relative to prenatal and postnatal effects for children is complete. Further, for spinosad, the NOAELs in the dog chronic feeding study which was used to calculate the RfD (0.027 mg/kg/day) are already lower than the NOAELs from the developmental studies in rats and rabbits by a factor of more than 10–fold. Concerning the reproduction study in rats, the pup effects shown at the HDT were attributed to maternal toxicity. Therefore, it is concluded that an additional uncertainty factor (UF) is not needed and that the RfD at 0.027 mg/kg/day is appropriate for assessing risk to infants and children. In addition, EPA has determined that the 10X factor to account for enhanced sensitivity of infants and children is not needed because:

i. The data provided no indication of increased susceptibility of rats or rabbits to *in utero* and/or postnatal exposure to spinosad. In the prenatal developmental toxicity studies in rats and rabbits and 2–generation reproduction in rats, effects in the offspring were observed only at or below treatment levels that resulted in evidence of parental toxicity.

ii. No neurotoxic signs have been observed in any of the standard required studies conducted.

iii. The toxicology data base is complete and there are no data gaps.

iv. Exposure data are complete or is estimated based on data that reasonably account for potential exposure.

Using the conservative exposure assumptions previously described (tolerance level residues), the percent RfD utilized by the aggregate exposure to residues of spinosad on existing crop uses is 81.9% for children 1 to 6 years old, the most sensitive population subgroup from an EPA assessment based on the cPAD (as posted in the **Federal Register** of May 3, 2000). Additional refinements to the dietary exposure based on market share information would reduce the exposure of children 1 to 6 years old to less than 50% the cPAD. Grain treated under a temporary tolerance is expected to contribute only a negligible impact to the RfD. Thus, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to spinosad residues on the above proposed uses, including existing crop uses.

F. International Tolerances

There is no Codex maximum residue levels established for residues of spinosad.

[FR Doc. 01–30913 Filed 12–13–01; 8:45 am]
BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–1060; FRL–6813–2]

Notice of Filing Pesticide Petitions 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 pesticide petitions 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 PF–1060, must be received on or before January 14, 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 control number PF–1060 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Shaja R. Brothers, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–3194; e-mail address: brothers.shaja@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 control number PF–1060. 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–1060 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 control number PF–1060. 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 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: November 29, 2001.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

The petitioner's summaries of pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of petitions were prepared by the petitioner and represents the view of the petitioner. EPA is publishing the petitions summaries verbatim without editing them in any way. The petitions summaries 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 methods are needed.

Interregional Research Project Number 4 (IR–4) and Uniroyal Chemical Company

PP 0E6167, 1E6347, and 1F6235

EPA has received pesticide petitions (0E6167, 1E6347 and 1F6235) from the Interregional Research Project Number 4 (IR-4), 681 US Highway #1 South, North Brunswick, NJ 08902 and Uniroyal Chemical Company Inc., Middlebury, CT 06749 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 180.377 by establishing tolerances for residues of diflubenzuron, (*N*-(4-chlorophenyl)amino]carbonyl-2,6-difluorobenzamide) in or on the following raw agricultural commodities:

- PP 0E6167 proposes the establishment of a tolerance for pear at 0.5 part per million (ppm).
- PP 1E6347 proposes the establishment of a tolerance for the grass, forage, fodder, and hay group at 6.0 ppm.
- PP 1F6235 proposes the establishment of tolerances for stonefruit (except cherries) at 0.05 ppm, tree nuts and pistachios at 0.05 ppm, almond hulls at 5.0 ppm, peppers at 1.0 ppm, and meat-by-products at 0.15 ppm.

EPA has determined that the petitions contain 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 petitions. Additional data may be needed before EPA rules on the petitions. This notice of filing contains a summary of the petition provided by Uniroyal Chemical Company, Inc., the registrant.

