examination. The revised DOE certification plan will continue to cover only employees of the Bonneville Power Administration. The DOE estimates that there will be 100 applicators certified in the new wood treatment category. There are presently approximately 150 applicators certified in the right-of-way category, whose certification will be unaffected by this action.

No comments were received on EPA's notice of intention to approve the revised DOE certification plan. Therefore, EPA approves the revised DOE certification plan.

List of Subjects

Environmental protection.

Dated: September 9, 1997.

Lynn R. Goldman,

Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 97–25337 Filed 9–23–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–764; FRL–5745–8]

E.I. DuPont de Nemours and Co., Inc.; Pesticide Tolerance Petition Filing

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–764, must be received on or before October 24, 1997.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Division (7506C), 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 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. 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: James Stone, PM-25 Team, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 257, Crystal Mall #2 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–7391; e-mail: stone.james@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemical 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 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 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–764] (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 control number (PF–764) 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: September 11, 1997.

Peter Caulkins,

Acting 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.

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

PP 4F4391

EPA has received a pesticide petition (PP 4F4391) from E.I. DuPont de Nemours and Co., Inc (DuPont), Barley Mill Plaza, P.O. Box 80083, Wilmington, DE 19880–0038 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 pyrithiobac sodium salt (sodium 2-chloro-6-[(4,6-dimethoxypyrimidin-2-yl)thio]benzoate) in or on the raw agricultural commodities cottonseed at 0.02 part per million (ppm) and cotton gin byproducts at 0.10 (ppm). The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by using ultraviolet detection. 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). 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 CBTS, in consultation with the 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 are independently validated practical analytical methods available using liquid chromatography (HPLC) with column switching and ultraviolet (UV) detection, to measure levels of pyrithiobac sodium in or on cottonseed and cotton gin byproducts, with limits of quantitation that will allow for monitoring of crop residues at or above tolerance levels. EPA has previously provided information on the method for cottonseed to FDA for future publication in PAM II.

3. *Magnitude of residues*. Crop field trial residue data from 60 day PHI studies show that the proposed pyrithiobac sodium tolerances on these raw agricultural commodities will not be exceeded when pyrithiobac sodium is used as directed. An adequate cottonseed processing study shows that pyrithiobac sodium does not concentrate in cottonseed processed commodities. No tolerances on processed 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 of 2,000 mg/kg, the highest dose tested (HDT). Pyrithiobac sodium has been placed in Toxicity Category III for acute oral toxicity based on acute oral LD_{50}s of 3,200 mg/kg for both male and female 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 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. *Genotoxicty*. 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 male and female 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 two 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 NOEL of 1,500 ppm (103 mg/kg/day) and a maternal LOEL of 7,500 ppm (508 mg/kg/day), based on decreased body weight gain and food efficacy. An offspring NOEL of 7,500 ppm (508 mg/kg/day) and LOEL of 20,000 ppm (1,551 mg/kg/day) were also demonstrated based on decreased offspring body weight. Pyrithiobac sodium was not teratogenic when administered to rats or rabbits.

A developmental toxicity study with pyrithiobac sodium in rats demonstrated a maternal NOEL of 200 mg/kg and LOEL of 600 mg/kg due to increased incidence of salivation. A developmental NOEL of 600 mg/kg and LOEL 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 NOELs of 300 mg/kg and a maternal LOEL of 1,000 mg/kg based on mortality, decreased body weight gain and feed consumption, increased incidence of clinical signs, and an increase in early resorptions. A developmental LOEL of 1,000 mg/kg was based on decreased fetal body weight 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 NOEL was 500 ppm (31.8 and 40.5 mg/kg/day, m/f and the LOEL was 7,000 ppm (466 and 588 mg/kg/day, m/f) based on decreased body weight gains and increased rate of hepatic B-oxidation in males.

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 NOEL was 500 ppm (83.1 and 112 mg/kg/day, m/f) and the L0EL was 1,500 ppm (263 and 384 mg/kg/day, m/f) based on increased liver weight and increased incidence of hepatocellular hypertrophy in males and decreased neutrophil count in females.

