based on the completeness and reliability of the toxicity data supporting these petitions, there is a reasonable certainty that no harm will result from aggregate exposure to residues of fludioxonil as a result of these requested tolerances.

2. *Infants and children*. Infants and children are not expected to show any particular sensitivity to fludioxonil. This can be demonstrated by referencing several data points, including the equivalence of the maternal and fetal toxicity NOAEL in the fludioxonil 2–generation rat study.

i. *Acute risk*. The risk from acute dietary exposure to fludioxonil is considered to be very low. Under the highly conservative exposure assumptions of residue levels being at tolerance level and 100% market share for the majority of crops with proposed and established fludioxonil registrations, the utilization of the acute RfD of the most exposed group is 83.4% (children, 1–6 years).

ii. *Chronic risk*. Using highly conservative aggregate exposures 23.0% and 19.2% of the RfD were obtained for the most sensitive sub-populations, non-nursing infants (< 1–year old) and children (1–6 years), respectively. Therefore, a reasonable certainty exists that no harm will result from aggregate exposure to fludioxonil if the proposed uses are registered.

F. International Tolerances

There are no Codex maximum residue levels established for residues of fludioxonil in or on strawberrry, dry bulb onion, green onion, and stone fruit crop fruit.

[FR Doc. 00–7740 Filed 3–28–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

PF–919; FRL–6493–8

Notice of Filing Pesticide Petitions To Establish a Tolerance for Certain Pesticide Chemicals in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket control number PF–919, must be received on or before April 28, 2000.

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 control number PF–919 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number/e-mail address	Address	Petition number(s)
Mary Waller (PM 21)	Rm. 249, CM #2, 703-308-9354, e-mail:waller.mary@epamail.epa.gov	1921 Jefferson Davis Hwy, Arlington, VA	PP 9F3727
Joe Travano (PM 10)	Rm. 214, CM #2, 703–305–6411, e-mail: travano.joe@epamail.epa.gov.	Do.	PP 0F6069

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'' 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 control number PF–919. 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 control number PF–919 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, Ariel Rios Bldg., 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 control number PF–919. 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 control 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 pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain 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 supports 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: March 16, 2000.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. 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.

1. McLaughlin Gormley King Company

0F6069

EPA has received a pesticide petition (0F6069) from McLaughlin Gormley King Company, 8810 Tenth Avenue North, Minneapolis, MN 55427-4372 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 in or on food products in food handling establishments at 0.1 parts per million (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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Radiocarbon plant and animal metabolism studies have been conducted with pyriproxyfen. These studies demonstrate that the nature of the residues in these matrices is primarily pyriproxyfen.

2. *Analytical method*. An analytical method is available to detect residues of pyriproxyfen in or on food commodities. Pyriproxyfen can be extracted from samples and analyzed by high performance liquid chromotography (HPLC), or nitrogen phosphorous/gas liquid chromotography (NP-GLC). The HPLC method has been validated by an independent laboratory.

3. *Magnitude of residues*. Studies were conducted to determine levels of residues resulting from the application of Nylar to representative food commodities in simulated feed and/or food processing and simulated warehouse situations. The representative foods were potatoes, loaves of bread, flour, lettuce, meat, candy, butter, banana cream pies, navy beans, Spanish peanuts, dried prunes, and granulated sugar. No significant residues were found in covered samples; however, residues were detectable in uncovered samples and samples with permeable wrapping.

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 (UDS) in HeLa S3 cells, *in vitro* gene mutation in V79 Chinese hamster cells (CHO), and *in vitro* chromosomal aberration with and without S9 in CHO 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 multi-generational 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/kilograms bodyweight/day (mg/kg bwt/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 bwt/day and greater. The maternal no observed adverse effect level (NOAEL) was 100 mg/kg bwt/day. A transient increase in skeletal variations was observed in rat fetuses from females exposed to 300 mg/kg bwt/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 bwt/day. An increased incidence of visceral and skeletal variations was observed postnatally at 1,000 mg/kg bwt/day. The NOAEL for postnatal developmental toxicity was 300 mg/kg bwt/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 bwt/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 bwt/day or higher. The maternal NOAEL was 100 mg/kg bwt/day. No developmental effects were observed in the rabbit fetuses. The NOAEL for developmental toxicity in rabbits was > 1,000 mg/kg bwt/day.

