

EPA Region II

Enforcement Coordinator: Barbara McGarry—mcgarry.barbara@epa.gov
 Enforcement Division Director: Richard Caspe—caspe.richard@epa.gov

EPA Region III

Enforcement Coordinator: Samantha Fairchild—
fairchild.samantha@epa.gov

EPA Region IV

Enforcement Coordinator: Sherri Fields—fields.sherri@epa.gov
 Enforcement Division Director: William Anderson—
anderson.william@epa.gov

EPA Region V

Enforcement Coordinator: Tinka Hyde—
hyde.tinka@epa.gov

EPA Region VI

Enforcement Coordinator: Walter Biggins—biggins.walter@epa.gov
 Enforcement Division Director: Samuel Coleman—coleman.samuel@epa.gov

EPA Region VII

Enforcement Coordinator: Cecilia Tapia—tapia.cecilia@epa.gov

EPA Region VIII

Enforcement Coordinator: Eddie Sierra—sierra.eddie@epa.gov
 Enforcement Division Director: Carol Rushin—rushin.carol@epa.gov

EPA Region IX

Enforcement Coordinator: Sally Seymour—seymour.sally@epa.gov

EPA Region X

Enforcement Coordinator: Lauris Davies—davies.lauris@epa.gov

Dated: May 21, 2002.

Michael M. Stahl,

Director, Office of Compliance.

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BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

[FRL-7218-8]

Environmental Laboratory Advisory Board Meeting Date, and Agenda

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of public meeting.

SUMMARY: The Environmental Protection Agency's Environmental Laboratory Advisory Board (ELAB) will hold an Open Forum on Tuesday July 9, 2002 at 5-6 p.m. EDT and an Open Meeting on Thursday July 11, 2002 at 1:30-5 p.m.

EDT at the Wyndham Harbour Island Hotel, 725 S. Harbour Island Boulevard, Tampa, Florida. Members of the public are invited to attend both events. Items to be discussed include: (1) Update on recommendations to restructure the National Environmental Laboratory Accreditation Conference (NELAC) to allow it to better serve the future needs of EPA, the States, and the private sector, (2) discussion of ELAB recommendations to EPA, and (3) review of Action Items from the June 19 ELAB meeting. ELAB is soliciting input from the public on these and other issues related to the National Environmental Laboratory Accreditation Program (NELAP) and the NELAC standards. Written comments on NELAP laboratory accreditation and the NELAC standards are encouraged and should be sent to Mr. Edward Kantor, DFO, P.O. Box 93478, Las Vegas, NV 89193, faxed to (702) 798-2261, or e-mailed to kantor.edward@epa.gov. or can be presented in person at the Open Forum, July 9. Members of the public are invited to raise issues or to make comments at the Open Forum and time permitting, will be allowed to comment on discussions ensued from the ELAB Open Meeting.

Dated: May 20, 2002.

John G. Lyon,

*Director, Environmental Sciences Division,
 National Environmental Research Laboratory.*

[FR Doc. 02-13351 Filed 5-28-02; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[FRL-7218-7]

Environmental Laboratory Advisory Board (ELAB) Meeting Date, and Agenda

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of teleconference meeting.

SUMMARY: The Environmental Protection Agency's Environmental Laboratory Advisory Board (ELAB) will have a teleconference meeting on June 19, 2002, at 11 a.m. EDT to discuss the ideas and views presented at the previous ELAB meetings, as well as new business. Items to be discussed include: (1) Update on recommendations to restructure the National Environmental Laboratory Accreditation Conference (NELAC) to allow it to better serve the future needs of EPA, the States, and the private sector, (2) discussion of ELAB recommendations to EPA, (3) review of

Action Items from the April 17 ELAB meeting, and (4) ELAB upcoming meeting at NELAC 8. ELAB is soliciting input from the public on these and other issues related to the National Environmental Laboratory Accreditation Program (NELAP) and the NELAC standards. Written comments on NELAP laboratory accreditation and the NELAC standards are encouraged and should be sent to Mr. Edward Kantor, DFO, P.O. Box 93478, Las Vegas NV 89193, faxed to (702) 798-2261, or emailed to kantor.edward@epa.gov. Members of the public are invited to listen to the teleconference calls and, time permitting, will be allowed to comment on issues discussed during this and previous ELAB meetings. Those persons interested in attending should call Edward Kantor at 702-798-2690 to obtain teleconference information. The number of lines are limited and will be distributed on a first come, first serve basis. Preference will be given to a group wishing to attend over a request from an individual.