A. Residue Chemistry

1. *Plant metabolism.* The nature of the residue in plants is adequately understood. The metabolism of diflubenzuron was investigated in soybeans, oranges, and rice. The main component of residues in rice was p-chlorophenylurea (CPU); levels of p-chloroaniline (PCA) were negligible to non-detectable. The main component of the residues in soybeans and oranges was the parent diflubenzuron (DFB). A considerable portion of the residues were bound. DFB showed very limited absorption and translocation in plants

with most of the residues remaining on the surface.

2. *Analytical method*. Practical analytical methods for detecting levels of DFB, CPU and PCA, in or on food with a limit of detection that allows monitoring of the residue at or above the level set in the tolerance was used to determine residues in the proposed commodities. Residues of the individual analytes are detectable and quantifiable using three separate analytical methods. Residues of DFB are extracted from the proposed commodities with dichloromethane. Extracts are purified with deactivated florisil. An aliquot of the extract is hydrolyzed with phosphoric acid and the DFB is partitioned into hexane. The resulting extract is derivatized in heptafluorobutyric anhydride (HFBA). Quantification of DFB is accompanied by gas chromatography using an electron capture detector.

The analytical method for quantitation of the 4-chlorophenylurea requires ethyl acetate extraction of the residue from the matrix. Column chromatography is utilized for clean-up of the extract immediately prior to derivitization with HFBA. Derivatized extracts are analyzed by gas chromatography equipped with an electron capture detector.

The analysis for the determination of PCA residues from the proposed commodities utilize an internal standard method. Samples of matrix to be analyzed are fortified with the internal standard. Residues of 12C-PCA and the internal standard are subjected to acid and base hydrolysis. The final extract is passed through florisil column for clean-up and derivatized with HFBA in hexane. An aliquot of the derivatized extract is analyzed by gas chromatography using a mass spectrometry detector in the selective ion monitoring mode. Recovery of PCA is determined by the combined peak areas for the two mass spectral ions obtained from the derivatized 12C-PCA relative to the response factor derived from the combined areas of the corresponding two mass spectral ions from the internal standard.

3. *Magnitude of residues*. Individual residue trials have been conducted with diflubenzuron on the proposed commodities. Analyses of these trials show that the maximum total residue for diflubenzuron and its conversion products PCA and CPU will be at or below the proposed tolerance levels.

B. Toxicological Profile

1. *Acute toxicity*. Studies for diflubenzuron technical indicate the acute oral toxicity in rats and mice is >4,640milligram per kilogram (mg/kg), and the acute dermal toxicity in rats is >10,000 mg/kg. The acute inhalation lethal concentration $(LC)_{50}$ in rats is >35 mg/L (6 hours). Diflubenzuron technical is not an eye or skin irritant to rabbits, and is not a dermal sensitizer in guinea pigs.

2. *Genotoxicity*. Diflubenzuron did not show any mutagenic activity in point mutation assays employing *S. typhimurium*, *S. cerevisiae*, or L5178Y Mouse Lymphoma cells. Diflubenzuron did not induce chromosomal aberrations in chinese hamster ovary (CHO) cells and it did not induce unscheduled DNA synthesis (UDS) in human WI–38 cells. Diflubenzuron was also negative in mouse micronucleus and mouse dominant lethal assays and it did not induce cell transformation in Balb/3T3 cells.

3. *Developmental and reproductive*. In a rat developmental toxicity study, diflubenzuron was administered by oral gavage to pregnant female rats at dosage levels of 0, 1, 2, and 4 mg/kg/day. No treatment-related effects were seen. A subsequent study was conducted in pregnant Sprague Dawley rats at a dose of 0 and 1,000 mg/kg/day. No maternal toxicity was observed. The incidence of fetuses with skeletal abnormalities was slightly increased in the treated group, but was within historical background range. The no observed adverse effect level (NOAEL) for maternal and developmental toxicity in rats was greater than 1,000 mg/kg/day.

