TABLE 2—REGISTRANTS REQUESTING VOLUNTARY CANCELLATION—Continued

| EPA Company No. | Company Name and Address |
|---|---|
| 033688 | Registration & Regulatory Services, Agent For: CFPI Agro, S.A., 7474 Creedmoor Rd., Suite 239, Raleigh, NC 27613. |
| 041878 | LJB Laboratories, 1001 E Cass, St. Johns, MI 48879. |
| 045639 | Agrevo USA Co., Little Falls Centre One, 2711 Centerville Rd., Wilmington, DE 19808. |
| 046515 | Celex, Division of United Industries Corp., Box 15842, St Louis, MO 63114. |
| 046813 | CCL Custom Mfg. Inc., 1 W. Hegeler Ln, Danville, IL 61832. |
| 051036 | Micro-Flo Co, Box 772099, Memphis, TN 38117. |
| 057476 | Chempura Pools, Ltd., 586 Benjamin's Way, Box 56, Lewisville, TX 75067. |
| 064240 | Combat Insect Control Systems, c/o PS&RC, Box 493, Pleasanton, CA 94566. |
| 064248 | Maxforce Insect Control Systems, c/o PS&RC, Box 493, Pleasanton, CA 94566. |
| 065247 | Forbio America, 2603 E. Ower Terrace, Boise, ID 83706. |

### III. Procedures for Withdrawal of Request

Registrants who choose to withdraw a request for cancellation must submit such withdrawal in writing to James A. Hollins, at the address given above, postmarked before January 10, 2000. This written withdrawal of the request for cancellation will apply only to the applicable 6(f)(1) request listed in this notice. If the product(s) have been subject to a previous cancellation action, the effective date of cancellation and all other provisions of any earlier cancellation action are controlling. The withdrawal request must also include a commitment to pay any reregistration fees due, and to fulfill any applicable unsatisfied data requirements.

### IV. Provisions for Disposition of Existing Stocks

The effective date of cancellation will be the date of the cancellation order. The orders effecting these requested cancellations will generally permit a registrant to sell or distribute existing stocks for 1 year after the date the cancellation request was received. This policy is in accordance with the Agency's statement of policy as prescribed in **Federal Register** (56 FR 29362) June 26, 1991; [FRL 3846–4]. Exceptions to this general rule will be made if a product poses a risk concern, or is in noncompliance with reregistration requirements, or is subject to a data call-in. In all cases, product-specific disposition dates will be given in the cancellation orders.

Existing stocks are those stocks of registered pesticide products which are currently in the United States and which have been packaged, labeled, and released for shipment prior to the effective date of the cancellation action. Unless the provisions of an earlier order apply, existing stocks already in the hands of dealers or users can be distributed, sold or used legally until they are exhausted, provided that such further sale and use comply with the EPA-approved label and labeling of the affected product(s). Exceptions to these general rules will be made in specific cases when more stringent restrictions on sale, distribution, or use of the products or their ingredients have already been imposed, as in Special Review actions, or where the Agency has identified significant potential risk concerns associated with a particular chemical.

### List of Subjects

Environmental protection, Pesticides and pests, Product registrations.

Dated: June 14, 1999.

**Richard D. Schmitt,**

*Acting Director, Information Resources and Services Division, Office of Pesticide Programs.*

[FR Doc. 99–17775 Filed 7–13–99; 8:45 am]

**BILLING CODE 6560–50–F**

## ENVIRONMENTAL PROTECTION AGENCY

**[PF–877; FRL–6085–5]**

### 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–877], must be received on or before August 13, 1999.

**ADDRESSES:** By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information (CBI) 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 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. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

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

| Product Manager | Office location/telephone number | Address |
|---|---|---|
| Eugene Wilson ..... | Rm. 235, CM #2, 703–305–6103, e-mail:wilson.eugene@epa.gov. | 1921 Jefferson Davis Hwy, Arlington, VA |
| James A. Tompkins ..... | Rm. 239, CM #2, 703–305–5697, e-mail: tompkins.james@epa.gov. | |
| Bipin Gandhi ..... | Rm. 713J, CM #2 703–308–8380, e-mail:gandhi.bipin@epa.gov. | Do. |

**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–877] (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 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF-877]) 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: June 25,1999.**

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.