Dated: May 20, 2002.

John G. Lyon,

*Director, Environmental Sciences Division,
 National Environmental Research Laboratory.*

[FR Doc. 02-13352 Filed 5-28-02; 8:45 am]

BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

[OPP-2002-0074; FRL-7178-3]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket control number OPP-2002-0074, must be received on or before June 28, 2002.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the

SUPPLEMENTARY INFORMATION. To ensure proper receipt by EPA, it is imperative that you identify docket ID number OPP-2002-0074 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Joseph Tavano, Registration

Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305-6411; and e-mail address: tavano.joseph@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at <http://www.epa.gov/>. To access this document, on the Home Page select "Laws and Regulations," "Regulations and Proposed Rules," and then look up the entry for this document under the "Federal Register"—Environmental Documents. You can also go directly to the **Federal Register** listings at <http://www.epa.gov/fedrgstr/>.

2. *In person.* The Agency has established an official record for this action under docket ID number OPP-2002-0074. The official record consists

of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305-5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket ID number OPP-2002-0074 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305-5805.

3. *Electronically.* You may submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket ID number OPP-2002-0074. Electronic comments

may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.
2. Describe any assumptions that you used.
3. Provide copies of any technical information and/or data you used that support your views.
4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.
5. Provide specific examples to illustrate your concerns.
6. Make sure to submit your comments by the deadline in this notice.
7. To ensure proper receipt by EPA, be sure to identify the docket ID number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA

has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: May 16, 2002.

Debra Edwards,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by Valent U.S.A. Corporation and represents the view of Valent. EPA is publishing the petition summary verbatim without editing it in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

Valent U.S.A. Corporation

PP 2F6385

EPA has received a pesticide petition (2F6385) from Valent U.S.A. Corporation, 1333 North California Boulevard, Suite 600, Walnut Creek, CA 94596-8025 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180, by establishing a tolerance for residues of pyriproxyfen, 2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine, in or on the raw agricultural commodity vegetable, brassica, leafy, group (crop group 5) at 2.5 parts per million (ppm); vegetable, cucurbit, group (crop group 9) at 0.1 ppm; and olive at 1.0 ppm; and in the processed commodity olive, oil at 3.0 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* Metabolism of ^{14}C -pyriproxyfen labelled in the phenoxyphenyl ring and in the pyridyl

ring has been studied in cotton, apples, tomatoes, lactating goats, and laying hens (and rats). The major metabolic pathways in plants is aryl hydroxylation and cleavage of the ether linkage, followed by further metabolism into more polar products by further oxidation and/or conjugation reactions. However, the bulk of the radiochemical residue on raw agricultural commodity samples remained as parent. Comparing metabolites detected and quantified from cotton, apple, tomato, goat, and hen (and rat) shows that there are no significant aglycones in plants which are not also present in the excreta or tissues of animals. The residue of concern is best defined as the parent, pyriproxyfen.

Ruminant and poultry metabolism studies demonstrated that transfer of administered ^{14}C -residues to tissues was low. Total ^{14}C -residues in goat milk, muscle and tissues accounted for less than 2% of the administered dose, and were less than 1 ppm in all cases. In poultry, total ^{14}C -residues in eggs, muscle and tissues accounted for about 2.7% of the administered dose, and were less than 1 ppm in all cases except for gizzard.

2. *Analytical method.* Practical analytical methods for detecting and measuring levels of pyriproxyfen (and relevant metabolites) have been developed and validated in/on all appropriate agricultural commodities, respective processing fractions, milk, animal tissues, and environmental samples. The extraction methodology has been validated using aged radiochemical residue samples from metabolism studies. The methods have been validated in cottonseed, apples, soil, and oranges at independent laboratories. EPA has successfully validated the analytical methods for analysis of cottonseed, pome fruit, nutmeats, almond hulls, and fruiting vegetables. The limit of detection of pyriproxyfen in the methods is 0.01 ppm which will allow monitoring of food with residues at the levels proposed for the tolerances.