Diflubenzuron was also administered by oral gavage to pregnant New Zealand white rabbits at dosage levels of 0, 1, 2, and 4 mg/kg/day. No treatment-related effects were seen. A subsequent study was conducted in pregnant rabbits at a dose of 0 and 1,000 mg/kg/day. No maternal or developmental toxicity was seen. The NOAEL for maternal and developmental toxicity in rabbits was greater than 1,000 mg/kg/day.

In a rat reproduction study, diflubenzuron was fed to 2–generations of male and female rats at dietary concentrations of 0, 10, 20, 40, and 160 ppm. No effects were seen on parental body weight gain and there were no reproductive effects. A subsequent study was conducted on one generation (1 litter) of rats at dietary concentrations of 0, 1,000, and 100,000 ppm. Systemic effects were seen in adults at these doses but there was no effect on reproductive parameters. The NOAEL for reproductive toxicity was greater than 100,000 ppm (5 g/kg/day).

4. *Subchronic toxicity.* To assess subchronic toxicity, a 4–week inhalation study and a 3–week dermal study were conducted. In the inhalation study rats were exposed nose only to 10, 30, or 100 milligram per cubic meters (mg/m^3) for 6 hours per day, 5 days per week for 4 weeks. Treatment-related findings were a slight reduction in erythrocytes, hemoglobin and hematocrit in male and female rats at a concentration of 100 mg/m^3 and an increase in total bilirubin in high dose female rats. There was no effect on methemoglobin concentration at any dose level. The NOAEL for subchronic inhalation toxicity was 30 mg/m^3.

To assess subacute dermal toxicity, diflubenzuron was applied to the backs of male and female CD rats for 3 weeks at dose levels of 20, 500, and 1,000 mg/kg/day. Hematology evaluation showed reductions in red blood cell (RBC), hemoglobin (Hgb) and hematocrit values at 500 and 1,000 mg/kg/day. An increased incidence of *polychromasia, hypochromasia,* and *anisocytosis* was seen at 500 and 1,000 mg/kg/day. An increase in methemoglobin and sulfhemoglobin values was seen at 1,000 mg/kg/day. The NOAEL for systemic toxicity was 20 mg/kg/day. Also, a dermal absorption factor of 0.5%, for systemic absorption, was derived from a study where rats were dosed with either 0.005 or 0.05 mg/cm^2 of (^{14}C) diflubenzuron technical. This value can be used for converting dermal exposure to oral equivalents.

5. *Chronic toxicity*. Diflubenzuron was given by capsule to male and female Beagle dogs for 1 year at dose levels of 0, 2, 10, 50, and 250 mg/kg/day. Body weight (bwt) gain was slightly reduced in females at 250 mg/kg/day. Absolute liver and spleen weights were increased in males given 50 and 250 mg/kg/day. A reduction in hemoglobin and mean corpuscular hemoglobin concentration, with an elevation in reticulocyte count, was seen at 50 and 250 mg/kg/day. Methemoglobin and sulfhemoglobin values were increased at doses of 10 mg/kg/day and greater. Histopathological findings were limited to pigmented macrophages and Kupffer cells in the liver at doses of 50 and 250 mg/kg/day. The NOAEL for chronic toxicity in dogs was 2 mg/kg/day.

Diflubenzuron was fed to male and female Sprague Dawley rats for 2 years at dose levels of 0, 156, 625, 2,500, and 10,000 ppm. Methemoglobin values were elevated in female rats at all dose levels and in male rats at the two highest dose levels. Sulfhemoglobin was elevated in females, only, at dose levels of 2,500 and 10,000 ppm. Mean corpuscular volume (MCV) and reticulocyte counts were increased in high dose females. Spleen and liver weights were elevated at the two highest doses. Histopathological examination

demonstrated an increase in hemosiderosis of the liver and spleen, bone marrow and erythroid hyperplasia and areas of cellular alteration in the liver. In another study diflubenzuron was administered to male and female CD rats for 2 years at dose levels of 0, 10, 20, 40, and 160 ppm. Elevated methemoglobin levels were seen in high dose males and females. No additional effects, including carcinogenic findings, were observed. The NOAEL for chronic toxicity in rats was 40 ppm (2 mg/kg/day).

