The Environmental Protection Agency's (EPA) Environmental Financial Advisory Board (EFAB) has been renewed for a two-year period, as a necessary committee which is in the public interest, in accordance with the provisions of the Federal Advisory Committee Act (FACA). The purpose of EFAB is to provide authoritative analysis and advice to the EPA Administrator regarding environmental finance issues to assist EPA in carrying out its environmental mandates. EFAB will strive to increase the total investment in environmental protection by facilitating greater leverage of public and private environmental resources.

Dated: March 9, 1998.

Michael W.S. Ryan,

Comproller.

[FR Doc. 98–7138 Filed 3–18–98; 8:45 am]

BILLING CODE 6560–50–M

ENVIRONMENTAL PROTECTION AGENCY

[PF–797; FRL–5776–7]

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 agricultural commodities.

DATES: Comments, identified by the docket control number PF–797, must be received on or before April 20, 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 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. 119 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: James A. Tompkins, Product Manager (PM) 25, Registration Division, (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 239, 1921 Jefferson Davis Hwy., Arlington, VA., (703) 305–5697; e-mail: Tompkins.jim@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 raw agricultural 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 grantinig of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice, as well as the public version, has been established for this notice of filing under docket control number PF–797 (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 file format or ASCII file format. All comments and data in electronic form must be identified by the docket control number PF–797 and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

Authority: 21 U.S.C. 346a.

List of Subjects

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

Dated: March 3, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

E.I. du Pont de Nemours & Company, Agricultural Products

PP 3F4215

EPA has received a pesticide petition (PP 3F4215) from E.I. du Pont de Nemours & Company, Agricultural Products, P.O. Box 80038, 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 metsulfuron methyl (methyl-2-[[[[(4-methoxy-6-methyl-1–3, 5-triazin-2-yl)amino]carbonyl] amino]sulfonyl]benzoate) in or on the raw agricultural commodities sorghum grain at 0.1 parts per million (ppm), sorghum forage at 0.2 ppm, and sorghum fodder at 0.2 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residues of metsulfuron methyl is adequately understood. Metabolism studies conducted with radioactive 14C-metsulfuron methyl on wheat and barley under field conditions and on wheat under greenhouse conditions showed that residues dissipate rapidly in plants, primarily due to growth dilution. In these metabolism studies conducted at exaggerated rates, wheat and barley grain did not contain any detectable level of metsulfuron methyl or its metabolites (<0.01 mg/kg). Residues of individual metabolites were very low in straw in studies conducted at 35 g a.i./ha (0.5 oz a.i./acre, <0.01 to 0.02 mg/kg). The only situation where residues of an

individual substance was detected in straw above 0.1 mg/kg was under greenhouse conditions at 70 g a.i./ha (1 oz a.i./acre), (8 X maximum recommended rate), metsulfuron methyl residue level measured in straw at maturity was 0.44 mg/kg (other individual metabolites were below 0.1 mg/kg).

The initial step of the metabolic breakdown of metsulfuron methyl involves either hydroxylation of the phenyl ring and subsequent conjugation with glucose or cleavage of the sulfonylurea bridge. The latter process results in triazine amine derivatives from one side of the molecule and sulfonamide derivatives from the other side, which may further evolve to saccharin through cyclization.

Plant/animal comparative metabolism showed two plant unique metabolites (4-hydroxy metsulfuron methyl and its glucose conjugate). However they do not occur at detectable levels (< 0.01 mg/kg) in cereal grain, even at exaggerated rates of application. For this reason they were not subject to any testing and were not of concern for the purpose of establishing the proposed tolerance.

Based on the absence of detectable residue in food commodities (wheat and barley grain) and on the expected low residue levels of individual substances in feed items (straw) under normal use conditions, and the Residue Chemistry Guidelines (OPPTS 860–1300, D, ii) which states that; one metabolism study will be required for each of the crop groups defined in CFR 40 180.34(f) except for herbs and spices, a plant metabolism study in grain sorghum was not required.