3. *Magnitude of residues—i. Vegetable, brassica, leafy, group.* Seven field trials in cabbage were conducted in 1999 and 2000. Similarly, seven field trials were conducted for cauliflower and six field trials were conducted for mustard greens. The proposed use pattern for the three vegetable, brassica, leafy, crops is identical. The analytical data show that the average measured residue in/on cabbage samples was 0.14 ppm ($n = 14$, $\sigma_{n-1} = 0.12$ ppm) pyriproxyfen. Similarly, the analytical data show that the average measured residue in/on cauliflower samples was

0.03 ppm ($n = 14$, $\sigma_{n-1} = 0.05$ ppm), and in/on mustard green samples was 0.70 ppm ($n = 12$, $\sigma_{n-1} = 0.53$ ppm), of pyriproxyfen. The highest average residue (HAR) from field trials was 1.6 ppm. These data support a proposed tolerance for pyriproxyfen in/on the vegetable, brassica, leafy, group at 2.5 ppm.

ii. *Vegetable, cucurbit, group.* Seven field trials in cantaloupe were conducted in 1999 and 2000. Similarly, six field trials were conducted for cucumber and six field trials were conducted for summer squash. The proposed use pattern for the three vegetable, cucurbit, crops is identical. The analytical data show that the average measured residue in/on cantaloupe samples was 0.02 ppm ($n = 14$, $\sigma_{n-1} = 0.01$ ppm) pyriproxyfen. Similarly, the analytical data show that the average measured residue of pyriproxyfen in/on cucumber and summer squash samples was below the residue method "Limit of Detection" of 0.01 ppm. The HAR from field trials was 0.04 ppm. These data support a proposed tolerance for pyriproxyfen in/on the vegetable, cucurbit, group at 0.1 ppm.

iii. *Olive.* Four field trials in olive were conducted in 2000. The analytical data show that the average measured residue in/on olive samples was 0.37 ppm ($n = 8$, $\sigma_{n-1} = 0.24$ ppm) pyriproxyfen. A processing study in olive demonstrated that pyriproxyfen concentrated in olive oil (3-fold). The HAR from field trials was 0.73 ppm. These data support proposed tolerances for pyriproxyfen in/on olive at 1.0 ppm and olive oil at 3.0 ppm.

iv. *Secondary residues.* No additional feed commodities are associated with the new proposed use on vegetable, brassica, leafy, group; vegetable, cucurbit, group; and olive. Using established tolerances to calculate the maximum feed exposure to fed animals, and using the very low potential for residue transfer demonstrated in the milk cow feeding residue study, detectable secondary residues in animal tissues, milk, and eggs are not expected. Therefore, no tolerances are required for these commodities.

v. *Rotational crops.* The results of a confined rotational crops accumulation study indicate that no rotational crop planting restrictions or rotational crop tolerances are required.

B. Toxicological Profile

1. *Acute toxicity.* The acute toxicity of technical grade pyriproxyfen is low by all routes. The compound is classified as Category III for acute dermal and inhalation toxicity, and Category IV for

acute oral toxicity, and skin/eye irritation. Pyriproxyfen is not a skin sensitizing agent.

2. *Genotoxicity.* Pyriproxyfen does not present a genetic hazard. Pyriproxyfen was negative in the following tests for mutagenicity: Ames assay with and without S9, *in vitro* unscheduled DNA synthesis in HeLa S3 cells, *in vitro* gene mutation in V79 chinese hamster cells, and *in vitro* chromosomal aberration with and without S9 in Chinese hamster ovary cells.

3. *Reproductive and developmental toxicity.* Pyriproxyfen is not a developmental or reproductive toxicant. Developmental toxicity studies have been performed in rats and rabbits, and multigenerational effects on reproduction were tested in rats. These studies have been reviewed and found to be acceptable to the Agency.

In the developmental toxicity study conducted with rats, technical pyriproxyfen was administered by gavage at levels of 0, 100, 300, and 1,000 milligrams/kilogram body weight/day (mg/kg bw/day) during gestation days 7–17. Maternal toxicity (mortality, decreased body weight gain and food consumption, and clinical signs of toxicity) was observed at doses of 300 mg/kg bw/day and greater. The maternal no observed adverse effect level (NOAEL) was 100 mg/kg bw/day. A transient increase in skeletal variations was observed in rat fetuses from females exposed to 300 mg/kg bw/day and greater. These effects were not present in animals examined at the end of the postnatal period; therefore, the NOAEL for prenatal developmental toxicity was 100 mg/kg bw/day. An increased incidence of visceral and skeletal variations was observed postnatally at 1,000 mg/kg bw/day. The NOAEL for postnatal developmental toxicity was 300 mg/kg bw/day.

