The cancer risk and percent of the RfD that will be utilized by aggregate exposure to residues of triallate is less than 1 x $10^{-6}$ and 0.04 percent of the RfD, respectively, for all populations and subgroups including infants and children. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposures to triallate.

*G. Estrogenic Effects*

The toxicity studies required by EPA for the registration of pesticides measure numerous endpoints with sufficient sensitivity to detect potential endocrine-modulating activity. No effects have been identified in subchronic, chronic, developmental, or reproductive toxicity studies to indicate any endocrine-modulating activity by triallate. The subchronic and chronic toxicity studies examines tissues from the male and female reproductive system. The multi-generation reproduction study in rodents is a complex study design which measures a broad range of endpoints in the reproductive system and in developing offspring that are sensitive to alterations by chemical agents. Triallate only caused effects in the reproduction study at doses that were maternally toxic including an increase in mortality. Thus, these results demonstrate that triallate is not a specific reproductive toxin.

*H. Chemical Residue*

Permanent tolerances are established for triallate parent at 0.05 ppm for peas, lentils, barley and wheat, as established under 40 CFR 180.314. Triallate is metabolized in plants and animals to one major metabolite, TCPSA (2,3,3-trichloroprop-2-enesulfonic acid), and numerous natural constituents. Since the establishment of permanent tolerances for triallate, EPA has decided that TCPSA should also be regulated. Based on results of residue trials, tolerances have been proposed by Monsanto for combined residues of triallate and TCPSA in sugarbeet commodities at 0.1 ppm in sugarbeet roots, 0.5 ppm in sugarbeet tops, and 0.2 ppm in sugarbeet pulp. A practical method for determining triallate has been approved by EPA and is available from the Field Operations Division, Office of Pesticide Programs. Monsanto is in the process of developing a practical method for TCPSA. These methods include extraction followed by partitioning with methylene chloride to isolate triallate fromTCPSA. The triallate portion is eluted through a Florsil clean-up column, concentrated and quantitated by capillary GC using electron capture detection (ECD). The TCPSA portion is isolated using a phase transfer catalyst, derivatized cleaned up using SPE, and quantitated by capillary GC using ECD. Residue studies show that TCPSA is the major residue in sugarbeet foliage, but is not a significant residue in sugarbeet roots since it was not detected above the lower limit of method validation (0.01 ppm) when triallate was applied at maximum application rates. Since sugarbeet foliage seldom enters interstate commerce, EPA has informed the petitioner that enforcement of the proposed tolerances would be limited to sugarbeet roots and dried pulp. As triallate is the primary residue in sugarbeet roots and dried pulp, EPA has concluded that the currently available enforcement for parent only is adequate to enforce the tolerances on a time-limited basis.

Sugarbeet foliage is considered by EPA as an animal feed item. However, EPA has informed the petitioner that based on animal metabolism studies and animal residue studies, secondary residues are not expected to occur in meat, milk, poultry, and eggs as a result of this proposed use.

*I. Environmental Fate*

Laboratory studies indicate that triallate degrades in soil with a half-lives ranging from 18 to 21 days. Field dissipation studies show that triallate degrades with half-lives ranging from 20 to 190 days, but 190 days is clearly an outlier based on all other data. Average field half-life from all other locations is 49 days. Triallate metabolizes to $CO_2$, bound residues, and TCPSA. Triallate and TCPSA do not appear to move below a 6-inch depth.

In a laboratory study conducted with worst-case conditions, 50 percent of applied triallate volatized from agricultural sand with a very low organic content. Triallate volatility decreases from soils with higher organic content since triallate binds to organic matter in the soil. Triallate is typically soil incorporated when applied so volatization is minimized. Triallate is fairly stable to hydrolysis and photolysis.