A 91–week carcinogenicity study in CFLP mice was conducted at doses of 0, 16, 80, 400, 2,000, and 10,000 ppm. There was no increase in tumor incidence as a result of diflubenzuron administration. Target organ effects included: Increased methemoglobin and sulfhemoglobin values, Heinz bodies, increased liver and spleen weight, hepatocyte enlargement, and vacuolation, extramedullary hemopoiesis in the liver and spleen, siderocytosis in the spleen and pigmented Kupffer cells. A NOAEL for these effects was 16 ppm (2 mg/kg/day).

Diflubenzuron was fed to male and female Sprague Dawley rats for 2 years at dose levels of 0, 156, 625, 2,500, and 10,000 ppm. Methemoglobin values were elevated in female rats at all dose levels and in male rats at the two highest dose levels. Blood sulfhemoglobin was elevated in females, only, at dose levels of 2,500, and 10,000 ppm. MCV and reticulocyte counts were increased in high dose females. Spleen and liver weights were elevated at the two highest doses. Histopathological examination demonstrated an increase in hemosiderosis of the liver and spleen, bone marrow and erythroid hyperplasia, and areas of cellular alteration in the liver. There was no increase in tumor formation. In another study, diflubenzuron was administered to male and female CD rats for 2 years at dose levels of 0, 10, 20, 40, and 160 ppm. Elevated methemoglobin levels were seen in high dose males and females. No additional effects, including carcinogenic findings, were observed.

6. *Animal metabolism*. DFB in rats at a single dose of 100 mg/kg and 5 mg/kg single and multiple oral doses depicted limited absorption from the gastrointestinal tract. No major difference was observed between the single and multiple doses. In single dose treatments, after 7 days, 20% and 3% of the applied dose 5 and 100 mg/kg, respectively, were excreted in urine, while 79% and 98% of the applied dose 5 and 100 mg/kg, respectively, were eliminated in the feces. Very little bioaccumulation in the tissues was observed. In the feces, only unchanged parent compound was detected. Several metabolites were observed in the urine which are, among others, 2,6-diflurobenzoic acid (DFBA), 2,6-difluorophippuric acid, 2,6-difluorobenzamide (DFBAM), and 2-hydroxydiflubenzuron (2-HDFB). An unresolved peak that was characterized as *p-chloroaniline* (PCA) and/or *p-chlorophenylurea* (CPU) was found. This latter peak accounted for about 2% of the administered dose (5 mg/kg). To resolve if PCA and CPU are indeed metabolites of DFB, rats were administered a single oral dose, 100 mg/kg of 14C DFB. The major metabolites identified in rat urine were 4-chloroaniline-2-sulfate, accounting for almost 50% of the total radioactive residue (TRR) in the urine and *N*-(4-chlorophenyl)oxamic acid which accounted for about 15% of the (TRR). Neither CPU, PCA nor their *N*-hydroxyl derivatives were found in rat urine at a limit of detection of 23 parts per billion (ppb). As in the previous study, DFB was the only residue found in the feces.

7. *Metabolite toxicology*. NCI/NTP conducted chronic feeding and gavage studies with p-chloroaniline (PCA), a minor potential metabolite of diflubenzuron, in Fischer 344 rats and B6C3F1 mice.

PCA was administered in the diet to Fischer 344 rats at dietary concentrations of 250 and 500 ppm for 78 weeks, followed by a 24–week observation period. A slight body weight depression was seen in high dose females rats, compared to controls. Survival was reduced in high dose males compared to controls. In male rats there was a slight increase in uncommon fibromas or fibrosarcomas of the spleen, which was not statistically significant. Non-neoplastic proliferative and chronic inflammatory lesions were found in spleens of treated rats. It was concluded that, under the conditions of the assay, sufficient evidence was not found to establish the carcinogenicity of PCA for Fischer 344 rats.