In the developmental toxicity study conducted with rabbits, technical pyriproxyfen was administered by gavage at levels of 0, 100, 300, and 1,000 mg/kg bw/day during gestation days 6–18. Maternal toxicity (clinical signs of toxicity including one death, decreased body weight gain and food consumption, and abortions or premature deliveries) was observed at oral doses of 300 mg/kg bw/day or higher. The maternal NOAEL was 100 mg/kg bw/day. No developmental effects were observed in the rabbit fetuses. The NOAEL for developmental toxicity in rabbits was 1,000 mg/kg bw/day.

In the rat reproduction study, pyriproxyfen was administered in the diet at levels of 0, 200, 1,000, and 5,000 ppm through two generations of rats.

Adult systemic toxicity (reduced body weights, liver and kidney histopathology, and increased liver weight) was produced at the 5,000 ppm dose (453 mg/kg bw/day in males, 498 mg/kg bw/day in females) during the pre-mating period. The systemic NOAEL was 1,000 ppm (87 mg/kg bw/day in males, 96 mg/kg bw/day in females). No effects on reproduction were produced at 5,000 ppm, the highest dose tested (HDT).

4. *Subchronic toxicity.* Subchronic oral toxicity studies conducted with pyriproxyfen technical in the rat, mouse and dog indicate a low level of toxicity. Effects observed at high dose levels consisted primarily of decreased body weight gain; increased liver weights; histopathological changes in the liver and kidney; decreased red blood cell counts, hemoglobin and hematocrit; altered blood chemistry parameters; and, at 5,000 and 10,000 ppm in mice, a decrease in survival rates. The NOAELs from these studies were 400 ppm (23.5 mg/kg bw/day for males, 27.7 mg/kg bw/day for females) in rats, 1,000 ppm (149.4 mg/kg bw/day for males, 196.5 mg/kg bw/day for females) in mice, and 100 mg/kg bw/day in dogs.

In a 4-week inhalation study of pyriproxyfen technical in rats, decreased body weight and increased water consumption were observed at 1,000 mg/m³. The NOAEL in this study was 482 mg/m³.

A 21-day dermal toxicity study in rats with pyriproxyfen technical did not produce any signs of dermal or systemic toxicity at 1,000 mg/kg bw/day, the highest dose tested. In a 21-day dermal study conducted with KNACK® Insect Growth Regulator, the test material produced a NOAEL of 1,000 mg/kg bw/day HDT for systemic effects, and a NOAEL for skin irritation of 100 mg/kg bw/day.

5. *Chronic toxicity.* Pyriproxyfen technical has been tested in chronic studies with dogs, rats and mice. EPA has established a reference dose (RfD) for pyriproxyfen of 0.35 mg/kg bw/day, based on the NOAEL in female rats from the 2-year chronic/oncogenicity study. Effects cited by EPA in the Reference Dose Tracking Report include negative trend in mean red blood cell volume, increased hepatocyte cytoplasm and cytoplasm: nucleus ratios, and decreased sinusoidal spaces.

Pyriproxyfen is not a carcinogen. Studies with pyriproxyfen have shown that repeated high dose exposures produced changes in the liver, kidney and red blood cells, but did not produce cancer in test animals. No oncogenic response was observed in a rat 2-year chronic feeding/oncogenicity study or

in a 78-eight week study on mice. The oncogenicity classification of pyriproxyfen is “E” (no evidence of carcinogenicity for humans).

Pyriproxyfen technical was administered to dogs in capsules at doses of 0, 30, 100, 300, and 1,000 mg/kg bw/day for 1 year. Dogs exposed to dose levels of 300 mg/kg bw/day or higher showed overt clinical signs of toxicity, elevated levels of blood enzymes and liver damage. The NOAEL in this study was 100 mg/kg bw/day.

Pyriproxyfen technical was administered to mice at doses of 0, 120, 600 and 3,000 ppm in diet for 78 weeks. The NOAEL for systemic effects in this study was 600 ppm (84 mg/kg bw/day in males, 109.5 mg/kg bw/day in females), and a LOAEL of 3,000 ppm (420 mg/kg bw/day in males, 547 mg/kg bw/day in females) was established based on an increase in kidney lesions.

In a 2-year study in rats, pyriproxyfen technical was administered in the diet at levels of 0, 120, 600, and 3,000 ppm. The NOAEL for systemic effects in this study was 600 ppm (27.31 mg/kg bw/day in males, 35.1 mg/kg bw/day in females). A LOAEL of 3,000 ppm (138 mg/kg bw/day in males, 182.7 mg/kg bw/day in females) was established based on a depression in body weight gain in females.