Triallate is not likely to leach into ground water. Triallate was immobile in batch adsorption/desorption studies, and soil column and soil tlc results confirmed its low mobility. Triallate is unlikely to runoff into surface water, it would stick to the soil. If triallate did get into surface water, it would be part of the sediment and undergo microbial degradation.

[FR Doc. 97–12910 Filed 5–15–97; 8:45 am]
BILLING CODE 6560–50–F

## ENVIRONMENTAL PROTECTION AGENCY

**[PF–734; FRL–5717–7]**

### 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 food commodities.

**DATES:** Comments, identified by the docket control number PF–734, must be received on or before June 16, 1997.

**ADDRESSES:** By mail submit written comments to: Public Response and Program Resources Branch, Field Operations Divison (7505C), 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:** Joanne I. Miller, Product Manager, (PM) 23, 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. 237, CM#2 1921 Jefferson Davis

Hwy., Arlington, VA 22202, (703) 305-6224; e-mail: miller.joanne@epamail.epa.gov.

**SUPPLEMENTARY INFORMATION:** EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports grantinig 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–734] (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–734] 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: May 7, 1997.

**James Jones,**

*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.

**1. E. I. DuPONT**

*PP 4F4367*

EPA has received a pesticide petition (PP) 4F4367 pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, as amended, 21 U.S.C. Section 346a(d), by the Food Quality Protection Act of 1996 (Pub. L. 104–170, 110 Stat. 1489) from E. I. DuPont de Nemours and Co., Inc. (DuPont), Barley Mill Plaza, P.O. Box 80083, Wilmington, DE 19880–0038, proposing to amend 40 CFR 180.445 by establishing a tolerance for residues of the herbicide bensulfuron methyl, (methyl-2[[[[[(4,6-dimethoxy-pyrimidin-2-yl)amino] carbonyl]amino] sulfonyl]methyl]benzoate) in or on crayfish at 0.05 ppm. The petitioner has also proposed an amendment to the directions for use for Londax* herbicide, to permit crayfish farming in treated rice fields. 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.

An adequately validated analytical method is available for enforcement purposes.

*A. Residue Chemistry*

1. *Plant metabolism*. The qualitative nature of the residues of bensulfuron methyl in rice is adequately understood. Metabolism studies with bensulfuron methyl indicate the major metabolic pathway being oxidative *o*-dealkylation of the parent to a desmethyl metabolite. The desmethyl metabolite is cleaved at the C-N bond to form sulfonamide which quickly undergoes ring closure forming homosaccharin; the end product. Hydroxylation of the 5 position of the pyrimidine ring forms a hydroxyl metabolite which can also be cleaved to form sulfonamide. An alternative pathway is the direct cleavage of the C-N bond in the parent to sulfonamide. One side reaction may lead to the formation of a free acid metabolite. CBTS previously concluded that due to the very low level of total residue, the small percentage of the hydroxyl and free acid metabolites present, and no expressed concerns over the low levels of residue in rice plants for homosaccharin, sulfonamide, and the desmethyl metabolite, the only residue of concern in rice plants (grain and straw) was the parent herbicide, bensulfuron methyl. In consideration of PP 4F4367 CBTS has again concluded that the nature of the residue in crayfish is adequately understood and that the only residue of concern is the parent, bensulfuron methyl.

2. *Analytical method*. There is an adequately validated practical analytical method available using HPLC-UV with column and eluent switching, to measure levels of bensulfuron methyl in or on crayfish with a limit of quantitation that allows monitoring of crayfish at or above the proposed tolerance level.

3. *Magnitude of the residue*. Crayfish field trial residue data show that bensulfuron methyl residues will not exceed the proposed tolerance of 0.05 ppm on crayfish. No detectable residues at a limit of quantitation (LOQ) of 0.025 ppm were found in whole body or cooked crayfish at 1, 3, 7, 14, or 21 days after bensulfuron methyl application. In consideration of PP 4F4367 CBTS has concluded that processing data for crayfish is not required.