PCA was administered 5 days/week by oral gavage, as a hydrochloride salt in water, to male and female F344/N rats at doses of 0, 2, 6, or 18 mg/kg/day. Mean body weights of dosed rats were generally within 5% of those of controls throughout the study. High dose animals generally showed mild hemolytic anemia and dose-related methemoglobinemia. Non-neoplastic lesions seen were bone marrow hyperplasia, hepatic hemosiderosis, and splenic fibrosis, suggesting treatment-related effects on the hematopoietic system. Adrenal medullary hyperplasia was observed in high dose female rats. The incidence of uncommon sarcomas of the spleen was significantly increased in high dose male rats. A marginal increase in pheochromocytomas of the adrenal gland was seen in high dose male and female rats. It was concluded that, under the conditions of this 2–year gavage study, there was clear evidence of carcinogenic activity of PCA hydrochloride for male F344/N rats and equivocal evidence of carcinogenic activity of PCA hydrochloride for female F344/N rats.

PCA was administered in the diet to B6C3F6 mice at dietary concentrations of 2,500 and 5,000 ppm for 78 weeks followed by a 13–week observation period. A body weight depression was seen in treated mice of both sexes, compared to controls. An increased incidence of hemangiomas and hemangiosarcomas in spleen, kidney, liver, and other sites was seen in treated mice of both sexes; however this increase was not statistically significant compared to controls. Non-neoplastic proliferative and chronic inflammatory lesions were found in spleens of treated mice. The evidence was considered insufficient to conclusively relate the hemangiomatous tumors in mice to compound administration. It was concluded that, under the conditions of the assay, sufficient evidence was not found to establish the carcinogenicity of PCA for B6C3F1 mice.

PCA hydrochloride was administered 5 days/week by oral gavage to male and female B6C3F1 mice at doses of 0, 3, 10, or 30 mg/kg/day. Mean body weights of high dose male and female mice were generally within 5% of those of controls throughout the study. The incidence of hepatocellular adenomas or carcinomas (combined) was increased in a non-dose-dependent manner in treated male mice. Metastasis of carcinoma to the lung was seen in the high dose group. An increased incidence of hemangiosarcomas of the liver or spleen was seen in high dose male mice. It was concluded that, under the conditions of this 2–year gavage study, there was some evidence of carcinogenic activity of PCA hydrochloride for male B6C3F1 mice and no evidence of carcinogenic activity of PCA hydrochloride for female B6C3F1 mice.

In addition to PCA, 4-chlorophenylurea (CPU) is also a potential minor metabolite of diflubenzuron. By association with PCA, EPA has concluded that CPU has carcinogenic potential and the same carcinogenic potency (q_1^*) as PCA. In the NTP report of the PCA bioassay, it is proposed that PCA undergoes *N*-hydroxylation to form the corresponding *N*-hydroxylamine

metabolites; *N*-hydroxylation of aromatic amines is a well know mechanism of aromatic amine carcinogenicity. This metabolite, or proximate carcinogen, is then conjugated to form the ultimate carcinogen capable of ionizing and reacting with DNA to form adducts which result in splenic tumor formation. An alternate mechanism involving toxicity resulting in erythrocyte damage, splenic scavenging, hemorrhage, hyperplasia and fibrosis and ultimately splenic tumor formation is also proposed, but both mechanisms are based on the formation of *N*-hydroxy PCA.

This metabolite also causes methemoglobinemia in animals. Therefore, methemoglobin formation can be used as an indicator of the presence of PCA and *N*-hydroxy metabolite. However, in recent CPU rat toxicity studies, both dietary (7–day) and gavage, and a CPU rat metabolism study, it has been demonstrated that CPU does not induce methemoglobin formation and it is neither metabolized to PCA nor forms an *N*-hydroxylamine derivative. Since *N*-hydroxylation is the required first step in the mechanism of action of PCA's carcinogenicity, it can be concluded that CPU's mechanism of action and toxicity is different from that of PCA's.