6. *Animal metabolism.* The absorption, tissue distribution, metabolism and excretion of ¹⁴C-labeled pyriproxyfen were studied in rats after single oral doses of 2 or 1,000 mg/kg bw (phenoxyphenyl and pyridyl label), and after a single oral dose of 2 mg/kg bw (phenoxyphenyl label only) following 14 daily oral doses at 2 mg/kg bw of unlabelled material. For all dose groups, most (88–96%) of the administered radiolabel was excreted in the urine and feces within 2 days after radiolabeled test material dosing, and 92–98% of the administered dose was excreted within 7 days. Seven days after dosing, tissue residues were generally low, accounting for no more than 0.3% of the dosed ¹⁴C. Radiocarbon concentrations in fat were the higher than in other tissues analyzed. Recovery in tissues over time indicates that the potential for bioaccumulation is minimal. There were no significant sex or dose-related differences in excretion or metabolism.

7. *Metabolite toxicology.* Metabolism studies of pyriproxyfen in rats, goats and hens, as well as the fish bioaccumulation study demonstrate that the parent is very rapidly metabolized and eliminated. In the rat, most (88–96%) of the administered radiolabel was excreted in the urine and feces within 2 days of dosing, and 92–98% of the administered dose was excreted within

7 days. Tissue residues were low 7 days after dosing, accounting for no more than 0.3% of the dosed ^{14}C . Because parent and metabolites are not retained in the body, the potential for acute toxicity from in situ formed metabolites is low. The potential for chronic toxicity is adequately tested by chronic exposure to the parent at the maximum tolerance dose (MTD) and consequent chronic exposure to the internally formed metabolites.

Seven metabolites of pyriproxyfen, 4'-OH-pyriproxyfen, 5''-OH-pyriproxyfen, desphenyl-pyriproxyfen, POPA, PYPAC, 2-OH-pyridine and 2,5-diOH-pyridine, have been tested for mutagenicity (Ames) and acute oral toxicity to mice. All seven metabolites were tested in the Ames assay with and without S9 at doses up to 5,000 micro-grams per plate or up to the growth inhibitory dose. The metabolites did not induce any significant increases in revertant colonies in any of the test strains. Positive control chemicals showed marked increases in revertant colonies. The acute toxicity to mice of 4'-OH-pyriproxyfen, 5''-OH-pyriproxyfen, desphenyl-pyriproxyfen, POPA, and PYPAC did not appear to markedly differ from pyriproxyfen, with all metabolites having acute oral LD_{50} values greater than 2,000 mg/kg bw. The

two pyridines, 2-OH-pyridine and 2,5-diOH-pyridine, gave acute oral LD_{50} values of 124 (male) and 166 (female) mg/kg bw, and 1,105 (male) and 1,000 (female) mg/kg bw, respectively.

8. *Endocrine disruption.* Pyriproxyfen is specifically designed to be an insect growth regulator and is known to produce juvenoid effects on arthropod development. However, this mechanism-of-action in target insects and other some arthropods has no relevance to any mammalian endocrine system. While specific tests, uniquely designed to evaluate the potential effects of pyriproxyfen on mammalian endocrine systems have not been conducted, the toxicology of pyriproxyfen has been extensively evaluated in acute, sub-chronic, chronic, developmental, and reproductive toxicology studies including detailed histopathology of numerous tissues. The results of these studies show no evidence of any endocrine-mediated effects and no pathology of the endocrine organs. Consequently, it is concluded that pyriproxyfen does not possess estrogenic or endocrine disrupting properties applicable to mammals.

C. Aggregate Exposure

1. *Dietary exposure.* An evaluation of chronic dietary exposure including both

food and drinking water has been performed for the U.S. population and various sub-populations including infants and children. No acute dietary endpoint; and dose was identified in the toxicology data base for pyriproxyfen; therefore, the Agency has concluded that there is a reasonable certainty of no harm from acute dietary exposure.

i. *Food.* Chronic dietary exposure to pyriproxyfen residues was calculated for the U.S. population and 25 population subgroups assuming tolerance level residues, processing factors from residue studies, and 100% of the crop treated. The analyses included residue data for all existing uses, pending uses, and proposed new uses. The results from several representative subgroups are listed below. Chronic exposure to the overall U.S. population is estimated to be 0.002984 mg/kg bw/day, representing 0.9% of the RfD. For the most highly exposed sub-population, children 1 to 6 years of age, exposure is calculated to be 0.007438 mg/kg bw/day, or 2.1% of the RfD. Generally speaking, the Agency has no cause for concern if total residue contribution for established and proposed tolerances is less than 100 percent of the RfD.