A confined crop rotation study was conducted using sugar beets, oats, rape and soybeans as following crops, an application rate of 16 g a.i./ha, (0.23 oz a.i./acre), (2 × the maximum recommended rate) and a 120–day treatment-to-planting interval. A field crop rotation study was also conducted using oats, rape, sorghum and soybean as following crops, a 30 g a.s./ha, (0.86 oz a.i./acre), application rate and a 1-year treatment-to-planting interval. Residues of metsulfuron methyl or its degradation products were not detected in any edible crop commodities (<0.01 mg/kg), suggesting that use of metsulfuron methyl should not expose consumers to detectable residues in food through following crops.

2. *Analytical method*. The quantification of metsulfuron methyl is by HPLC/UV (high performance liquid chromatography/ultra violet) utilizing eluent and column switching with UV absorbance detection at 254 nm. The LOQ (limits of quantitation) of the analytical method for sorghum is 0.10 ppm for metsulfuron methyl and its metabolite (4-hydroxy metsulfuron methyl) in grain and fodder, 0.050 ppm for metsulfuron methyl and its metabolite in forage, 0.070 ppm for the glucose conjugate metabolite in grain and forage, and 0.14 ppm for the glucose conjugate metabolite in fodder. The LOQ of the analytical method for metsulfuron methyl and its metabolite in wheat and barley is 0.05 ppm for wheat/barley forage or grain and 0.10 ppm for wheat/barley straw.

3.—a. *Magnitude of residues*. The results of an analyses of sorghum grain, fodder and stover (at seed maturity), forage and hay (30 days), after application of metsulfuron methyl at the maximum proposed label rate and twice the rate, show that all residues of metsulfuron methyl and its metabolites (4-hydroxy metsulfuron methyl and its glucose conjugate) were below the limit of quantitation (0.05 or 0.1 ppm).

b. *Magnitude of residues in processed commodities.* Sorghum was field treated with metsulfuron methyl at exaggerated rates and samples were analyzed for metsulfuron methyl and its metabolites in bran, large grits, small grits, flour, grain dust, starch and gluten. All residues of metsulfuron methyl and it's metabolites in sorghum seeds and its processed fractions were below the limit of quantitation (<0.02–0.05 ppm).

B. Toxicological Profile

1. *Acute toxicity*. Based on EPA criteria, technical metsulfuron methyl is in acute toxicity Category IV for oral and inhalation routes of exposure and for dermal irritation and Category III for the dermal route of exposure and for eye irritation. It is not a skin sensitizer.

Acute oral toxicity in rats LD50>5000 mg/kg

Acute dermal toxicity in rabbits LD50>2000 mg/kg

Acute inhalation toxicity in rats LD50>5.0 mg/L

Primary eye irritation in rabbits Effects reversed within 72 hours.

Primary dermal irritation in rabbits No irritation observed.

Dermal sensitization in guinea pigs Non-sensitizer.

2. *Genotoxicty*. Metsulfuron methyl has shown no genotoxic activity in the following listed *in-vitro* and *in-vivo* tests, except for *in-vitro* chromosomal aberration (CHO):

Ames Negative

Mammalian gene mutation (CHO/HGPRT) Negative

Unscheduled DNA synthesis Negative

In-vivo bone marrow cytogenetics Negative

In-vivo mouse micronucleus Negative

In-vitro chromosomal aberration (CHO) Positive

Metsulfuron methyl was only positive at concentrations > 1,000 mg/L in an *in vitro* test for induction of chromosome aberrations in Chinese Hamster Ovary cells. *In vivo* studies included the assessment of chromosome aberrations by metaphase analysis in bone marrow of male and female rats and the evaluation of micronuclei in bone marrow polychromatic erythrocytes of male and female mice. The results of both studies were negative when exposures were conducted up to 5,000 mg/kg. The fact that no effects were observed in the more definitive *in vivo* tests and considering the negative results in all other genotoxicity studies, the weight-of-evidence indicates that metsulfuron methyl is neither genotoxic nor mutagenic.

