infants and children to residues of imidacloprid, the data from developmental studies in both rat and rabbit and a 2-generation reproduction study in the rat have been considered. The developmental toxicity studies evaluate potential adverse effects on the developing animal resulting from pesticide exposure of the mother during prenatal development. The reproduction study evaluates effects from exposure to the pesticide on the reproductive capability of mating animals through two generations, as well as any observed systemic toxicity.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post- natal effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for imidacloprid relative to pre- and post- natal effects is complete. Further for imidacloprid, the NOEL of 5.7 mg/kg/bwt from the 2–year rat feeding/ carcinogenic study, which was used to calculate the RfD (discussed above), is already lower than the NOELs from the developmental studies in rats and rabbits by a factor of 4.2 to 17.5 times. Since a 100–fold uncertainty factor is already used to calculate the RfD, it is surmised that an additional uncertainty factor is not warranted and that the RfD at 0.057 mg/kg/bwt/day is appropriate for assessing aggregate risk to infants and children.

Using the conservative exposure assumptions described above under aggregate exposure, Bayer has determined from a chronic dietary analysis that the percent of the RfD utilized by aggregate exposure to residues of imidacloprid ranges from 9.3% for nursing infants up to 32.2% for children (1–6 years). EPA generally has no concern for exposure below 100 percent of the RfD. In addition, the MOEs for all infant and children population groups do not exceed EPA's level of concern for acute dietary exposure. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Bayer concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the residues of imidacloprid, including all anticipated dietary exposure and all other non-occupational exposures.

F. International Tolerances

No Codex Maximum Residue Levels (MRLs) have been established for residues of imidacloprid on any crops at this time. (PM 05)

[FR Doc. 98–4803 Filed 2-24-98; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–795; FRL–5775–3]

Notice of Filing of Pesticide Petitions

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 the docket control number PF–795, must be received on or before March 27, 1998.
ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 119 at the Virginia address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Paul Schroeder, Registration Division, (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M. St., SW., Washington, DC 20460. Office location and telephone number: Rm. 255, CM #2, 1921 Jefferson Davis Highway, Arlington, VA, 703–305–6602, e-mail: schroeder.paul@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: 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.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–795] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:
opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [PF–795] and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

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

Dated: February 18, 1998.

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. EPA is publishing the petition summaries verbatim without editing them 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.

Uniroyal Chemical Company

PP 6G4771

EPA has received a pesticide petition (PP 6G4771) from Uniroyal Chemical Co., Inc., Bethany, Connecticut proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the combined residues of the insect growth regulator, diflubenzuron and metabolites convertible to p-chloroaniline, expressed as diflubenzuron in or on rice at 0.02 parts per million (ppm) and rice straw at 0.8 ppm. The proposed analytical method for detecting and measuring residues of diflubenzuron and 4-chloroaniline is gas chromatography with electron capture detection. p-Chloroaniline is determined using an internal standard method and detected by mass spectrometry.

Pursuant to the section 408(d)(2)(A)(I) of the FFDCA, as amended, Uniroyal Chemical Company has submitted the following summary of information, data and arguments in support of their pesticide petition. This summary was prepared by Uniroyal Chemical and EPA has not fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments were the petitioners and not necessarily EPA's and to remove certain extraneous material.

A. Toxicology Profile

1. *Data summary.* Diflubenzuron is not acutely toxic and is not an irritant. In a 3-week dermal toxicity study in rats the no observed effect level (NOEL) for systemic toxicity was 20 milligrams/kilograms (mg/kg/day). In developmental toxicity studies in rats and rabbits, diflubenzuron was without maternal or developmental effects at doses up to 1,000 mg/kg/day. Systemic effects were seen on parental animals in a rat reproduction study at doses of 1,000 and 100,000 ppm; however, there were no reproductive effects and the NOEL for reproductive toxicity was greater than 5,000 mg/kg/day. In a chronic dog feeding study, target organ effects were seen in the blood and liver. Methemoglobinemia was evident at dose levels of 10 mg/kg/day and greater. The NOEL for chronic toxicity in dogs was 2 mg/kg/day. In a chronic rat feeding study, target organ effects were seen in the blood, liver, spleen and bone marrow. Methemoglobinemia was evident at doses of 160 ppm and greater. The NOEL for chronic toxicity in rats was 2 mg/kg/day. Diflubenzuron was negative in a complete battery of mutagenicity assays. In a mouse oncogenicity study, diflubenzuron was negative at doses up to 1,429 mg/kg/day. Additionally, diflubenzuron was negative for carcinogenicity in a rat chronic feeding study at doses up to 500 mg/kg/day. None of the studies conducted on diflubenzuron have provided evidence of endocrine organ involvement.

2. *Acute toxicity.* Studies for diflubenzuron technical indicate the acute oral toxicity in rats and mice is >4,640 mg/kg, and the acute dermal toxicity in rats is >10,000 mg/kg. The acute inhalation 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.

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 NOEL for systemic toxicity was 20 mg/kg/day.