**1. AgrEvo USA Company**

*PP 8F3607 and 5F4578*

EPA has received pesticide petitions (PP 8F3607 and 5F4578) from AgrEvo USA Company, Little Falls Centre One, 2711 Centerville Road, Wilmington, DE 19808, 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 180.473(a)(1) and (b)(1) by establishing permanent tolerances for residues of the herbicide, glufosinate-ammonium: butanoic acid-amino-4-(hydroxymethylphosphinyl)-, monoammonium salt and its metabolite, 3-methylphosphinicopropionic acid expressed as 2-amino-4-(hydroxymethylphosphinyl) butanoic acid equivalents in or on the following raw agricultural commodities (RAC): almond hulls at 0.50 parts per million (ppm), apples at 0.05 ppm, bananas at 0.3 ppm (not more than 0.2 ppm shall be present in the pulp after the peel is removed), cattle, fat at 0.05 ppm, cattle, meat at 0.05 ppm, cattle, meat-by-products at 0.10 ppm, eggs at 0.05 ppm, goats, fat at 0.05 ppm, goats, meat at 0.05 ppm, goats, meat-by-products at 0.10 ppm, grapes at 0.05 ppm, hogs, fat at 0.05 ppm, hogs, meat at 0.05 ppm, hogs, meat-by-products at 0.10 ppm, horses, fat at 0.05 ppm, horses, meat at 0.05 ppm, horses, meat-by-products at 0.10 ppm, milk at 0.02 ppm, poultry, fat at 0.05 ppm, poultry, meat-by-products at 0.10 ppm, poultry, meat at 0.05 ppm, sheep, fat at 0.05 ppm, sheep, meat at 0.05 ppm, sheep, meat-by-products at 0.10 ppm, and the tree nuts group at 0.10 ppm.

AgrEvo has also proposed to amend 40 CFR 180.473(c) by establishing permanent tolerances for residues of the herbicide, glufosinate-ammonium: butanoic acid, 2-amino-4-(hydroxymethylphosphinyl)-, monoammonium salt and its metabolites, 3-methylphosphinicopropionic acid, and 2-acetamido-4-methylphosphinicobutanoic acid expressed as 2-amino-4-(hydroxymethylphosphinyl) butanoic acid equivalents in or on the following raw agricultural commodities: aspirated grain fractions at 25.0 ppm, corn, field, forage at 4.0 ppm, corn, field, grain at 0.2 ppm, corn, field, stover at 6.0 ppm, soybean, hulls at 5.0 ppm, and soybeans at 2.0 ppm. The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by gas chromatography.

The preceding tolerances for glufosinate-ammonium and its metabolites have already been established for the aforementioned commodities on a time-limited basis in 40 CFR 180.473 (a)(1), (b)(1) and (c). These time-limited tolerances expire on July 13, 1999. AgrEvo has proposed to re-establish these tolerances on the same crop commodities and at the same levels on a permanent basis.

A notice of filing and petitioner summary of the pesticide petition for EPA Pesticide Petitions 7F4910 and 7E4911 was published in the **Federal Register** of October 8, 1997 (62 FR 52544) (FRL–5746–9). These petitions pertain to additional tolerances for residues of glufosinate-ammonium and its metabolites on sugar beets, canola and potatoes. Data and assessments pertaining to residue chemistry, toxicological profile, endocrine effects, aggregate exposure, cumulative effects, safety determinations and international tolerances for both the existing and the proposed additional crop tolerances are provided in this publication. The petitioner's risk assessment presentation represents the maximum exposure scenario as it assesses the summative exposure from the existing time-limited

tolerances delineated above in addition to the tolerances proposed in the aforementioned petitions.