CALCULATED CHRONIC DIETARY EXPOSURES TO THE TOTAL U.S. POPULATION AND SELECTED SUB-POPULATIONS TO PYRIPROXYFEN RESIDUES IN FOOD

Population subgroup	Exposure (mg/kg bw/day)	Percent of RfD
Total U.S. population (all seasons)	0.002984	0.853
Children (1–6 Years)	0.007438	2.125
Non-Nursing Infants (<1 Year Old)	0.006483	1.852
All Infants (<1 Year Old)	0.005604	1.601
Children (7–12 Years)	0.004159	1.188
Children (1–6 Years)	0.007438	2.125
Females (13+/Nursing)	0.002964	0.847
Nursing Infants (<1 Year Old)	0.002601	0.743

ii. *Drinking water.* Since pyriproxyfen is applied outdoors to growing agricultural crops, the potential exists for pyriproxyfen or its metabolites to reach ground or surface water that may be used for drinking water. Because of the physical properties of pyriproxyfen, it is unlikely that pyriproxyfen or its metabolites can leach to potable ground water. To quantify potential exposure from drinking water, surface water concentrations for pyriproxyfen were

estimated using GENEEC 1.3. The average 56-day concentration predicted in the simulated pond water was 0.16 ppb. Using standard assumptions about body weight and water consumption, the chronic exposure to pyriproxyfen from this drinking water would be 4.57×10^{-6} and 1.6×10^{-5} mg/kg bw/day for adults and children, respectively; 0.0046% of the RfD (0.35 mg/kg/day) for children. Based on this worse case

analysis, the contribution of water to the dietary risk is negligible.

2. *Non-dietary exposure.* Pyriproxyfen is currently registered for use on residential non-food sites. Pyriproxyfen is the active ingredient in numerous registered products for flea and tick control. Formulations include foggers, aerosol sprays, emulsifiable concentrates, and impregnated materials (pet collars). With the exception of the pet collar uses, consumer use of

pyriproxyfen typically results in acute and short-term intermittent exposures. No acute dermal, or inhalation dose or endpoint was identified in the toxicity data for pyriproxyfen. Similarly, doses and endpoints were not identified for short- and intermediate-term dermal or inhalation exposure to pyriproxyfen. The Agency has concluded that there are reasonable certainties of no harm from acute, short-term, and intermediate-term dermal and inhalation occupational and residential exposures due to the lack of significant toxicological effects observed.

Chronic residential post-application exposure and risk assessments were conducted to estimate the potential risks from pet collar uses. The risk assessment was conducted using the following assumptions: Application rate of 0.58 mg a.i./day (product label), average body weight for a 1–6 year old child of 10 kg, the active ingredient dissipates uniformly through 365 days (the label instruct to change collar once a year), 1% of the active ingredient is available for dermal and inhalation exposure per day (assumption from Draft EPA Standard Operating Procedures (SOPs) for Residential Exposure Assessments, December 18, 1997). The assessment also assumes an absorption rate of 100%. This is a conservative assumption since the dermal absorption was estimated to be 10%. The estimated chronic term MOE was 61,000 for children, and 430,000 for adults. The risk estimates indicate that potential risks from pet collar uses do not exceed the Agency's level of concern.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that the Agency must consider "available information" concerning the cumulative effects of a particular pesticide's residues and "other substances that have a common mechanism of toxicity." Available information in this context include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not, at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way.

There are no other pesticidal compounds that are structurally related

to pyriproxyfen and have similar effects on animals. In consideration of potential cumulative effects of pyriproxyfen and other substances that may have a common mechanism of toxicity, there are currently no available data or other reliable information indicating that any toxic effects produced by pyriproxyfen would be cumulative with those of other chemical compounds. Thus, only the potential risks of pyriproxyfen have been considered in this assessment of aggregate exposure and effects.

Valent will submit information for EPA to consider concerning potential cumulative effects of pyriproxyfen consistent with the schedule established by EPA at (62 FR 42019) (FRL-5734-6) August 4, 1997 and other subsequent EPA publications pursuant to the Food Quality Protection Act.