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 NOEL was 5,000 ppm (165 mg/kg/day) and the LOEL 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 NOEL was 500 mg/kg/day and the dermal irritation LOEL was 1,200 mg/kg/day. There were no systemic effects observed at this high dose; therefore, the systemic NOEL 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 NOEL of 5,000 ppm (143 and 166 mg/kg/day, m/f) and a LOEL of 20,000 ppm (580 and 647 mg/kg/day, m/f) based on decreases in body weight 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

NOEL is 1,500 ppm (217 and 319 mg/kg/day, m/f) and the LEL is 5,000 ppm (745 and 1,101 mg/kg/day, m/f), based on decreased body weight 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 males. The incidence of these liver tumors was not significantly increased in the 5,000 ppm males or in females 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 males and 0, 5, 25, 5,000 or 15,000 ppm for females. The NOEL for systemic effects was 1,500 ppm (58.7 mg/kg/day) for males and 5,000 ppm (278 mg/kg/day) for females. The LEL was 5,000 ppm (200 mg/kg/day for males)/15,000 ppm (918 mg/kg/day) for females. The LEL was based on the following: decreased body weight, body weight gain and food efficiency (for females); mild changes in hematology and urinalysis, clinical signs indicative of urinary tract dysfunction (both sexes); increased incidence of focal cystic degereration in the liver and increased rate of hepatic peroxisome beta-oxidation (males); and an increased incidence of inflammatory and degenerative microscopic lesions in the kidney (females). There was evidence of oncogenicity based on an increased trend for kidney tubular combined adenoma/carcinoma in 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 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).

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** in 1986 (51 FR 33992, Sept. 24, 1986) 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 (Q_1*). 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, Q_1* $(mg/kg/day)^{-1}$, of pyrithiobac sodium is 1.05×10^{-3} (mg/kg/day)-1 in human equivalents based on male kidney tumors.

6. *Animal metabolism*. Disposition and metabolism of pyrithiobac sodium were tested in male and female rats using two radiolabeled forms of pyrithiobac sodium. Either phenyl-labeled or pryimidine-labeled compounds were administered orally at 5 or 250 mg/kg. In addition, i.v. 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) 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 1000 times the anticipated residues of pyrithiobac sodium and its metabolites in cottonseed, and greater than 100 times the anticipated residues in cotton gin byproducts. Of the total administered dose 76–80% was recovered in the excreta plus cagewashes. 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.

7. *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.

i. *Neurotoxicity*. A 90–day rat neurotoxicity screen battery conducted with pyrithiobac sodium resulted in a systemic no observed-effect level (NOEL) of 7,000 ppm (466 and 588 mg/kg/day, m/f) and a systemic lowest-observed-effect level (LOEL) of 20,000 ppm (1,376 and 1,609 mg/kg/day, m/f) based on reduced body weight gain and food efficiency and increased liver weight. Slight reductions in hind-leg grip strength and slightly increased foot splay in males were observed in 20,000 ppm males. However, because these were of small magnitude, lacked statistical significance and corresponding histopathology, pyrithiobac sodium was not considered a neurotoxin. The NOEL for neurotoxicity was 20,000 ppm (HDT).

ii. *Endocrine effects*. 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*. It is proposed that pyrithiobac sodium be defined as the residue for enforcement purposes. Monitoring for pyrithiobac sodium residues in field samples will provide an adequate estimate of this compound in edible portions of treated crops.

2. *Food*—i. *acute dietary exposure*. A Tier I acute dietary exposure analysis was conducted using the Dietary Exposure Evaluation Model (DEEM ver. 5.10) and assuming tolerance level residues for cottonseed oil, cottonseed meal, and a very conservative residue value of 6 parts per billion (ppb) for all sources of dietary water. Using the acute endpoint of 200 mg/kg from a developmental toxicity study in rats, the margins of exposure were greater than 100,000 for all 22 population subgroups at the 95th percentile exposure.

ii. *Chronic dietary exposure.* For purposes of assessing the potential chronic dietary exposure under this tolerance, an estimate of aggregate exposure is made using the proposed tolerance on cottonseed at 0.02 ppm, cotton gin byproducts at 0.10 ppm, and a very conservative contribution from drinking water based on GENEEC modeling. 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 and cotton gin byproducts are fed to animals, thus exposure of humans to residues of pyrithiobac sodium might result if such residues are transferred to meat, milk, poultry, or eggs. However, in previous consideration of PP 4F4391 CBTS has concluded that secondary residues in meat, milk, poultry and eggs are not expected from the use of cottonseed as an animal feed. A ruminant (goat) metabolism study further demonstrates that residues of pyrithiobac sodium in cotton gin byproducts will not result in secondary meat or milk residues when this commodity is fed to livestock. There are no other established tolerances or registered uses for pyrithiobac sodium in the United States. Based on a NOEL 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 < 0.1 mg/kg/day is calculated using the DEEM computer software (version 5.1, Novigen Sciences, Inc., 1997). 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, non-nursing infants less than 1 year old, the TMRC is also < 0.1 mg/kg/day, which is still less than 1% of the RfD. The unit risk, Q_1* $(mg/kg/day)^{-1}$, of pyrithiobac sodium is 1.05×10^{-3} $(mg/kg/day)^{-1}$ in human equivalents based on male kidney tumors. Based on this upper bound potency factor (Q*), a 70–year lifespan, 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 1 incidence in a billion (1.0×10^{-9}).