In the rat reproduction study, pyriproxyfen was administered in the diet at levels of 0, 200, 1,000, and 5,000 ppm through 2 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 bwt/day in males, 498 mg/kg bwt/day in females) during the pre-mating period. The systemic NOAEL was 1,000 ppm (87 mg/kg bwt/day in males, 96 mg/kg bwt/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 bwt/day for males, 27.7 mg/kg bw/day for females) in rats, 1,000 ppm (149.4 mg/kg bwt/day for males, 196.5 mg/kg bwt/day for females) in mice, and 100 mg/kg bwt/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/m^3. The NOAEL in this study was 482 mg/m^3.

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 bwt/day, the HDT.

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 bwt/day, based on the NOAEL in female rats from the 2-year chronic/oncogenicity study. Effects cited by EPA in the RfD 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-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 bwt/day for 1-year. Dogs exposed to dose levels of 300 mg/kg bwt/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 bwt/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 bwt/day in males, 109.5 mg/kg bwt/day in females), and a lowest observed adverse effect level (LOAEL) of 3,000 ppm (420 mg/kg bwt/day in males, 547 mg/kg bwt/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 bwt/day in males, 35.1 mg/kg bwt/day in females). A LOAEL of 3,000 ppm (138 mg/kg bwt/day in males, 182.7 mg/kg bwt/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 ^{14}C-labeled pyriproxyfen were studied in rats after single oral doses of 2 or 1,000 mg/kg bwt (phenoxyphenyl and pyridyl label), and after a single oral dose of 2 mg/kg bwt (phenoxyphenyl label only) following 14 daily oral doses at 2 mg/kg bwt of unlabeled 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 ^{14}C. Radiocarbon concentrations in fat were 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 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 micrograms 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 bwt. 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 bwt, and 1,105 (male) and 1,000 (female) mg/kg/bwt, 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 some other 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*—i. *Food*. An evaluation of chronic dietary exposure to potential pyriproxyfen residues in all foods that may be exposed to pyriproxyfen through agricultural and food handling establishment treatments, including exposure from drinking water, was estimated for the overall U.S. population and 26 sub-populations, including infants and children.

Chronic dietary exposure was estimated using the chronic module of the DEEM™ software. Residue data used in the analysis included current and pending tolerances for agricultural crops, results from warehouse simulation studies, and processing data. The data base providing levels of food consumption was the USDA Continuing Surveys of Food Intake by Individuals conducted from 1994 through 1996. MGK provided estimated marketshare information.

Chronic dietary exposure was estimated to be 0.000550 mg/kg bwt/day, or 0.2% of the RfD. Exposure for the most highly exposed population subgroup, non-nursing infants, was calculated to be 0.002438 mg/kg bwt/day, or 0.7% of the RfD.

ii. *Drinking water*. The generic expected environmental concentration (GENEEC) modeling was used to estimate potential pyriproxyfen residues in surface water and/or ground water. The chronic drinking water estimated concentration value of 0.053 parts per billion (ppb) for pyriproxyfen was compared to the drinking water levels of concern (DWLOC) calculated for pyriproxyfen for adult males, adult females, and toddlers, that were 12,545 ppb, 10,489 ppb, and 5,229 ppb, respectively. There is reasonable certainty that no harm will result from aggregate exposure to potential pyriproxyfen residues.

2. *Non-dietary exposure*. Many products for indoor, non-food applications such as pet care products and carpet treatments containing pyriproxyfen as an active ingredient are registered with EPA. Typically, the directions for use of these products describe intermittent application, with no resulting chronic exposures. Since neither acute oral, dermal, or inhalation toxicity endpoints, nor doses and endpoints for short- and intermediate-term dermal or inhalation exposures have been identified for pyriproxyfen, the Agency has concluded that there is reasonable certainty of no harm from non-dietary exposures to pyriproxyfen.

D. Cumulative Effects

There are no other compounds that are structurally related to pyriproxyfen and have similar effects on animals. No other data are available that indicate that any toxicological effects produced by pyriproxyfen would be cumulative with those of any other compound, so only the potential risks of pyriproxyfen have been considered in the risk assessment.