3. *Developmental/reproductive effects.* 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 NOEL 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 NOEL for maternal and developmental toxicity in rabbits was greater than 1,000 mg/kg/day.

In a rat reproduction study, diflubenzuron was fed to two 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 1-generation (one 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 NOEL for reproductive toxicity was greater than 100,000 ppm (5 g/kg/day).

4. *Chronic effects.* Diflubenzuron was given by capsule to male and female Beagle dogs for one year at dose levels of 0, 2, 10, 50 and 250 mg/kg/day. Body weight 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 NOEL 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 NOEL for chronic toxicity in rats was 40 ppm (2 mg/kg/day).

5. *Carcinogenicity*. A 91-week oncogenicity 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 NOEL 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.

NCI/NTP conducted chronic feeding and gavage studies with p-chloroaniline (PCA), a minor 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 methemolglobinemia. 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.

6. *Mutagenicity*. 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 cells and it did not induce unscheduled DNA synthesis 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.

7. *Endocrine effects*. 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.

8. *Rat metabolism*. Diflubenzuron (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. 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 p-chloroaniline (PCA) and/or p-chlorophenylurea (CPU) was found. This latter peak accounted for about 2% of the administered dose (5 mg/kg). In the feces, only unchanged parent compound was detected.

B. Aggregate Exposure

1. *Dietary exposure*—i. *Diflubenzuron*. The dietary exposure from diflubenzuron (DFB) was estimated based on the average residue values from the various currently labeled raw agricultural commodities (RACs) and the proposed rice use. Percent of crop treated was also factored into the estimate. Current animal commodity tolerances, which

adequately cover the rice use, were used for meat, milk, and egg products. The dietary exposure analysis was estimated based on 1977 USDA food consumption data.

For the general U.S. population (48 states, all seasons), the dietary exposure of diflubenzuron was estimated as 0.000706 mg/kg/day. For nursing and non-nursing infants, the exposure was estimated as and 0.000799 and 0.003461 mg/kg/day, respectively. For children, the exposure was 0.001888 and 0.001178 mg/kg/day for 1-6 year olds and 7-12 year olds, respectively.

ii. *p-Chloroaniline and related product.* The dietary exposure estimate for PCA and related products is a conservative estimate, in that it includes rice straw as an animal feed. Rice straw, however, will be restricted as a animal feed, in the proposed Experimental Use Program. The dietary exposure from p-chloroaniline (PCA) and a related product, 4-chlorophenylurea (CPU), which have been detected in some food products was also determined. EPA has used a 2% *in vivo* conversion factor of DFB to PCA for foods derived from plant products. For mushrooms, PCA and CPU average residue data was combined with a 2% *in vivo* conversion of DFB to PCA. Calculations for levels of PCA/CPU in animal products were based on metabolism studies, extrapolation to anticipated animal dietary burdens and the 2% conversion of DFB to PCA. The percent treated of each crop was also factored into the exposure estimate.

For the general U.S. population, the dietary exposure of PCA/CPU was estimated as 0.000001 mg/kg/day. For nursing and non-nursing infants, the exposure was estimated as 0.000002 and 0.000006 mg/kg/day, respectively. For children, the exposure was 0.000004 and 0.000002 mg/kg/day for 1-6 year olds and 7-12 year olds, respectively.

2. *Drinking water exposure.* Diflubenzuron degrades in soil relatively quickly with an aerobic half-life ranging from 3-7 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 5-10 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. Thus the aggregate risk to diflubenzuron does not include drinking water.

3. *Non-occupational 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. Based on very low residues detected in forestry dissipation studies, low dermal absorption rate (0.05%), and extremely low dermal and inhalation toxicity, these uses are expected to result in insignificant risk, and will, therefore, not be included in the aggregate risk assessment. Reference: ''Reregistration Eligibility Document: Diflubenzuron,'' EPA, August 1997.

C. Cumulative Risk

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.

D. 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 exposure route. Dietary exposure to the general U.S. population from diflubenzuron was estimated at 0.000706 mg/kg/day. Based on the 0.02 mg/kg/day RfD (reference dose) derived from the dog chronic NOEL of 2 mg/kg/day and a 100-fold safety factor, this dietary exposure is 3.5% of the RfD.

For PCA and CPU, Uniroyal has also determined that the total possible non-occupational aggregate exposure would occur from the dietary exposure route. Dietary exposure to the general U.S. population from PCA/CPU was estimated as 0.000001 mg/kg/day. The risk from diflubenzuron-derived PCA/CPU 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 5 days/week for 103 weeks (NTP TR 351). EPA has determined the q1* as 0.059 by combining the incidences of splenic sarcomas from both male and female rats.