*B. Toxicological Profile*

1. *Acute toxicity*. Bensulfuron methyl technical has been placed in EPA 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 (highest dose tested). Bensulfuron methyl has been placed in Category IV for the remaining acute toxicity tests based on the following: A rat acute oral study with an $LD_{50}$ of >5,000 mg/kg; a rat acute inhalation study with an $LC_{50}$ of >5.0 mg/l; and primary eye and dermal irritation tests that demonstrated no significant irritation in the rabbit. A dermal sensitization test with bensulfuron methyl technical in guinea pigs demonstrated no significant effects. Based on these results, DuPont believes that bensulfuron methyl represents a minimal acute toxicity risk.

2. *Genotoxicity*. Bensulfuron methyl technical was negative (non-mutagenic) in the Ames microbial mutation assay using four strains of *Salmonella typhimurium* and in a hypoxanthine-guanine phosphoribosyl transferase gene mutation assay using Chinese hamster ovary cells. In an *in vivo* bone marrow chromosome study in which

rats were dosed with 0, 500, 1,500 or 5,000 mg/kg of bensulfuron methyl technical, no dose related toxicity or effects on mitotic index or chromosome aberrations were observed. In an *in vitro* sister chromatid exchange assay Chinese hamster ovary cells were dosed with bensulfuron methyl technical at concentrations ranging from 0.135 to 2.7 mM. A slight (1.4 fold) increase in sister chromatid exchanges was observed in the nonactivated system at the maximum concentration however, a negative response was observed in the activated system at the same concentration. In an *in vitro* assay to assess unscheduled DNA synthesis in primary rat hepatocytes, bensulfuron methyl technical was negative. Based on the weight of these data, DuPont believes that bensulfuron methyl 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, 50, 750, or 7,500 ppm of bensulfuron methyl failed to reveal any evidence suggestive of an adverse effect on reproductive potential. A reproductive NOEL was demonstrated at the highest dose tested of 7,500 ppm (309 and 405 mg/kg/day in males and females respectively). In a developmental toxicity study with bensulfuron methyl technical, pregnant rats were administered oral doses of 0, 50, 500 or 2,000 mg/kg/day on gestation days 7–16. There were no indications of compound related teratogenicity or maternal effects at any dose. Fetuses from the 200 mg/kg group exhibited signs of minimal toxicity, which included an increased incidence of minor skeletal variations. These consisted of extra ossification centers in the lumbar region and incompletely ossified sternebrae and hyoid. The fetal NOEL was 500 mg/kg/day based on these observations at the high dose. In a developmental toxicity study with bensulfuron methyl technical, pregnant rabbits were administered oral doses of 0, 30, 300 or 1,500 mg/kg/day on gestation days 7–19. Clinical signs of maternal toxicity and some decrease in fetal weight gain at the high dose defined maternal and fetotoxic NOEL's at 300 mg/kg/day. There were no dose related fetal malformations or variations. A teratogenic NOEL of 1,500 mg/kg/day was defined. Based on the weight of these data, DuPont believes that bensulfuron methyl is not a reproductive toxicant. Developmental effects observed in the absence of maternal toxicity were minimal, were only observed in the rat and had a clearly defined NOEL. This NOEL, 500 mg/kg/day, far exceeds any expected human occupational or consumer exposure.