**2. E. I. DuPont de Nemours and Co., Inc.**

*4F4391*

EPA has received a pesticide petition (4F4391) from DuPont, P.O. Box 80038, Wilmington, DE 19880-0038 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 extending time-limited tolerance for residues of pyrithiobac sodium salt (sodium 2-chloro-6-[(4,6-dimethoxypyrimidin-2-yl)thio]benzoate) in or on the raw agricultural commodity (RAC) cottonseed at 0.02 ppm until September 30, 2001. In the **Federal Register** of October 25, 1995 (60 FR 54607) (FRL 4982-8), EPA established a time-limited tolerance pursuant to the FFDCA for residues of the herbicide pyrithiobac sodium salt in or on the RAC cottonseed at 0.02 ppm. In the **Federal Register** of October 22, 1997 (62 FR 54778) (FRL 5746-6), EPA extended the time-limited tolerance pursuant to the FFDCA for residues of the herbicide pyrithiobac sodium salt in or on the RAC cottonseed at 0.02 ppm. This time-limited tolerance expires September 30, 1999. The tolerance was issued and renewed as a time-limited tolerance because EPA required additional residue data on the commodity of cotton gin byproducts. 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 qualitative nature of the residues of pyrithiobac sodium in cotton is adequately understood. Metabolism studies with pyrithiobac sodium indicate the major metabolic pathway being o-dealkylation of the parent compound resulting in o-desmethyl pyrithiobac sodium (O-DPS), both free and conjugated, was the major metabolite identified in cotton foliage. The results of a confined crop rotation study with pyrithiobac sodium revealed the presence of a metabolite 2-chloro-6-sulfobenzoic acid (CSBA) not seen in the cotton metabolism study. This metabolite appeared to originate from soil metabolism of pyrithiobac sodium. Since preemergence applications of pyrithiobac sodium are allowed, crop residues of CSBA were considered a possibility. In consideration of PP 4F4391, Chemistry Branch Tolerance Support (CBTS), EPA, in consultation with the Health Effects Division (HED) Metabolism Committee has previously concluded that for the proposed use on cotton, none of the pyrithiobac sodium metabolites including O-DPS and CSBA warrant inclusion in the tolerance regulation, and that the only residue of concern is the parent, pyrithiobac sodium.

2. *Analytical method*. There is a adequately validated practical analytical method available using HPLC-UV with column switching, to measure levels of pyrithiobac sodium in or on cotton with a limit of quantitation (LOQ) that allows monitoring of cottonseed at or above tolerance levels. EPA has provided information on this method to FDA for future publication in PAM II.

3. *Magnitude of residues*. Crop field trial residue data from a 60– day pre-harvest interval (PHI) study shows that the established pyrithiobac sodium time-limited tolerance on cottonseed of 0.02 ppm will not be exceeded when Staple Herbicide is used as directed. An adequate cottonseed processing study shows that pyrithiobac sodium does not concentrate in cottonseed processed commodities; thus, no tolerances on these commodities are required.

*B. Toxicological Profile*

1. *Acute toxicity*. Pyrithiobac sodium technical has been placed in EPA Toxicity Category II for acute eye irritation based on the test article inducing irritation in the form of corneal opacity, iritis and conjunctival redness, and discharge in the eyes of rabbits after receiving ocular doses of 36 mg (0.1 ml). Signs of irritation were clear within 14–days of treatment. Pyrithiobac sodium has been placed in Toxicity Category III for acute dermal toxicity based on the test article being nonlethal and nonirritating at the limit dose (LTD) of 2,000 milligrams/kilograms (mg/kg) highest dose tested (HDT). Pyrithiobac sodium has been placed in Toxicity Category III for acute oral toxicity based on acute oral $LD_{50}$ of 3,200 mg/kg for both male and female (M/F) rats. Pyrithiobac sodium has been placed in Category IV for the remaining acute toxicity tests based on the following: a rat acute inhalation study with an $LC_{50}$ of > 6.9 milligrams/per liter (mg/l); and a primary dermal irritation test that did not induce a dermal irritation response. A dermal sensitization test with pyrithiobac sodium technical in guinea pigs demonstrated no significant effects. Based on these results, pyrithiobac sodium does not pose an acute dietary or exposure risk.

