short-, intermediate- and long-term inhalation risk, oral NOAELs were selected for inhalation exposure risk assessment using route-to-route extrapolation. NOAELs of 15, 2 and 1 mg/kg/day were used for short, intermediate and long- term assessment, respectively. Total MOEs are also calculated for short-term duration, because there is a common endpoint (decreased body weight gain). The uncertainty factor of 100 is applied to all routes and exposure durations.

The Cancer Assessment Review Committee (CARC) characterized tebufenpyrad as a "cannot be determined, suggestive" carcinogen. Therefore, an occupational cancer risk assessment was not conducted.

No **handler** exposure studies were conducted by the registrant, therefore surrogate data from the Pesticide Handlers Exposure Database (PHED) Version 1.1, were used to assess the potential exposures resulting from handling and applying tebufenpyrad.

For mixer/loader/applicator using high pressure handwand, all MOEs at baseline or PPE exceed the target MOE of 100 (range 200-1,100,000). In the case of mixer/loader/applicator using low pressure handwand, all baseline MOEs exceed 100 with the exception of the long-term MOE of 90. With the additional PPE (gloves), the MOE increases to 21,000. For back pack mixer/loader/applicators, data are not available for baseline dermal exposure (i.e., without gloves). Therefore dermal MOEs for this scenario were calculated using gloves only. All MOE's for this scenario (baseline inhalation, dermal with gloves) exceed the target MOE of 100.

No **postapplication** exposure studies were conducted by the registrant. Therefore, postapplication exposures to occupational workers were estimated using assumptions for a surrogate postapplication assessment presented in the Standard Operating Procedures (SOPs) for Residential Exposure Assessments (12/18/1997). These data were used in this assessment in conjunction with ARTF (Exposure SAC Policy guidance 3.1, 8/00) transfer coefficients to assess potential exposures to workers reentering treated sites. The results of the **occupational postapplication** assessments indicate that re-entry restrictions for cut flowers did not exceed EPA's level of concern provided a 12 hour REI is observed. All MOE's for postapplication exposure met or exceeded the target MOE of 100 (range 100-11,000).

Environmental Characteristics

The major routes of dissipation for tebufenpyrad appear to be microbially-mediated degradation and adsorption to soil. It is stable to hydrolysis at pH 5, 7 and 9. There appears to be little potential for tebufenpyrad to be transported with water, although transport of residues adsorbed to eroding soil is possible. Because tebufenpyrad is registered for greenhouse use only, environmental exposure will be limited.

Ecological Characteristics/Risk

Terrestrial: Tebufenpyrad is practically non-toxic to birds on an acute and subacute dietary basis ($LD_{50} > 2000$ mg/kg; $LC_{50} > 5000$ ppm). Since the registered use in greenhouses is not expected to result in significant exposure to non-target organisms, chronic testing was not required.

Data were not submitted to assess the toxicity of tebufenpyrad to honey bees; however, the use of tebufenpyrad in greenhouses is not expected to result in honey bee exposure.

Aquatic: Tebufenpyrad is very highly toxic to fish based on 96-hour acute toxicity studies in rainbow trout and bluegill sunfish. However, the use of tebufenpyrad in greenhouses is not expected to result in exposure of non-target aquatic organisms to tebufenpyrad. For this same reason, chronic aquatic testing was not required.

4. SUMMARY OF REGULATORY POSITION AND RATIONALE

Available data provide adequate information to support the unconditional registration of tebufenpyrad technical and end-use products for use on ornamental crops grown in commercial greenhouses.

Use, Formulation, Manufacturing Process or Geographic Restrictions

Restrictions for Use on Ornamental Crops Grown in Greenhouses:

- < Do not apply through any type of irrigation system.
- < Do not apply this product in a way that will contact workers or other persons, either directly or through drift. Only protected handlers may be in the area during application.
- < Do not enter or allow worker entry into treated areas during the restricted entry interval (REI) of 12 hours.
- < Do not contaminate water, food or feed by storage or disposal.
- < DO NOT contaminate water when disposing of equipment washwaters.

5. SUMMARY OF DATA GAPS

- < 90-Day Inhalation Study (Guideline 870.3465). [This study was requested to confirm the occupational inhalation exposure MOEs that were derived based on route-to-route extrapolation using oral study endpoints.]

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