E. Safety Determination

1. *U.S. population—i. Chronic dietary exposure and risk.—Adult sub-populations.* The results of the chronic dietary exposure assessment described above demonstrate that estimates of chronic dietary exposure for all existing, pending and proposed uses of pyriproxyfen are well below the chronic RfD of 0.35 mg/kg bw/day. The estimated chronic dietary exposure from food for the overall U.S. population and many non-child/infant subgroups is from 0.002123 to 0.003884 mg/kg bw/day, 0.607 to 1.100% of the RfD. Addition of the small but worse case potential chronic exposure from drinking water (calculated above) increases exposure by only 4.57×10^{-6} mg/kg bw/day and does not change the maximum occupancy of the RfD significantly. Generally, the Agency has no cause for concern if total residue contribution is less than 100% of the RfD. It can be concluded that there is a reasonable certainty that no harm will result to the overall U.S. population or any non-child/infant subgroups from aggregate, chronic dietary exposure to pyriproxyfen residues.

ii. *Acute dietary exposure and risk.—Adult sub-populations.* No acute dietary endpoint and dose were identified in the toxicology data base for pyriproxyfen; therefore, it can be concluded that there is a reasonable certainty that no harm will result to the overall U.S. population or any non-child/infant subgroups from aggregate, acute dietary exposure to pyriproxyfen residues.

iii. *Non-dietary exposure and aggregate risk.—Adult sub-populations.* Acute, short-term, and intermediate-term dermal and inhalation risk assessments for residential exposure are not required due to the lack of

significant toxicological effects observed. The results of a chronic residential post-application exposure and risk assessment for pet collar uses demonstrate that potential risks from pet collar uses do not exceed the Agency's level of concern. The estimated chronic term MOE for adults was 430,000.

2. *Infants and children—i. Safety factor.* In assessing the potential for additional sensitivity of infants and children to residues of pyriproxyfen, FFDCA section 408 provides that EPA shall apply an additional margin of safety, up to ten-fold, for added protection for infants and children in the case of threshold effects unless EPA determines that a different margin of safety will be safe for infants and children.

The toxicological data base for evaluating prenatal and postnatal toxicity for pyriproxyfen is complete with respect to current data requirements. There are no special prenatal or postnatal toxicity concerns for infants and children, based on the results of the rat and rabbit developmental toxicity studies or the 2-generation reproductive toxicity study in rats. Valent concludes that reliable data support use of the standard 100-fold uncertainty factor and that an additional uncertainty factor is not needed for pyriproxyfen to be further protective of infants and children.

ii. *Chronic dietary exposure and risk.* Using the conservative exposure assumptions described above, the percentage of the RfD that will be utilized by chronic dietary (food only) exposure to residues of pyriproxyfen ranges from 0.002601 mg/kg bw/day for nursing infants, up to 0.007438 mg/kg bw/day for children (1–6 years of age), 0.743 to 2.125% of the RfD, respectively. Adding the worse case potential incremental exposure to infants and children from pyriproxyfen in drinking water (1.6×10^{-5} mg/kg bw/day) does not materially increase the aggregate, chronic dietary exposure and only increases the occupancy of the RfD by 0.0046% to 2.130% for children (1–6 years of age). EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. It can be concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate, chronic dietary exposure to pyriproxyfen residues.

iii. *Acute dietary exposure and risk.* No acute dietary endpoint and dose were identified in the toxicology data

base for pyriproxyfen; therefore, it can be concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate, acute dietary exposure to pyriproxyfen residues.

iv. *Non-dietary exposure and aggregate risk.* Acute, short-term, and intermediate-term dermal and inhalation risk assessments for residential exposure are not required due to the lack of significant toxicological effects observed. The results of a chronic residential post-application exposure and risk assessment for pet collar uses demonstrate that potential risks from pet collar uses do not exceed the Agency's level of concern. The estimated chronic term MOE for children was 61,000.

F. International Tolerances

There are no presently existing Codex maximum residue levels maximum residue levels for pyriproxyfen.

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BILLING CODE 6560-50-S

ENVIRONMENTAL PROTECTION AGENCY

[FRL-7218-9]

Agreement and Covenant Not To Sue, Sharon Steel Superfund Site, Midvale, UT

AGENCY: Environmental Protection Agency (U.S. EPA).

ACTION: Notice; Agreement and Covenant Not to Sue.