2. *Genotoxicity*. Pyrithiobac sodium technical was negative (non-mutagenic and non-genotoxic) in the following tests: Ames microbial mutation assay; the hypoxanthine-guanine phosphoribosyl transferase gene mutation assay using Chinese hamster ovary cells; and induction of unscheduled DNA synthesis (UDS) in primary rat hepatocytes. Pyrithiobac sodium was positive in an *in vitro* assay for chromosome aberrations in human lymphocytes. It was negative for the induction of micronuclei in the bone marrow cells of M/F CD-1 mice administered the test article by oral gavage at 500, 1,000 or 2,000 mg/kg. Based on the weight of these data, pyrithiobac sodium is neither genotoxic nor mutagenic.

3. *Reproductive and developmental toxicity*. A 2-generation, 4 litter reproduction study with CD rats treated at dietary levels of 0, 25, 1,500, 7,500 or 20,000 ppm of pyrithiobac sodium demonstrated a maternal no observed adverse effect level (NOAEL) of 1,500 ppm (103 mg/kg/day) and a maternal lowest observed adverse effect level (LOAEL) of 7,500 ppm (508 mg/kg/day), based on decreased body weight (bwt) gain and food efficacy. An offspring NOAEL of 7,500 ppm (508 mg/kg/day) and LOAEL of 20,000 ppm (1,551 mg/kg/day) were also demonstrated based on decreased offspring bwt. Pyrithiobac sodium was not teratogenic when administered to rats or rabbits.

A developmental toxicity study with pyrithiobac sodium in rats demonstrated a maternal NOAEL of 200 mg/kg and LOAEL of 600 mg/kg due to increased incidence of salivation.

A developmental NOAEL of 600 mg/kg and LOAEL of 1,800 mg/kg were demonstrated based on an increased incidence of skeletal variations.

A developmental toxicity study with pyrithiobac sodium in rabbits demonstrated maternal and developmental NOAELs of 300 mg/kg and a maternal LOAEL of 1,000 mg/kg based on mortality, decreased bwt gain and feed consumption, increased incidence of clinical signs, and an increase in early resorptions. A developmental LOAEL of 1,000 mg/kg was based on decreased fetal bwt gain. Based on the weight of these data, pyrithiobac sodium is not considered a reproductive or developmental hazard.

4. *Subchronic toxicity*. In a 90–day feeding study in rats conducted with pyrithiobac sodium at dietary levels of 0, 10, 50, 500, 7,000 and 20,000 ppm, the NOAEL was 500 ppm (31.8 and 40.5 mg/kg/day, Ms and Fs and the LOAEL

was 7,000 ppm (466 and 588 mg/kg/day, Ms/Fs) based on decreased bwt gains and increased rate of hepatic B-oxidation in Ms.

In a 90–day feeding study in mice conducted with pyrithiobac sodium at dietary levels of 0, 10, 50, 500, 1,500 and 7,000 ppm, the NOAEL was 500 ppm (83.1 and 112 mg/kg/day, Ms/Fs) and the L0AEL was 1,500 ppm (263 and 384 mg/kg/day, Ms/Fs) based on increased liver weight and increased incidence of hepatocellular hypertrophy in Ms and decreased neutrophil count in Fs.

In a 90–day feeding study in dogs conducted with pyrithiobac sodium at dietary levels of 0, 50, 5,000, or 20,000 ppm, the NOAEL was 5,000 ppm (165 mg/kg/day) and the LOAEL was 20,000 ppm (626 mg/kg/day) based on decreased red blood cell count, hemoglobin, and hematocrit in females and increased liver weight in both sexes.

In a 21–day dermal study with rats conducted with pyrithiobac sodium at exposure levels of 0, 50, 500, or 1,200 mg/kg/day, the dermal irritation NOAEL was 500 mg/kg/day and the dermal irritation LOAEL was 1,200 mg/kg/day. There were no systemic effects observed at this high dose; therefore, the systemic NOAEL is considered to be 1,200 mg/kg/day.

5. *Chronic toxicity*. A 1–year feeding study in dogs conducted with pyrithiobac sodium at dietary levels of 0, 100, 5,000, and 20,000 ppm resulted in a NOAEL of 5,000 ppm (143 and 166 mg/kg/day, Ms/Fs) and a LOAEL of 20,000 ppm (580 and 647 mg/kg/day, Ms/Fs) based on decreases in bwt gain and increased liver weight.