4. *Subchronic toxicity*. In a 90–day feeding study in rats conducted with bensulfuron methyl technical at dietary levels of 0, 100, 1,500, and 7,500 ppm, the NOEL was 1,500 ppm (93 and 111 mg/kg/day, M/F) and the LEL was 7,500 ppm (474 and 567 mg/kg/day, M/F) based on increased cholesterol, slight reductions in erythrocytes among males, slightly elevated liver weights, and reduced uptake of stain in the cytoplasm of liver cells fixed for histological evaluation in both sexes. The latter was not considered to be associated with an adverse effect. In a 90–day feeding study in mice conducted with bensulfuron methyl technical at dietary levels of 0, 300, 1,000, 3,000 and 10,000 ppm, the NOEL was 1,000 ppm (132 and 133 mg/kg/day, M/F) and the LEL was 3,000 ppm (387 and 407 mg/kg/day, M/F) based on fatty deposition in the cortico-medullary junction of the adrenals in females, and centrilobular hepatocyte swelling and increased liver weights in males and females. In a 90–day feeding study in dogs conducted with bensulfuron methyl technical at dietary levels of 0, 100, 1,000, and 10,000 ppm, the NOEL was 1,000 ppm (32.1 and 36.3 mg/kg/day, M/F) and the LEL was 10,000 ppm (340 and 360 mg/kg/day, M/F) based on elevated alkaline phosphatase and alanine aminotransferase (ALT or SGPT), elevated liver weights, gross liver enlargement and discoloration, and microscopic findings of gall bladder calculus, bile stasis, centrilobular hepatocyte swelling, and vacuolation of the seminiferous tubules at the highest dose tested.

5. *Chronic toxicity/oncogenicity*. A 1–year feeding study in dogs was conducted with bensulfuron methyl technical at dietary levels of 0, 50, 750, and 7,500 ppm. Very little toxicity and no mortality were observed in this study. Gross findings suggest that bensulfuron methyl may have directly irritated the oral mucosa, especially in the high dose males and females. The major target organ was the liver as demonstrated by elevated alkaline phosphatase and SGPT (ALT), elevated liver weights, and microscopic findings of brown pigment in the biliary canaliculi of the liver at the highest dose tested. The defined systemic NOEL is 750 ppm (21.4 and 19.9 mg/kg/day, M/F) and the systemic LEL is 7,500 ppm (237.3 and 222.6 mg/kg/day, M/F). A 2–year combined chronic toxicity and oncogenicity study in mice was conducted with bensulfuron methyl technical at dietary levels of 0, 10, 150, 2,500 and 5,000 ppm. Very little toxicity was observed in this study. There were no dose-related effects on mortality, clinical signs, body weights, food consumption, or food efficiency. The systemic NOEL was 2,500 ppm (226 and 227 mg/kg/day, M/F) and the systemic LEL was 5,000 ppm (455 and 460 mg/kg/day, M/F) based on reduced water consumption; increased alkaline phosphatase, SGOT, SGPT, and total cholesterol; enlarged liver, abdominal cavity ascites, and benign nodules and masses in the liver; increased liver weights; centrilobular hepatocyte swelling, focal hepatocellular necrosis, and increased brown pigment deposition of stellate cells in the liver. There were no oncogenic effects found at the maximum dose of 5,000 ppm (455 and 460 mg/kg/day, M/F). A 2–year combined chronic toxicity and oncogenicity study in rats was conducted with bensulfuron methyl technical at dietary levels of 0, 50, 750 and 7,500 ppm. Bensulfuron methyl caused little toxicity at the doses used in this study. The systemic NOEL was 750 ppm (30 and 40 mg/kg/day, M/F) and the systemic LEL was 7,500 ppm (309 and 405 mg/kg/day, M/F) based on decreased body weight gain in females, increased BUN and creatinine in males, diffuse fatty changes in male livers, and centrilobular hepatocellular hypertrophy and centrilobular hepatocyte cytoplasmic basophilia margination in both sexes. Although effects were minimal to mild for chronic feeding/oncogenicity studies with bensulfuron methyl, these studies have been found acceptable by EPA as noted in the New Chemical Standard Toxicology Chapter for DPX-F5384 (bensulfuron methyl) -''because of the mild toxicity and lack of oncogenic response at substantial maximum doses in the chronic and subchronic studies in rats and mice. There was also a lack of an oncogenic response in structurally related chemicals.''