E. Safety Determination

1. *U.S. population*. Based on the estimated aggregate exposures to residues of pyriproxyfen from food and drinking water, and the reliable toxicology data base, the chronic exposure to pyriproxyfen for the overall U.S. population is 0.000550 mg/kg bwt/day, representing only 0.2% of the RfD. EPA has no concerns about exposure which are less than 100% of the RfD as 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 is therefore, safe to conclude that there is reasonable certainty that no harm to the overall U.S. population will result from chronic exposure to pyriproxyfen residues.

2. *Infants and children*. EPA has the right to apply an additional margin of safety, up to ten-fold, for the protection of infants and children due to their additional sensitivities, unless EPA can determine that a different margin of safety will adequately protect them. Rat and rabbit developmental toxicity studies and the 2–generation reproductive toxicity study in rats demonstrated that no special prenatal or postnatal toxicity concerns apply for exposure to pyriproxyfen. Therefore, an additional uncertainty factor does not need to be added for the safety determination of pyriproxyfen.

Based on the estimated aggregate exposures to residues of pyriproxyfen from food and drinking water, and the reliable toxicology data base, the chronic exposure to pyriproxyfen for infants and children ranged from 0.000739 mg/kg bwt/day for children 7–12 years old, representing 0.2% of the RfD, to 0.002438 mg/kg bwt/day for non-nursing infants < 1–year old, representing 0.7% if the RfD. It is safe to conclude that there is reasonable certainty that no harm to any subgroup of children will result from chronic exposure to pyriproxyfen residues.

F. International Tolerances

No Codex MRLs presently exist for pyriproxyfen, although they may be established in the future.

2. Uniroyal Chemical Company, Inc.

9F3727

EPA has received a pesticide petition (9F3727) from Uniroyal Chemical Company Inc., 74 Amity Rd, Bethany, CT proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by

establishing a tolerance for residues of carboxin (5,6-dihydro-2-methyl-1,4-oxathiin-3-carboxanilide) and its sulfoxide metabolite (5,6-dihydro-3-carboxanilide-2-methyl-1,4-oxathiin-4-oxide) in or on the RAC onions (dry bulb) at 0.2 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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of carboxin in plants is adequately understood. The major metabolites in all commodities of wheat were carboxin sulfoxide and sulfone. Metabolites in cotton seeds were at too low a level to be identified. The metabolism of carboxin in soybeans is characterized by the oxidation of sulfur (present as sulfoxides and sulfones), cleavage of the oxathiin ring, and conjugation with glucose.

2. *Analytical method*. The analytical method employed for analysis of residues of carboxin in the onions from the trials described below used a caustic reducing medium to hydrolyze extracted residues of carboxin and its sulfoxide metabolite to liberate aniline, which is distilled and concentrated. The aniline is analyzed with a gas chromatograph equipped with a microcoulometric nitrogen detector. The limit of detection by this method is 0.1 ppm. The current method for the analysis of residues of carboxin in animal tissues, milk and eggs employs alkaline hydrolysis with the liberated aniline derivatized with heptafluorobutyric anhydride. Analysis is by gas chromatography of the derivatized aniline, with mass selective detection (GC/MSD). Thus the sensivity of the method limit of quantitation (LOQ) in all tissue was 0.02 ppm, and the precision of the method as indicated by the coefficient of variation (COV) was 1.9%.

3. *Magnitude of residues*. Uniroyal Chemical Company has submitted data to determine residues of carboxin in mature onions grown from seed, which was treated prior to planting with PRO-GRO. Nine trials were conducted in the following States; Michigan (3), Oregon (2), Washington (1), New York (2), Illinois (1), and one trial was conducted in Ontario, Canada. At each trial site onion seed, which had been treated with 2.5 lbs. PRO-GRO containing 0.75 lbs. active ingredient per 100 lbs. seed (1x the label rate), was planted and onions were grown to maturity. Mature onions, depending upon variety, were harvested from 118 days to 197 days after treatment. Residues of carboxin, and its sulfoxide metabolite, both quantitated as carboxin, were as follows. Seventeen of 18 onion samples grown from seed treated at the 1x rate had residues of total carboxin less than the limit of detection of 0.1 ppm. One sample had a total carboxin residue value of 0.1 ppm. One onion sample grown from seed which had been treated with PRO-GRO at 2x the label rate had no carboxin residues above the 0.1 ppm limit of detection. The submitted field trial data indicate that residues of carboxin will not exceed the proposed tolerance of 0.2 ppm in mature onions grown from seed which had been treated with PRO-GRO at the label rate.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicity studies on carboxin demonstrate that the oral and dermal LD_{50} values for the technical material are 2.86 and > 4.0 g/kg, respectively. The 4-hour inhalation LC_{50} in rats is 4.7 milligrams/Liter (mg/L). Irritation tests in rabbits showed carboxin to be a mild eye irritant and non-irritating to the skin. Carboxin did not cause skin sensitization in studies with guinea pigs.