3. *Reproductive and developmental toxicity*. The results of a series of studies indicated that there were no reproductive, developmental or teratogenic hazards associated with the use of metsulfuron methyl. In a rat multigeneration reproduction study, reduced parental body weights were observed for both generations at the highest dose tested, 5,000 ppm. There were no effects on fertility, lactation, litter size or pup survival. The NOEL was 500 ppm (or 34 to 43 mg/kg bw/day).

In studies conducted to evaluate developmental toxicity potential, metsulfuron methyl was neither teratogenic nor uniquely toxic to the conceptus (i.e., not considered a developmental toxin). In the rat study, maternal toxicity, presented as reduced food consumption and body weight gain, was observed at 250 mg/kg bw and above. The systemic NOEL for the dams was 40 mg/kg/day. There were no effects on the conceptus at the highest dose tested, 1,000 mg/kg/day. Therefore, the fetal NOEL for rats is greater than 1000 mg/kg/day. In the rabbit developmental toxicity study, maternal mortality, reduced food consumption, and reduced body weights were observed at or above 100 mg/kg bw. The NOEL for maternal toxicity in rabbits was 25 mg/kg, based on maternal mortality and body weight decreases. Impact on the fetuses was minimum at these maternally toxic doses and was characterized only by a non-statistically significant trend in incomplete ossification of frontal bones at 100 and 300 mg/kg bw and above. The NOEL for fetal toxicity in rabbits was >700 mg/kg, the highest dose tested.

4. *Subchronic toxicity*. Repeated dietary exposures to metsulfuron methyl presented low toxicity manifested as reduced food consumption and body weight gain in the rat and the dog. There were no adverse effects observed in mice in subchronic studies at the highest dose tested, 5,000 ppm. The NOEL for subchronic exposure in mice was >5000 ppm (814 and 944 mg/kg/day, M/F). The rat was the most sensitive species tested in subchronic toxicity studies. The NOEL was 1,000 ppm (68 and 84 mg/kg/day for males amd females respectively) based on decreased body weights, body weight gains, and total serum protein in females, and decreased relative liver weights in males exposed at 7,500 ppm. In a 90–day feeding study in dogs, the NOEL was 5,000 ppm (134 and 129 mg/kg/day, M/F), the highest dose tested.

A 21–day dermal study was conducted in rabbits at 0, 125, 500 or 2,000 mg/kg/day. The NOEL was 125 mg/kg/day based on dermal effects at the application site; the NOEL for systemic toxicity was 2,000 mg/kg/day.

5. *Chronic toxicity*. Chronic Toxicity studies of metsulfuron methyl resulted in only minimal effects in the rat, mouse, or dog. Metsulfuron methyl was not oncogenic in the chronic rat and mouse bioassays.

A 1–year feeding study in dogs, the NOEL for chronic toxicity in beagle dogs was 500 ppm (or 13 mg/kg/day) and 5,000 ppm (or 127 mg/kg/day) in male and female dogs, respectively. Metsulfuron methyl produced minimal toxicity after 12 months administration to male beagle dogs, manifested as minimal interference with normal nutrition by decreasing food consumption toward the end of 1 year. This minimal interference was not considered adverse since it did not cause changes in body weights or body weight gains.

In an 18–month study in mice, the NOEL was 5,000 ppm (666 and 836 mg/kg/day for males and females, respectively), the highest dose tested. Metsulfuron methyl is not an oncogen in this study.

A 2–year combined chronic toxicity and oncogenicity study in rats, the NOEL was 500 ppm (or 23 and 30 mg/kg/day for males and females, respectively). Metsulfuron methyl was not oncogenic in rats nor was target organ toxicity evident after two years administration. Chronic toxicity was manifested as minimal interference with normal nutrition and subsequent decreases in body weight gain that were more pronounced during the early growth phase of the animals life span and became less evident toward the end of the study.