Although EPA has assumed that CPU is also carcinogenic purportedly based on its structural similarity to PCA, Uniroyal has indicated to the Agency in previous correspondence that this assumption is not warranted. It may be more appropriate to compare the carcinogenicity potential of CPU to acetanilide, which is also a structural analog of CPU, and for which no evidence of carcinogenicity has been demonstrated possibly because the *N*-hydroxy metabolite is not formed in significant amounts. Formation of the *N*-hydroxy metabolite of CPU is also remote. Uniroyal has also argued that it is unlikely that significant degradation of CPU to form PCA would occur, since based on the known animal metabolism of phenylureas, only a small amount of aniline derivatives are produced. The major metabolic pathway for the phenylureas is ring hydroxylation and n-dealkylation, a process that would maintain the integrity of the parent urea molecule. Therefore, it would not be appropriate to combine CPU residues with PCA. However, for this safety assessment we have conservatively estimated the risk from dietary exposure to both PCA and CPU combined.

Using the q1* of 0.059 from the combined male and female incidence of splenic tumors in rats, the risk to the general U.S. population from dietary exposure to diflubenzuron-derived PCA/CPU is 8.7×10^{-8}.

2. *Infants and children.* The same assumptions as for the general U.S. population were used for the dietary exposure risk determination in infants and children. The dietary exposure of diflubenzuron was calculated as 0.000799 mg/kg/day and 0.003461 mg/kg/day respectively for nursing and non-nursing infants. These values are 4% and 17.3% respectively of the RfD for diflubenzuron. The dietary exposure from diflubenzuron in children 1-6 and 7-12 years old was determined as 0.001888 mg/kg/day and 0.001178 mg/kg/day, respectively. These values are 9.4% and 5.9% of the RfD, respectively.

As previously discussed, the NOELs for maternal and developmental toxicity in rats and rabbits were greater than

1,000 mg/kg/day, and the NOEL 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.

E. Residues in the Raw Agricultural Commodity and Processed Food/Feed

1. *Nature of residues in plants and livestock.* The nature of the residue in plants and livestock is adequately understood. In plants, the metabolism of diflubenzuron was investigated in soybeans, oranges and rice. The main component of residues in rice was CPU; levels of PCA were negligible to non-detectible. 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.

In livestock, goats treated for three days at about 1X (10 ppm feeding level) the dietary burden of ^{C}14 DFB gave DFB equivalent of ^{C}14 = 7-9 ppb in milk, 217-262 ppb in liver, 16-19 ppb in kidney, about 1 ppb in muscle, and about 4 ppb in fat. Milk residues were mainly CPU and DFBAM. PCA was not detectable. Liver residues were DFB, 2-hydroxy DFB, CPU, and DFBAM. Again, PCA was not detected at this dose however, it was detected in studies conducted at about 22X dose. Chickens were dosed with ^{C}14 DFB at 5 ppm level for 1-28 days. Residues in tissues as DFB equivalent were highest in liver and kidney. The main residues in tissues and eggs were DFB and DFBA. Trace amount of PCA and its acetanilide were detected, but not confirmed, in liver kidney and egg white.

2. *Magnitude of residues and proposed tolerances.* An adequate number of separate residue trials have been conducted with diflubenzuron on rice. Analyses of these trials show that the maximum total residue for diflubenzuron and its conversion products PCA and CPU will be at or below 0.01 ppm.

A tolerance has been requested for the combined residues of diflubenzuron and metabolites convertible to p-chloroaniline expressed as diflubenzuron on rice at 0.01 ppm. The proposed tolerance is adequate to cover residues likely to be present from the use of diflubenzuron on rice. Therefore, no special processing to reduce the residues will be necessary.

The meat by-products tolerances are adequate to cover residues resulting from the rice use. Uniroyal Chemical has submitted calculations from a goat metabolism study which supports the 0.05 ppm tolerance in meat by-products. Therefore, no increase in the meat by-products tolerances should be necessary.

F. Practical 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 rice and its respective processed fractions.

Residues of the individual analytes are detectable and quantifiable using three separate analytical methods. Residues of DFB are extracted from rice 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 in rice matrices utilizes 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.

G. List of All Pending Tolerances and Exemptions

A tolerance for diflubenzuron on range grass at 4.0 ppm is pending. There are no exemptions from tolerance for diflubenzuron.

H. List International Tolerances (Code MRLs)

There are no Codex Alimentarius Commission maximum residue levels for residues of diflubenzuron on rice. The Codex MRL on citrus is 1.0 mg/kg vs. 0.05 ppm for U.S. tolerance. The Codex MRL for mushrooms is 0.1 mg/kg vs. 0.2 ppm for U.S. tolerance. The Codex MRL for soybeans is 0.1 mg/kg/ vs. 0.05 ppm for the U.S. The Codex MRL is 1 mg/kg for apples, Brussels sprouts, cabbage, pears, plums and tomatoes for which there are no U.S. tolerances. The Codex MRL for meat, milk and eggs is 0.05 mg/kg/ which is the same as the established U.S. tolerances.

[FR Doc. 98–4812 Filed 2-24-98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–789; FRL–5767–5]

Notice of Filing of Pesticide Petition

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 the docket control number PF–789, must be received on or before March 27, 1998.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket @epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as