2. *Genotoxicity*. Bacterial/mammalian microsomal mutagenicity assays were performed and carboxin was found not to be mutagenic. Two chromosomal aberration assays were conducted, in CHO cells and in mouse bone marrow *in vivo*, and were also negative. A study was performed in rat hepatocytes and demonstated the induction of UDS.

3. *Reproductive and developmental toxicity*. In a developmental toxicity study in rats conducted in 1989, carboxin was administered by oral gavage to pregnant, Sprague Dawley rats at dosage levels of 10, 90, and 175 mg/kg/day. Decreased maternal body weight gain was seen at dose levels of 90 and 175 mg/kg/day. The report states that there was a slightly reduced mean fetal body weight in the high dose group compared to controls (3.3 g vs. 3.5 g). However, a recent evaluation of 59 studies of the historical control data in the final report shows that between 10/83 and 4/87, the range for fetal weight was 3.1 g to 5.1 g. Therefore, a mean fetal weight of 3.3 g in the 175 mg/kg/day group is within the historical control range. Maternal toxicity was also noted at this dosage level. Therefore, the NOAEL for developmental toxicity is greater than 175 mg/kg/day and the NOAEL for maternal toxicity, based on decreased body weight gain, is 10 mg/kg/day.

In a developmental toxicity study in rabbits, carboxin was administered by oral gavage to pregnant White rabbits at dosage levels of 75, 375, and 750 mg/kg/day. There were no treatment related effects at any dose level with the exception of three abortions in the high dose group and one abortion in the mid dose group. An evaluation of historical control data from 28 studies conducted at that time shows abortion rates of 3/17, and 5/16 in two studies, as well as a number of studies in which there were one or two abortions each. Therefore, considering that there was no maternal toxicity at dose levels of 375 or 750 mg/kg/day of carboxin, it would have to be concluded that the 1/16 and 3/16 abortions seen in the mid and high dose groups were spontaneous. The NOAEL for maternal and developmental toxicity was considered to be greater than 750 mg/kg/day.

In a dietary 2-generation rat reproduction study, carboxin was fed to male and female Sprague Dawley rats at dietary concentrations of 20, 200, and 400 ppm in males, and 20, 300 and 600 ppm in females. At the high dose level there was a decrease in body weight gain in parental males and females and a reduction in pup growth during lactation. No effects on reproduction were observed. The NOAEL for systemic, adult toxicity was 200 ppm (10 mg/kg/day). The NOAEL for offspring growth was 300 ppm (15 mg/kg/day) and the NOAEL for reproductive effects was greater than 400 ppm (20 mg/kg/day).

4. *Subchronic toxicity*. A 13-week rat feeding study was conducted at dietary concentrations of 200, 800, and 2,000 ppm. A reduction in body weight gain was seen in males at 800, and 2,000 ppm, and in females at 2,000 ppm. A reduction in blood levels of glucose, protein and/or globulin was seen in males at 800, and/or 2,000 ppm, and an increase in urea nitrogen was seen in females at 2,000 ppm. Nephritis was seen in males and females given 800 and 2,000 ppm and in males given 200 ppm. The NOAEL for subchronic toxicity in rats was 200 ppm (10 mg/kg/day) in females and less than 200 ppm in males.