3. *Drinking water* . Other potential dietary sources of exposure of the general population to pesticides are residues in drinking water. There is no Maximum Contaminant Level established for residues of pyrithiobac sodium. The petitioner has reported to the Environmental Fate and Groundwater Branch of EPA (EFGWB) the results of a prospective groundwater monitoring study conducted at a highly vulnerable site. This study confirms the previous interim conclusions of EFGWB that pyrithiobac sodium may not be stable enough to leach to groundwater at most use sites, even in sandy soils. The potential for pyrithiobac sodium to enter surface water is also very low. This is supported by modeling done using GENEEC which under worst case conditions (100% of area treated, long half-life, etc.) predicted peak surface water concentrations of only 6 ppb. All environmental fate data requirements for pyrithiobac sodium have now been satisfied and based on these studies, the conditions of use, and worst-case modeling, the potential for finding pyrithiobac sodium residues in drinking water is minimal.

4. *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 database, the EPA has adopted an RfD value of 0.58 mg/kg/day using the NOEL 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 treated, a Theoretical Maximum Residue Contribution (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 than 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 non-nursing infants less than 1 year old was also < 0.1 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, Q_1* $(mg/kg/day)^{-1}$, of pyrithiobac sodium is 1.05×10^{-3} $(mg/kg/day)^{-1}$ in human equivalents based on male kidney tumors. Based on this upper bound potency factor (Q_1*) and assuming a 70 year lifetime exposure an upper-bound limit of a dietary carcinogenic risk is calculated in the range of 1 incidence in a billion (1.0×10^{-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 pre-natal development. Reproduction studies provide information relating to reproductive and other effects on adults and offspring from pre-natal and post-natal exposure to the pesticide. Based on the weight of these data, pyrithiobac sodium was not a reproductive toxicant. Maternal and developmental effects (NOEL's, LOEL'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 pre and post-natal toxicity and the completeness of the database. Based on current toxicological data requirements, the database for pyrithiobac sodium relative to pre- and post-natal effects for children is complete. The NOEL 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 NOEL'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 U.S. tolerance. Compatibility is not a problem at this time.

[FR Doc. 97–25234 Filed 9-23-97; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–761; FRL–5740–9]

Yoshitomi Fine Chemicals Ltd.; Pesticide Tolerance Petition Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of tolerances for residues of 4,5-Dichloro-1,2-Dithiol-3-one (CASRN 1192–52–5) in or on paper and paperboard.

DATES: Comments, identified by the docket control number PF–761, must be received on or before October 24, 1997.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (7506C), 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 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. 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: By mail: Portia Jenkins, Acting Product Manager (34), Antimicrobials Division (7510C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 6C, Crystal Plaza #1, 2800 Crystal Drive, Arlington, VA, (703) 308–6230; e-mail: jenkins.portia@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition ((PP) 7F4902) from Yoshitomi Fine Chemicals, Ltd., 6–9, Hiranomachi 2-chome, Chuo-ku, Osaka, 541, Japan, 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 185 ''Tolerances for Pesticides in Food'' by establishing Subpart D ''Tolerance Exemptions for Pesticides in Foods'' and promulgating therein section 185.9000 establishing a tolerance exemption for residues of the slimicide 4,5-Dichloro-1,2-Dithiol-3-one (CASRN 1192–52–5) in or on paper and paperboard resulting from its addition to pulp and paper mill process water to control slime forming organisms. EPA has determined that the 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 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–761] (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''.

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–761) and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Administrative practice and procedure, Paper and paperboard, Slimicides, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: September 16, 1997.

Frank Sanders,

Director, Antimicrobials Division, Office of Pesticide Programs.

Summary of Petition

Petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by the petitioner and represent the views of the petitioner. 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.

Yoshitomi Fine Chemicals, Ltd.

A. Residue Chemistry

This petition is not for residues in or on raw agricultural commodities. It is for residues in or on food contact paper or paperboard. Accordingly, the residue chemistry data submitted are solely for the residues remaining in food contact paper and paperboard when the subject slimicide (4,5-Dichloro-1,2-Dithiol-3-one, CASRN 1192–52–5, hereafter referred to as RYH–86) is used in pulp and paper mill process water to control slime forming organisms.

1. *Residues in paper and paperboard.* GC-MS-SIM analysis of approximately 30 paper and paperboard samples manufactured in a papermill which used RYH–86 amended slurry water revealed no RYH–86 detectable with a detection limit of 100 µg/kilograms (Kg) of paper (i.e., 100 parts per billion (ppb)). Extraction of such samples with