8. *Endocrine disruption*. The standard battery of required studies has been completed and evaluated to determine potential estrogenic or endocrine effects of diflubenzuron. These studies include an evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure. These studies are generally considered to be sufficient to detect any endocrine effects. No such effects were noted in any of the studies with diflubenzuron.

C. Aggregate Exposure

1. *Dietary exposure*. Since 1–day single dose oral studies in rats and mice indicated only marginal effects, an acute exposure risk assessment is not needed, as there were no significant acute effects observed.

i. *Food*—a. *Diflubenzuron*. The chronic dietary exposure from diflubenzuron was estimated based on the average residue values from the various currently labeled raw agricultural commodities (RACs) and the proposed pear use. Percent of crop treated was also factored into the estimate. Residues in meat, milk, and egg products were obtained from extrapolation of metabolism study data to anticipated livestock dietary burdens. The dietary exposure analysis was estimated based on 1989–1992 USDA food consumption data.

For the U.S. population (total), the dietary exposure of diflubenzuron was estimated as 0.000027 mg/kg/day. For nursing and non-nursing infants, the exposure was estimated as and 0.000110 and 0.000304 mg/kg/day, respectively. For children, the exposure was 0.000046 and 0.000033 mg/kg/day for 1–6 year olds and 7–12 year olds, respectively.

b. *p-Chloroaniline*. The chronic dietary exposure from p-chloroaniline (PCA) which has been detected in some food products was also determined. Average residues from field trials for mushrooms, rice, pears, nut crops, and pistachios, stonefruit (except cherries), and peppers were used. Residues in liver were obtained from extrapolation of metabolism data to anticipated livestock dietary burdens. EPA has previously used a 2% *in vivo* conversion factor of DFB to PCA for foods derived from plant products. However, based on results of a recent rat metabolism study showing that no PCA is formed, this is no longer appropriate. The percent treated of each crop was also factored into the exposure estimate.

For the U.S. population (total), the dietary exposure of PCA was estimated as <0.000001 mg/kg/day. For nursing and non-nursing infants, the exposure was estimated as 0.000002 and 0.000007 mg/kg/day, respectively. For children 1 to 6 years old and 7 to 12 years old, the exposure was 0.000001 mg/kg/day.

ii. *Drinking water*. Diflubenzuron degrades in soil relatively quickly with an aerobic half-life ranging from 3 to 7 days. Major degradates include difluorobenzoic acid (DFBA) and CPU. DFBA is further metabolized through decarboxylation and ring cleavage by soil microbes whereas CPU is slowly degraded to soil-bound entities. Under anaerobic aquatic conditions, diflubenzuron has a half-life of 34 days with the main degradates being DFBA and CPU. In surface water, diflubenzuron is degraded by microbes with a half-life of 5 to 10 days. The soil mobility of diflubenzuron is considered quite limited based on a number of experimental studies as well as by computer modeling. CPU has also been shown to be relatively immobile in soil. Although DFBA shows mobility in soil, it is rapidly degraded. Therefore, based on results of laboratory and field studies, it is not likely that diflubenzuron or its degradates will impact ground water quality to any significant extent.

Based on EPA's PRZM/EXAMS modeling, the average annual mean concentration of diflubenzuron in surface water sources is not expected to exceed 0.05 ppb. These values were determined using the maximum concentrations for any diflubenzuron crop uses including the proposed commodities. The drinking water level of concern (DWLOC) for chronic (non-cancer) exposure to diflubenzuron in drinking water was determined as 700 ppb for the U.S. population (total) and approximately 200 ppb for infants and children. The estimated maximum concentration of diflubenzuron in surface and ground water (0.05 ppb) is much less than the DWLOCs as a contribution to chronic (non-cancer) aggregate exposure.