SUMMARY: In accordance with the requirements of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended ("CERCLA"), 42 U.S.C. 9601 *et seq.*, notice is hereby given of an Agreement and Covenant Not to Sue ("Agreement"), also known as a Prospective Purchaser Agreement ("PPA"), concerning the Sharon Steel Superfund Site in Midvale, Utah (the "Site"). The Agreement resolves any potential liability for response costs incurred and to be incurred by the United States Environmental Protection Agency ("EPA") and the State of Utah Department of Environmental Quality that may be acquired by the City of Midvale, UT when it takes title to certain permanent easements that traverse the Sharon Steel Superfund Site. The City of Midvale is taking title to these easements in order to construct the Provo/Jordan River Parkway Pedestrian & Bicycle Trail and the Bingham Junction Parkway.

DATES: Comments must be submitted to EPA on or before June 28, 2002.

ADDRESSES: Comments should be addressed to Nancy A. Mangone, (8ENF-L), Enforcement Attorney, U.S. Environmental Protection Agency, Region VIII, 999 18th Street, Suite 300, Denver, Colorado 80202-2466, and should refer to: In the Matter of: Sharon Steel Superfund Site Agreement and Covenant Not To Sue Midvale City.

FOR FURTHER INFORMATION CONTACT:

Nancy A. Mangone, (8ENF-L), Enforcement Attorney, U.S. Environmental Protection Agency, Region VIII, 999 18th Street, Suite 300, Denver, Colorado 80202-2466, (303) 312-6903.

SUPPLEMENTARY INFORMATION: Notice of Agreement and Covenant Not to Sue with the City of Midvale for the Sharon Steel Superfund Site: In accordance with CERCLA, 42 U.S.C. 9601, *et seq.*, notice is hereby given that the terms of a Prospective Purchaser Agreement and a covenant not to sue have been agreed to by the United States, Utah Department of Environmental Quality and the City of Midvale.

By the terms of the proposed Agreement, the City will acquire permanent public easements and access rights across certain portions of the Site in order to: (1) Construct a non-motorized, multiple-use recreational trail along the western edge of the Site, from 7980 South to 8500 South, known as the Provo/Jordan River Parkway Pedestrian & Bicycle Trail, including an access road known as the Oxbow Road, (collectively, the "Parkway Trail"); and (2) construct a new north/south road, the Bingham Junction Parkway, from 7800 South at approximately 1000 West, across the eastern portion of the Site, to Sandy Parkway ("Bingham Junction Parkway").

The PPA provides the City with covenants not to sue from EPA and UDEQ for liability for the existing contamination that has already been addressed by the remedial action performed at the Site in accordance with section 107(a) of CERCLA, 42 U.S.C. 9607(a) and the Utah Hazardous Substance Mitigation Act, section 19-6-301, *et seq.*, Utah Code Ann. The City will also receive contribution protection under section 113 CERCLA, 42 U.S.C. 9613, for claims that could be brought against it by third parties. In consideration for these covenants not to sue, the City has agreed to perform operation and maintenance ("O&M") activities on that portion of the Sharon Steel Site it is acquiring, which amounts to approximately 15 acres of the Site. The current annualized value of the

performance of these O&M activities is estimated to be \$22,505 for the Bingham Junction Parkway and \$4,938 for the Parkway Trail. The City is also providing O&M for the access road to the Site, known as Oxbow Road.

U.S. EPA will receive, for a period of thirty (30) days from the date of this publication, comments relating to the Agreement and Covenant Not to Sue for the Sharon Steel Superfund Site. A copy of the PPA may be obtained in person or by mail from Mike Rudy, Enforcement Specialist (ENF-T), Environmental Protection Agency, Region VIII, 999 18th Street, Suite 300, Denver, Colorado 80202-2466, (303) 312-6332.

Dated: May 17, 2002.

Michael T. Risner,

Acting Assistant Regional Administrator, Office of Enforcement, Compliance and Environmental Justice.

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BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

[FRL-7219-1]

Koppers Company Inc., (Morrisville Plant) Superfund Site; Notice of Proposed Settlement

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice; request for public comment.

SUMMARY: The United States Environmental Protection Agency is proposing to enter into a settlement with Beazer East Inc., pursuant to 122(h) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended, regarding Beazer East Inc., located in Morrisville, Wake County, North Carolina. EPA will consider public comments on the proposed settlement for thirty (30) days. EPA may withdraw from or modify the proposed settlement should such comments disclose facts or considerations which indicate the proposed settlement is inappropriate, improper or inadequate. Copies of the proposed settlement are available from: Ms. Paula V. Batchelor, U.S. EPA Region 4, Waste Management Division, 61 Forsyth Street SW., Atlanta, Georgia 30303, 404/562-8887.

Written comments may be submitted to Ms. Batchelor within 30 calendar days of the date of this publication.