6. *Animal metabolism*. The metabolism of metsulfuron methyl in animals (rat, hen and goat) is adequately understood and similar among the species evaluated. The rat metabolism and disposition data indicated rapid absorption, metabolism and elimination. In the rat, approximately 90% of the administered dose of metsulfuron methyl was excreted in the feces and urine within 72 hours. The biological half-lives were 9–16 hours for low-dose groups and 23–29 hours for high-dose groups. The major pathway was breakdown of the urea bridge to give rise to either aminosulfonyl benzoate or sulfonamide and the triazine amine derivative. The secondary biotransformation pathway was demethylation of aminosulfonyl benzoate to form saccharin. Preconditioning with low-dose exposures did not affect the metabolism of metsulfuron methyl. There was no evidence of accumulation of metsulfuron methyl or its metabolites in any organ or tissue. A significant portion (85–95%) of the recovered radioactivity from urine, feces and tissues was intact metsulfuron methyl. There were two major plant specific metabolites identified, that were not detected in the rat. However, in residue studies, no detectable residues of parent or major plant unique metabolites, were found in the feed and food items of cereal crops treated at the maximum seasonal use rate. Hence, toxicity testing of other degradation products of metsulfuron methyl was not needed.

Results from a metabolism study with two radioactive forms of metsulfuron methyl, (14C-Phenyl and 14C-Triazine) in the laying hens show that virtually all the radioactivity was eliminated in the excreta. The total radioactivity in edibale tissues and eggs represented </=0.2% of the total radioactivity administered for either radiolabel. Parent metsulfuron methyl was excreted largely unchanged, and a minor portion is metabolized to o- desmethyl metsulfuron methyl.

The fate of metsulfuron methyl and its metabolite was investigated in the lactating goat. Metsulfuron methyl and the metabolite were eliminated mostly in the urine and feces. Traces of radioactivity were found in some tissues and in milk (0.008–0.009%) of the parent and no radioactivity of the metabolite was detected in the milk or any organ or tissue sample.

In a cattle feeding study, metsulfuron methyl was rapidly excreted in the urine and feces of the treated cows. Less than 0.1% of the daily dose was excreted in the milk as metsulfuron methyl and <10% of the metsulfuron methyl residue level was found as the glucoronide conjugate. Residues (<0.1 ppm) were found in the kidney of cows slaughtered 12 hours after treatment stopped but not in cows slaughtered a week later.

Tolerances for metsulfuron methyl in fat (0.1 ppm), meat (0.1 ppm), meat by products (0.1 ppm), and kidney (0.5 ppm) of cattle, goats, hogs, horses and sheep, and a tolerance of 0.05 ppm in milk have been posted in 40CFR 180.428.

7. *Metabolite toxicology*. There is no evidence that the metabolites of metsulfuron methyl as identified in either the plant or animal metabolism studies are of any toxicological significance.

8. *Endocrine disruption*. No special studies investigating potential estrogenic or other endocrine effects of metsulfuron methyl have been conducted. However, the standard battery of required toxicology studies has been completed. 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 metsulfuron methyl has an adverse effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. Tolerances have been established (40 CFR 180.428) for the residues of metsulfuron methyl in or on various food commodities ranging from 0.05 ppm in milk to 0.5 in kidney. There are no potential sources of exposure of the general population to residues of metsulfuron methyl from drinking water or non-occupational sources such as in door and out door residential uses. There are no in door or out door residential uses registered for metsulfuron methyl. There are no acute dietary exposure or cancer risk endpoints of concern for metsulfuron methyl. Aggregate risk has been assessed from chronic exposure to food.

2. *Food*. Tolerances have been established for metsulfuron methyl on the following food crops: barley, wheat, and sugar cane. A tolerance of 0.1 ppm for sorghum grain was included in this assessment. Also included were tolerances for meat and milk commodities. The dietary exposure was estimated using the Dietary Exposure Evaluation Model (DEEM ver. 5.03) which utilizes the 1989–1991 CSFII food consumption database. In conducting this assessment the

conservative assumption was made that residues would be at the tolerance level. Use of the tolerances rather than actual field measurements will result in an overestimate of human dietary exposure. The existing metsulfuron methyl tolerances with the addition of the sorghum tolerance result in a theoretical maximum residue level (TMRC) that is equivalent to the following percentages of the RfD:

U.S. Population 0.3%
Nursing Infants (<1 year old) 0.1%
Non-Nursing Infants (<1 year old) 0.4%
Children (1–6 years old) 0.8%
Children (7–12 years old) 0.5%

Thus, the estimated exposure for the U.S. population and all subpopulation was less than 1% of the RfD. Metsulfuron methyl RfD (0.3 mg/kg/day)is based on the NOEL for the 2–year rat study. The most sensitive chronic toxicity/oncogenicity study. The subpopulation with the highest exposure was children ages 1–6 years (0.8% of the RfD). Based on the residue data, potential for dietary exposure is extremely low. Residue studies have shown no residue above LOQ (0.05 or 0.02 ppm) in sorghum samples evaluated including the sorghum grain processed fractions. No dietary exposure is anticipated from secondary residues in meat or milk. Although sorghum is considered a major foodstuff for cattle and poultry, residue studies and metabolism studies in the laying hen and lactating goat and cattle feeding studies showed residues below LOQ of processed fractions and less than 2% of the administered concentration in edible meat and eggs. Only traces of metsulfuron methyl were found in some goat meat and milk (0.008–0.009).

Direct human consumption of sorghum grain as a food commodity in the U.S. is extremely low. At the above levels of exposure, there is a reasonable certainty that no harm will result from dietary exposure to metsulfuron methyl.

3. *Drinking water*. Another potential source of dietary exposure to pesticides are residues in drinking water. There is no established Maximum Contaminant Level (MCL) for metsulfuron methyl in water. Based on the low use rate of metsulfuron methyl and a use pattern that is not widespread, DuPont does not anticipate residues of metsulfuron methyl in drinking water and exposure from this route is unlikely.

4. *Non-dietary exposure*. Metsulfuron methyl is registered for use in weed and brush control in non-crop situations including industrial, unimproved turf areas. Metsulfuron methyl is not to be used on lawns, walks, drive ways, tennis courts, golf courses, athletic fields, commercial sod operations, or other high maintenance, fine turf grass areas, or similar areas. Any non-occupational exposure to metsulfuron methyl in the unimproved areas is likely to be negligible.

D. Cumulative Effects

Metsulfuron methyl belongs to the sulfonylurea class of compounds. The herbicidal activity of the sulfonylurea is due to the inhibition of acetolactase 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 the sulfonylurea compounds in animals. We are aware of no information to indicate or suggest that metsulfuron methyl has any toxic effects on mammals that would be cumulative with those of any other chemicals.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above, and based on the most sensitive chronic NOEL of 25 mg/kg/day and an RfD of 0.3 mg/kg/day, the aggregate dietary exposure will utilize less than 1% of the RfD for the U.S. population. Generally, exposure below 100% of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose risk to human health. We therefore conclude that there is a reasonable certainty that no harm will result from aggregate exposure to metsulfuron methyl residues.

Although no formal acute dietary margin of exposure (MOE) determinations were made, it is highly unlikely that the MOE would exceed a level of concern due to the low acute mammalian toxicity of this compound].

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of metsulfuron methyl, data were considered from developmental toxicity studies in the rat and the rabbit, and a multi-generation reproduction study in the rats. These studies proved that metsulfuron methyl was not a teratogenic or a developmental toxin.

Using the conservative exposure assessment described above, the percent of the RfD that will be utilized ranges from 0.1 to 0.8% for infants and young children. Based on this we conclude that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to metsulfuron methyl residues.

Although no formal acute dietary margin of exposure determinations were made, it is highly unlikely that the MOE would exceed a level of concern due to the low mammalian toxicity of this compound.

F. International Tolerances

There are no Canadian, Mexican, or Codex Maximum Residue Level (MRLs) for metsulfuron methyl on sorghum grain.

[FR Doc. 98–7141 Filed 3–18–98; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–796; FRL–5776–6]

Notice of Filing of Pesticide Petitions

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ACTION: Notice.

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