A 78–week oncogenicity study in mice was conducted with pyrithiobac sodium at dietary levels of 0, 10, 150, 1,500 and 5,000 ppm. The systemic NOAEL is 1,500 ppm (217 and 319 mg/kg/day, Ms/Fs) and the LOAEL is 5,000 ppm (745 and 1,101 mg/kg/day, Ms/Fs), based on decreased bwt gain and liver lesions. Kidney effects were also observed at 5,000 ppm; however, these were present at low incidence and were of minimal severity and were considered to be of only minimal biological significance. Increased incidence of foci/focus of hepatocellular alteration was observed in males fed 5,000 ppm diets. Increased incidences of hepatocellular neoplasms (adenomas or adenomas plus carcinomas) were observed only in 150 and 1,500 ppm Ms. The incidence of these liver tumors was not significantly increased in the 5,000 ppm Ms or in Fs at any dose level; the 5,000 ppm male tumor incidence was within the historical control range.

A 2–year study in rats was conducted at dietary pyrithiobac sodium levels of 0, 5, 25, 1,500 or 5,000 ppm for Ms and 0, 5, 25, 5,000 or 15,000 ppm for Fs. The NOAEL for systemic effects was 1,500 ppm (58.7 mg/kg/day) for Ms and 5,000 ppm (278 mg/kg/day) for Fs. The lowest effect level (LEL) was 5,000 ppm (200 mg/kg/day for Ms)/15,000 ppm (918 mg/kg/day) for Fs. The LEL was based on the following: decreased bwt gain, and food efficiency (for Fs); mild changes in hematology and urinalysis, clinical signs indicative of urinary tract dysfunction (both sexes); increased incidence of focal cystic degeneration in the liver and increased rate of hepatic peroxisome beta-oxidation (Ms); and an increased incidence of inflammatory and degenerative microscopic lesions in the kidney (Fs). There was evidence of oncogenicity based on an increased trend for kidney tubular combined adenoma/carcinoma on male rats and an increased trend for kidney tubular adenomas in female rats. Although the incidences were low, they were statistically significant. The highest dose level tested (HDLT) in male rats (5,000 ppm) was considered adequate for assessment of oncogenic potential, that in female rats (15,000 ppm) exceeded the maximum tolerated dose (MTD).

6. *Carcinogenicity*. In consideration of PP 4F4391, the HED Carcinogenicity Peer Review Committee has previously concluded that the available data provide limited evidence of the carcinogenicity of pyrithiobac sodium in mice and rats and has classified pyrithiobac sodium as a Group C (possible human carcinogen with limited evidence of carcinogenicity in animals) in accordance with Agency guidelines published in the **Federal Register** of September 24, 1986 (51 FR 33992) and recommend that for the purpose of risk characterization a low-dose extrapolation model should be applied to the experimental animal tumor data for quantification for human risk (Q1*). This decision was based on liver adenomas, carcinomas and combined adenoma/carcinomas in the male mouse and kidney tubular adenomas, carcinomas and combined adenoma/carcinomas in the male rat. The unit risk, Q1* $(mg/kg/day)^{-1}$, of pyrithiobac sodium is $1.05 \times 10^{-3}$ $(mg/kg/day)^{-1}$ in human equivalents based on male kidney tumors.

7. *Animal metabolism*. Disposition and metabolism of pyrithiobac sodium were tested in M/F rats using two radio-labeled forms of pyrithiobac sodium. Either phenyl-labeled or pryimidine-labeled compounds were administered orally at 5 or 250 mg/kg. In addition, intravenous administration was evaluated at 5 mg/kg. Essentially all of the dose was excreted in the urine and feces, with greater than 90% being excreted within 48 hours. No label was detected in the expired air. Only minute quantities of radioactivity (at or near the limit of detection (LOD)) were detected in the major organs of metabolism and excretion. This study indicates that pyrithiobac sodium has low toxicity and does not accumulate within the body. The major compound eliminated in urine and feces was O-DPS (desmethyl metabolite), formed by demethylation of the pyrimidine ring. There was evidence that conjugation with glucuronic acid and 5-hydroxylation of the pyrimidine ring of pyrithiobac sodium were additional minor routes of metabolism in the rat.