6. *Animal metabolism*. Disposition and metabolism of bensulfuron methyl were tested in male and female rats at oral doses of 16 an 2,000 mg/kg. Absorption of the radiolabelled test article from the gut was nearly total at both dose levels. The major elimination route was urine for the low-dose groups and feces for the high-dose groups. No measurable quantities of $CO_2$ or volatile metabolites were released from the lungs. Minute quantities of radioactivity (2.1%) were distributed to the body tissues, chiefly the gastrointestinal tract. Approximately half the administered radioactivity was eliminated by 24 hours in the low-dose groups, and 48

hours in the high dose groups. Nearly 99% was eliminated by the time of sacrifice at 96 hours. This study indicates that bensulfuron methyl has low toxicity and does not accumulate within the body. The major compound eliminated in urine and feces was ODS DPX-F5384 (desmethyl metabolite), formed by demethylation of the pyrimidine ring. The parent compound was found in feces but not in urine.

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

8. *Endocrine effects.* No special studies investigating potential estrogenic or other endocrine effects of bensulfuron 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 bensulfuron methyl 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 these tolerances, an estimate of aggregate exposure is made using the tolerance on rice grain at 0.02 ppm and crayfish at 0.05 ppm. The potential exposure is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of rice, rice products and crayfish eaten by various population subgroups. Rice straw is fed to animals, thus exposure of humans to residues of rice straw might result if such residues are transferred to meat, milk, poultry, or eggs. However, based on the results of livestock metabolism studies in which no quantifiable residues were reported when feeding levels were approximately 500X the potential dietary burden from feeding bensulfuron methyl treated rice straw, the EPA has concluded that there is no reasonable expectation that measurable residues of bensulfuron methyl will occur in meat or milk. Rice straw is not a poultry feed item, thus no residues are expected in poultry or eggs. In consideration of pesticide petition 4F4367 CBTS has concluded that crayfish do not constitute a significant livestock feed item, and that no additional secondary residues in animal commodities are anticipated from the proposed use. There are no other established tolerances or registered uses for bensulfuron methyl in the United States. Based on a NOEL of 750 ppm (21.4 and 19.9 mg/kg/day, M/F) from the chronic dog toxicity study and a 100–fold safety factor, the reference dose (RfD) is 0.20 mg/kg/day. Assuming residues at tolerance levels and that 100% of the crop is being treated, a theoretical maximum residue contribution (TMRC) of <0.00001 mg/kg/day is estimated. With the above assumptions which clearly overestimate potential human exposure and are a most conservative assessment of risk, dietary (food) exposure to bensulfuron methyl will utilize <0.01% of the RfD.

2. *Dietary exposure*—(ii) *drinking water.* Other potential dietary sources of exposure of the general population to residues of pesticides are residues in drinking water. There is no Maximum Contaminant Level established for residues of bensulfuron methyl. The petitioner has been advised by the EPA that all environmental fate data requirements for bensulfuron methyl have been satisfied and based on these studies and the conditions of use, the potential for finding significant bensulfuron methyl residues in water, with the exception of flooded rice fields, is minimal. However, for purposes of assessing a potential dietary exposure from water an estimated exposure may be made using information from a prior Experimental Use Permit (EUP) which has since been withdrawn without prejudice. Under this EUP bensulfuron methyl was evaluated as an aquatic vegetation management herbicide applied directly to water at a rate identical to it's current registered use in rice. With this prior EUP, a temporary tolerance for bensulfuron methyl residues in potable water of 0.1 ppm was established. Assuming this extreme case scenario with residues at this tolerance level and using a consumption figure of 2 liters per day of drinking water (consistent with the National Primary Drinking Water Regulations —Synthetic Organic and Inorganic Chemicals, (56 FR 3526, January 30, 1991)), a theoretical maximum residue contribution (TMRC) of <0.000004 mg/kg/day was calculated (calculated and reported by the California Department of Food and Agriculture, Division of Pest Management, April, 1989). With the above assumptions which would now reflect an off-label use of bensulfuron methyl, and therefore clearly overestimate potential human exposure, dietary (drinking water) exposure to bensulfuron methyl would still only utilize <0.01% of the RfD.