5. *Chronic toxicity*. Carboxin was fed to Beagle dogs for 1–year at dietary concentrations of 40, 500 and 7,500 ppm. There was a reduction in body weight gain in female dogs at dose levels of 500 and 7,500 ppm. At a dose level of 7,500 ppm, there was a decreased hematocrit in males and an increase in serum alkaline phosphatase

in males and females. The NOAEL for chronic toxicity was 1 mg/kg/day.

Carboxin was fed to Sprague Dawley rats for 2 years at dietary concentrations of 20, 200, and 400 ppm in males, and 20, 300, and 600 ppm in females in a study completed in 1991. Survival was reduced in high dose males and body weight gain was significantly reduced in high dose males and females. Chronic nephritis was seen in mid and high dose rats, and this effect was more severe in males. There was no treatment related increase in tumor incidence in rats. The NOAEL for chronic toxicity was 1 mg/kg/day.

Carboxin was fed to B6C3F1 mice for 18 months at dietary concentrations of 50, 2,500, and 5,000 ppm. At dosage levels of 2,500, and 5,000 ppm there was in increased incidence of liver hypertrophy. There was no treatment related increase in tumor incidence.

6. *Animal metabolism*. In the rat metabolism study, the percentage of dose did not exceed 0.21% in any tissue and the total percentage of dose in all tissues was 0.26-0.40%. The majority of the dose was excreted in the urine (about 80% within 72 hours). The predominant metabolite was *p*-hydroxy carboxin sulfoxide and the other major metabolite was 4-acetamidophenol. Unchanged carboxin was not detected in the excreta.

7. *Metabolite toxicology*. Although no toxicology studies have been conducted on carboxin metabolites per se, none of these would be expected to have significant toxicity. The residue of concern is the parent compound only.

8. *Endocrine disruption*. No specific studies have been conducted to evaluate potential estrogenic or endocrine effects; however, the standard battery of required studies has not demonstrated any evidence which is suggestive of hormonal effects. Evaluation of the rat multigenerational study demonstrated no effect on the time to mating or on the mating and fertility indices. Chronic and subchronic toxicity studies in rats and dogs did not demonstrate any evidence of toxicity to the male or female reproductive tract or to any endocrine organ associated with endocrine disruption.

C. Aggregate Exposure

1. *Dietary exposure*. The potential dietary exposure from food was assessed using the conservative assumptions that all residues would be at tolerance levels (existing tolerances and the proposed onion tolerance) and that all the commodities would contain residues (100% crop treated). Since onions are not a livestock feed item, the existing tolerances for animal commodities would be adequate.

i. *Food*. The dietary exposure estimate was determined using the tolerance assessment system (TAS) exposure 1® software (1977 food consumption data). The chronic RfD used in the analysis was 0.01 mg/kg/day, based on the NOAEL of 1 mg/kg/day in the rat and dog chronic studies and a 100-fold safety factor. The calculated exposure contribution from carboxin use on onions to the general population was 0.000021 mg/kg/day, 0.21% of the RfD. Infant exposure was 0.000008 mg/kg/day, < 0.1% of the RfD. For the population subgroup children 1–6, the exposure contribution from carboxin was 0.000036 mg/kg/day, 0.36% of the RfD. Total estimated dietary exposure to the general population from the combined existing carboxin uses and the proposed use on onions was determined as 0.001037 mg/kg/day (10.4% of the RfD). For infants and children, the exposure was 0.002444 mg/kg/day (24.4% of the RfD) and 0.002245 mg/kg/day (22.4% of the RfD), respectively.

ii. *Drinking water*. There are no established MCLs for residues of carboxin in drinking water. Health advisory (HA) levels for carboxin in drinking water for adults are 4 and 0.7 mg/L (longer term and life time HA levels respectively) and 1 day, 10 day, and longer term HA levels are all 1 mg/L for children. Seed treatment uses do not typically require a drinking water assessment. Use of carboxin as a seed treatment (at an application rate of < one half ounce active ingredient per acre) is not expected to impact ground water or surface waters or result in significant human exposure. The estimated acute and chronic DWLOC were compared to estimated maximum acute and chronic concentrations of carboxin in surface and ground water from the proposed onion use, as calculated using GENEEC and screning concentration in ground water (SCI-GRO) models. These maximum estimates were well below the DWLOC values by 2-6 orders of magnitude, indicating carboxin would not pose a drinking water concern.