The ruminant metabolism of pyrithiobac sodium was studied in lactating goats fed at a level of 15 mg/kg for 5 consecutive days, equaling a dose greater than 1,000 times the anticipated residues of pyrithiobac sodium and its metabolites in cottonseed, and greater than 100 times the anticipated residues in cotton gin byproducts. Seventy-six to 80% of the total administered dose was recovered in the excreta plus cage washes. Concentrations of radioactivity in milk, muscle, fat, whole-blood, and plasma were negligible. Biotransformation of the parent compound was not substantial with 90% of urine radioactivity and 40% of fecal extract corresponding to parent test substance. The major biotransformation pathway was O-demethylation. The results of this study indicate low potential for transfer of residues of pyrithiobac sodium and/or its metabolites into edible tissues or milk of ruminants, even at highly exaggerated feeding levels.

8. *Metabolite toxicology*. There is no evidence that the metabolites of pyrithiobac sodium as identified in either the plant metabolism, confined crop rotation, or animal metabolism studies are of any toxicological significance.

9. *Neurotoxicity*. A 90–day rat neurotoxicity screen battery conducted with pyrithiobac sodium resulted in a systemic NOAEL of 7,000 ppm (466 and 588 mg/kg/day, Ms/Fs) and a systemic LOAEL of 20,000 ppm (1,376 and 1,609 mg/kg/day, Ms/Fs) based on reduced bwt gain and food efficiency and increased liver weight. Slight reductions in hind-leg grip strength and slightly increased foot splay in Ms were observed in 20,000 ppm Ms. However, because these were of small magnitude, lacked statistical significance, and corresponding histopathology,

pyrithiobac sodium was not considered a neurotoxin. The NOAEL for neurotoxicity was 20,000 ppm highest dose tested (HDT).

10. *Endocrine disruption*. No special studies investigating potential estrogenic or other endocrine effects of pyrithiobac sodium have been conducted. However, the standard battery of required toxicology studies has been completed and found acceptable. These 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 to doses that far exceed likely human exposures. Based on these studies there is no evidence to suggest that pyrithiobac sodium has an adverse effect on the endocrine system.

*C. Aggregate Exposure*

1. *Dietary exposure*—i. *Food*. For purposes of assessing the potential dietary exposure under this tolerance, an estimate of aggregate exposure is made using the tolerance on cottonseed at 0.02 ppm. The potential exposure is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of cottonseed products translated as cottonseed meal and cottonseed oil eaten by various population subgroups. Cottonseed is fed to animals, thus exposure of humans to residues of cottonseed might result if such residues are transferred to meat, milk, poultry, or eggs. However, in consideration of PP 4F4391, CBTS has previously concluded that secondary residues in meat, milk, poultry, and eggs are not expected from the use of cottonseed (undelinted) as an animal feed. There are no other established tolerances or registered uses for pyrithiobac sodium in the United States. Based on a NOAEL of 58.7 mg/kg/day, from the chronic rat toxicity study and a 100-fold safety factor, the reference dose (RfD) is 0.58 mg/kg/day. Assuming residues at tolerance levels and that 100% of the crop is being treated, a theoretical maximum residue contribution (TMRC) of > 0.000001 mg/kg/day is calculated. With the above assumptions which clearly overestimate potential human exposure and are a most conservative assessment of risk, dietary (food) exposure to pyrithiobac sodium will utilize significantly less than 1% of the RfD for the overall U.S. population. For the most highly exposed subgroup, children aged 1–6 years, the TMRC is 0.000001 mg/kg/day, which is still less than 1% of the RfD. The unit risk, Q1* $(mg/kg/day)^{-1}$, of pyrithiobac sodium is $1.05 \times 10^{-3}$ $(mg/kg/day-^{1}$ in human equivalents based on male kidney tumors. Based on this upper bound potency factor (Q1*), a 70–year life span, and the assumption that 100% of the crop is treated with pyrithiobac sodium, the upper-bound limit of a dietary carcinogenic risk is calculated in the range of one incidence in a billion $(1.0 \times 10^{-9})$.

ii. *Drinking water*. Other potential dietary sources of exposure of the general population to pesticides are residues in drinking water. There is no maxium concentration level (MCL) established for residues of pyrithiobac sodium. The petitioner has reported to the Environmental Fate and Ground Water (EFGWB) branch of EPA the interim results of a prospective ground water monitoring study conducted at a highly vulnerable site. In consideration of this information in support of PP 4F4391, EFGWB has previously concluded by preliminary evaluation, that pyrithiobac sodium may not be stable enough to leach to ground water at most use sites, even in sandy soils. All other environmental fate data requirements for pyrithiobac sodium have been satisfied and based on these studies and the conditions of use, the potential for finding pyrithiobac sodium residues in drinking water is minimal.