2. *Non-dietary exposure*. Diflubenzuron is a restricted use pesticide based on its toxicity to aquatic invertebrates. This restricted use classification makes it unavailable for use by homeowners. Occupational uses of diflubenzuron may expose people in residential locations, parks, or forests treated with diflubenzuron. However, diflubenzuron has very low residues detected in forestry dissipation studies, low dermal absorption rate (0.05%), and extremely low dermal and inhalation toxicity.

D. Cumulative Effects

Uniroyal Chemical Co. has considered the potential for cumulative effects of diflubenzuron and other substances with a common mechanism of toxicity. The mammalian toxicity of diflubenzuron is well defined. We are not aware of any other pesticide product registered in the United States that could be metabolized to p-chloroaniline. For this reason, consideration of potential cumulative effects of residues from pesticidal substances with a common mechanism of action as diflubenzuron is not appropriate. Thus only the potential exposures to diflubenzuron were considered in the total exposure assessment.

E. Safety Determination

1. *U.S. population*. Based on the available toxicology and exposure data base for diflubenzuron, Uniroyal has determined that the total possible non-occupational aggregate exposure from diflubenzuron would occur from the dietary route. Dietary exposure to the U.S. population (total) from diflubenzuron was estimated at 0.000027 mg/kg/day. Based on the 0.02 mg/kg/day RfD (reference dose) derived from the dog chronic NOAEL of 2 mg/kg/day and a 100–fold safety factor, this dietary exposure is 0.1% of the RfD. Despite the potential for exposure to diflubenzuron in drinking water,

aggregate exposure is not expected to exceed 100% of the RfD.

For PCA, Uniroyal has also determined that the total possible non-occupational aggregate exposure would occur from the dietary route. Dietary exposure to the U.S. population (total) from PCA was estimated as less than 0.000001 mg/kg/day. The risk from diflubenzuron-derived PCA can be estimated using a linear extrapolation of the dose-response from the rat chronic study conducted by the National Toxicology Program in which rats were dosed via gavage with p-chloroaniline (hydrochloride) for 24 months. EPA has determined the q^{1*} as 0.0638 based on the combined sarcoma incidence in the spleen of male rats.

In view of the results of recent CPU rat mechanistic and metabolism studies, and the DFB rat metabolism study, the dietary risk assessment included here considers only actual residues of PCA found in food and animal by-products. This is consistent with a parent compound, such as diflubenzuron, which is negative (category E) for carcinogenicity.

Using the q^{1*} of 0.0638, the risk to the U.S. population (total) from dietary exposure to diflubenzuron-derived PCA is 3.09×10^{-8}.

2. *Infants and children*. The same assumptions as for the U.S. population were used for the dietary exposure risk determination in infants and children. The dietary exposure of diflubenzuron was calculated as 0.000110 and 0.000304 mg/kg/day, respectively for nursing and non-nursing infants. These values are 0.6% and 1.5%, respectively of the RfD for diflubenzuron. The dietary exposure from diflubenzuron in children 1 to 6 years and 7 to 12 years old was determined as 0.000046 mg/kg/day and 0.000033 mg/kg/day, respectively. These values are 0.2% of the RfD.

As previously discussed, the NOAELs for maternal and developmental toxicity in rats and rabbits were greater than 1,000 mg/kg/day, and the NOAEL for reproductive toxicity was greater than 5,000 mg/kg/day. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Uniroyal concludes that there is reasonable certainty that no harm will result in infants and children from aggregate exposure to residues of diflubenzuron and its conversion products containing the p-chloroaniline moiety.

F. International Tolerances

There is a Codex maximum residue limit (MRL) for pears at 1.0 mg/kg, a Mexican MRL at 1.0 mg/kg, and no limits set for Canada for pears. A Codex MRL has also been established for plums (including prunes) at 1.0 mg/kg. There are no Codex maximum residue limits established for other stonefruit, tree nuts or peppers.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–1057; FRL–6812–4]

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 PF–1057, must be received on or before January 14, 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 control number PF–1057 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Dani Daniel, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–5409; e-mail address: daniel.dani@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	Crop production Animal production Food manufacturing
	32532	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–1057. 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.