2. *Non-dietary exposure*. Carboxin is registered only for commercial agricultural use, and not for homeowner use. Therefore, non-occupational exposure to the general population from carboxin is unlikely, and is not considered in the aggregate exposure assessments.

D. Cumulative Effects

The potential for cumulative effects of carboxin and other substances that have a common mechanism was considered. The mammalian toxicity of carboxin is well defined, with the kidney being identified as target organ. However, since the biochemical mechanism of toxicity of this compound is not known, it cannot be determined if toxic effects produced by carboxin would be cumulative with any other chemical compound. Thus, only the potential risk of carboxin is considered in the aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Exposure to carboxin would occur primarily from the dietary route. Maximum theoretical levels of carboxin in drinking water were well below drinking water levels of concern for adults and children. Non-occupational exposure to the general population is not expected. Because calculation of the dietary exposure used tolerance levels for all crops and animal commodities and assumed 100% of the crop was treated, the exposure values are considered to be overestimates. Consideration of anticipated residues and actual percent crop treated would likely result in a significantly lower dietary exposure.

Chronic dietary exposure to the general U.S. population from existing uses and the proposed onion use of carboxin was 10.4% of the RfD. For infants and children, the exposure was 24.4% and 22.4% of the RfD, respectively. Therefore, there is a reasonable certainty that no harm will result from dietary exposure to carboxin residues.

2. *Infants and children*. The potential for carboxin to induce toxic effects in children at a greater sensitivity than the general population has been assessed by the rat and rabbit developmental and 2–generation reproduction studies. There was no evidence of embryotoxicity or teratogenicity, and no effects on reproductive parameters as a result of carboxin exposure. The lowest NOAEL for any developmental effect in these studies (15 mg/kg/day reduced pup growth during lactation in the rat reproduction study) is considerably greater than the NOAEL for systemic toxicity in rats (1 mg/kg/day for nephritis in the rat chronic feeding study) which demonstrates that there is no prenatal or postnatal sensitivity to carboxin. Therefore, it is inappropriate to assume that infants and children are more sensitive than the general population to the effects from exposure to carboxin residues.

F. International Tolerances

A MRL has not been established for carboxin by the Codex Alimentarius Commission.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–00629; FRL–6499–9]

Indoor Residential Insecticide Product Label Statements; Notice of Availability

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the availability of a draft Pesticide Registration (PR) Notice that is part of EPA's continuing effort to reduce unnecessary exposures of human and pets to insecticides used in residential settings and to improve the safety of their use. The draft PR Notice is a guidance document, the intent of which is to clarify certain portions of residential insecticide product labels. In addition to helping reduce unnecessary exposure, the proposed label modification will help provide the Agency with additional methods of estimating residential exposure to pesticides as is mandated by the Federal Food, Drug and Cosmetic Act (FFDCA) as amended by the Food Quality Protection Act (FQPA), August 3, 1996. Please note that the guidance in the draft PR Notice should not be viewed as a substitution for the policies required when completing residential risk assessments.

DATES: Comments, identified by docket control number OPP–00629, must be received on or before May 30, 2000.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I. of the **SUPPLEMENTARY INFORMATION.** To ensure proper receipt by EPA, it is imperative that you identify docket control number OPP–00629 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: Mark Dow or Tracy Keigwin, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Building, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone numbers: (703) 305–5533 and (703) 305–6605 respectively; fax number: (703) 305–6596; e-mail addresses: dow.mark@epa.gov; keigwin.tracy@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed to the public in general. This action may, however, be of interest to state regulatory agencies, medical personnel, pesticide registrants. Since other entities may also be interested, the Agency has not attempted to describe all the specific entities that may be affected by this action. If you have any 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'' 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. *Fax on Demand.* You may request to receive a faxed copy of the draft PR Notice titled ''Indoor Residential Insecticide Product Label Statements'' by using a faxphone to call (202) 401–0527 and selecting item 6121. You may also follow the automated menu.

3. *In person.* The Agency has established an official record for this action under docket control number OPP–00629. 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 Hwy., 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 control number OPP–00629 in the subject line on the first page of your response.

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

2. *In person.* Deliver written comments to: Public Information and Records Integrity Branch, Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

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 in this unit. 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 control number OPP–00629. 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