2. *Non-dietary exposure*. Pyrithiobac sodium is not registered for any use which could result in non-occupational, non-dietary exposure to the general population.

*D. Cumulative Effects*

Pyrithiobac sodium is based on a new chemical class; there are no known registered herbicides with similar structure. Therefore, EPA should consider only the potential risks of pyrithiobac sodium in its exposure assessment. The herbicidal activity of pyrithiobac sodium is due to the inhibition of acetolactate synthase (ALS), an enzyme only found in plants. ALS is part of the biosynthetic pathway leading to the formation of branched chain amino acids. Animals lack ALS and this biosynthetic pathway. This lack of ALS contributes to the low toxicity of pyrithiobac sodium in animals. There is no evidence to indicate or suggest that pyrithiobac sodium has any toxic effects on mammals that would be cumulative with those of any other chemical.

*E. Safety Determination*

1. *U.S. population*. Based on a complete and reliable toxicity data base, the EPA has adopted an RfD value of 0.58 mg/kg/day using the NOAEL of 58.7 mg/kg/day, from the 2–year chronic toxicity study in rats and a 100-fold safety factor. Using crop tolerance levels and assuming 100% of the crop being treated a TMRC was calculated for the overall U.S. population and 22 population subgroups. This analysis concluded that aggregate exposure to pyrithiobac sodium will utilize significantly less that 1% of the RfD for either the entire U.S. population or any subgroup population. The TMRC for the most highly exposed subgroup identified as children aged 1–6 years was 0.000001 mg/kg/day. EPA generally has no concern for exposure below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to pyrithiobac sodium residues. The unit risk, Q1* $(mg/kg/day)^{-1}$, of pyrithiobac sodium is $1.05 \times 10^{-3}$ $(mg/kg/day)^{-1}$ in human equivalents based on male kidney tumors. Based on this upper bound potency factor (Q1*) and assuming a 70–year lifetime exposure an upper-bound limit of a dietary carcinogenic risk is calculated in the range of one incidence in a billion $(1.0 \times 10^{-9})$. This indicates a negligible cancer risk.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of pyrithiobac sodium, data from the previously discussed developmental and reproduction toxicity studies were considered. Developmental studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to reproductive and other effects on adults and offspring from prenatal and postnatal exposure to the pesticide. Based on the weight of these data, pyrithiobac sodium was not a reproductive toxicant. Maternal and developmental effects NOAEL's, LOAEL's were comparable indicating no increase in susceptibility of developing organisms. No evidence of endocrine effects were noted in any study. 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 prenatal and postnatal toxicity and the completeness of the data base. Based on current toxicological data requirements, the data base for pyrithiobac sodium relative to prenatal and postnatal effects for children is complete. The NOAEL of 58.7 mg/kg/day from the 2–year rat study with pyrithiobac sodium, which was used to calculate the RfD, is lower than any of the NOAEL's defined in the

developmental and reproductive toxicity studies with pyrithiobac sodium. When the weight of these facts is considered, an additional safety factor is not warranted for developmental effects. As stated above, aggregate exposure assessments utilized significantly less than 1% of the RfD for either the entire U.S. population or any of 22 population subgroups including infants and children. Therefore, it may be concluded that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to pyrithiobac sodium residues.

*F. International Tolerances*

There are no established Codex MRLs for pyrithiobac sodium on cottonseed. An established Mexican tolerance for pyrithiobac sodium on cottonseed is identical to the United States tolerance. Compatibility is not a problem at this time.

**3. Magna Bon Corporation**

*PP 8F4982*

EPA has received a pesticide petition [PP 8F4982] from Magna Bon Corporation, 3213 Ocean Drive, Vero Beach, FL 32963 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 to establish an exemption from the requirement of a tolerance for copper sulfate pentahydrate on the RAC copper sulfate pentahydrate at 0.050 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*. Copper sulfate pentahydrate has been used for years as a micronutrient, added to soils for up-take-into plants for sustaining vigorous growth. The metabolism is well-known in plant physiology as a vital component of plant growth. The labeled rate will not exceed any applications given during growth. The product will be applied post-harvest and no additional metabolism of harvested products is expected.

2. *Analytical method*. Standard methodology for copper sulfate is adequate.

3. *Magnitude of residues*. The cover letter (attached) notes the various clearances based on uses in plants, animals, humans and potable water.

The products will be applied according to labels approved by EPA which are at or below the levels on the current labeled rates for application to growing crops. The plants will only be exposed to washes of the product. Since the product is not systemic, the product can be washed from the surface of the plant or animal parts before being consumed.

*B. Toxicological Profile*

The toxicology of copper compounds are well-known. The toxicology file for Mega Bon Corporation registrations are incorporated by reference.

1. *Acute toxicity*. Copper and the salts are solids. Individuals use copper bracelets, and chains in contact with their skin as jewelry. There is no known skin sensitization. Please refer to 21 CFR 184.1261 when used as a human supplement.

2. *Genotoxic*. There is no known genotoxicity. All studies have been negative.

3. *Reproductive and developmental toxicity*.[1]

4. *Subchronic toxicity*.[1]

5. *Chronic toxicity*.[1]

6. *Animal metabolism*.[1]

7. *Metabolite toxicology*.[1]

*C. Aggregate Exposure*

1. *Dietary exposure*. Copper is used in vitamins and occurs on a very small part of the daily foods. However, the small amount that may occur on plants is washed off prior to food preparation.

Copper being used as a crop protector or as a post-harvest application may add little to the exposure given the use pattern and general application of new fungicides.

i. *Food*. The total consumption of all agricultural, fish, shell-fish, and meat treated with copper sulfate pentahydrate can be calculated as being at or below daily minimums of mineral requirements for humans. In addition, the plant and meat products are washed before cooking.

ii. *Drinking water*. A food additive tolerance of 2 ppm in potable water is established under 40 CFR 185.1200 for residues of copper from use of copper compounds.

2. *Non-dietary exposure*. The population is exposed to copper compounds on an almost daily basis. Dermal exposure is the most prevalent. There have been several impingements by the copper compounds with little to no effect.

[1]Although there are no guidline studies for this data requirement *per se*, there is adequate information in the extensive open literture on copper sulfate to characterize its toxicity.

*D. Cumulative Effects*

The amount of copper sulfate pentahydrate used to treat the harvested plant products, fish, shellfish, poultry, and meat would be a way of lowering bacterial, fungi and even-viral organisms from becoming a problem under most circumstances.

*E. Safety Determination*

1. *U.S. population*. Using the copper sulfate pentahydrate would reduce costs of protecting the above-mentioned products and giving adequate protection to such target post-harvested crops, fish, shellfish, poultry, and meat products without harm to humans, animals, plants, plant products, and the environment.

2. *Infants and children*. Foods are washed and processed. Copper sulfate pentahydrate is a solid, but will be washed. The foods are further processed with little or no detectable levels. The copper in the application is a vital nutrient for infants and children.

*F. International Tolerances*

The countries of the world have not restricted copper for the purposes we request.

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## ENVIRONMENTAL PROTECTION AGENCY

**[OPP–00609; FRL–6088–6]**

## Pesticides; Policy Issues Related to the Food Quality Protection Act

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Notice of availability.

**SUMMARY:** To assure that EPA's policies related to implementing the Food Quality Protection Act are transparent and open to public participation, EPA is soliciting comments on a draft science policy paper entitled ''The Role of Use-Related Information in Pesticide Risk Assessment and Risk Management.'' This notice is the tenth in a series concerning science policy documents related to the Food Quality Protection Act and developed through the Tolerance Reassessment Advisory Committee.

**DATES:** Written comments, identified by docket control number OPP–00609 should be submitted by September 13, 1999.

**ADDRESSES:** Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as