3. *Non-dietary exposure.* Bensulfuron methyl is not registered for any use which could result in non-occupational, non-dietary exposure to the general population.

*D. Cumulative Effects*

Bensulfuron methyl belongs to the sulfonylurea class of compounds. Other compounds in this class are registered herbicides. However, the herbicidal activity of the sulfonylureas 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. There is no evidence to indicate or suggest that bensulfuron methyl has any toxic effects on mammals that would be cumulative with those of any other chemical.

*E. Safety Determination*

1. *U S population in general.* Based on a complete and reliable toxicity database, the EPA has adopted an RfD value of 0.20 mg/kg/day using the NOEL of 750 ppm (21.4 and 19.9 mg/kg/day, M/F) from the chronic dog toxicity study and a hundredfold safety factor. Using crop tolerance levels, assuming 100% of the crop being treated, a drinking water estimate which is clearly an overestimate based on off-label use, and a complete battery of toxicity data, it is concluded that aggregate exposure to bensulfuron methyl will utilize significantly less than 0.1% of the RfD for either the entire U.S. population or any of the population subgroups for which consumption data is available, including infants and children. 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, DuPont believes that there is a reasonable certainty that no harm will result from aggregate exposure to bensulfuron methyl residues.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of bensulfuron methyl, 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, DuPont believes that bensulfuron methyl is not a reproductive toxicant. Developmental effects observed in the absence of maternal toxicity were minimal, and were only observed in the rat and at a dose that far exceeds any expected human exposure. 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 bensulfuron methyl relative to pre-and post-natal effects for children is complete. Further, as the NOEL of 20 mg/kg/day from the 1–year dog study with bensulfuron methyl which was used to calculate the RfD (discussed above), is already lower than any of the NOEL's defined in the developmental and reproductive toxicity studies with bensulfuron methyl, an additional safety factor is not warranted. As stated above, aggregate exposure assessments utilized significantly less than 0.1% of the RfD for either the entire U.S. population or any of the population subgroups for which consumption data was available, including infants and children. Therefore, DuPont believes that it may be concluded that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to bensulfuron methyl residues.

*F. International Tolerances*

There are no Canadian, Mexican, or Codex MRLs/ tolerances for bensulfuron methyl on rice straw. Compatibility is not a problem at this time.

**2. E. I. DuPONT**

*PP 5F4490*

EPA has received a pesticide petition (PP) 5F4490 pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, as amended, 21 U.S.C. Section 346a(d), by the Food Quality Protection Act of 1996 (Pub. L. 104-170, 110 Stat. 1489) from E. I. DuPont de Nemours and Co., Inc. (DuPont), Barley Mill Plaza, P.O. Box 80083, Wilmington, DE 19880–0038, proposing to amend 40 CFR 180.445 by amending the existing tolerance for residues of the herbicide bensulfuron methyl (methyl-2[[[[[(4,6-dimethoxy- pyrimidin-2-yl)amino]carbonyl] amino]sulfonyl]methyl]benzoate) in or on the raw agricultural commodity rice straw from 0.05 ppm to 0.3 ppm. The petitioner has also proposed an amendment to the directions for use for Londax* herbicide, to reduce the herbicides application pre-harvest interval from 80 to 60 days. 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.

An adequately validated analytical method is available for enforcement purposes.

*A. Residue Chemistry*

1. *Plant metabolism*. The qualitative nature of the residues of bensulfuron methyl in rice is adequately understood. Metabolism studies with bensulfuron methyl indicate the major metabolic pathway being oxidative *o*-dealkylation of the parent to a desmethyl metabolite. The desmethyl metabolite is cleaved at the C-N bond to form sulfonamide which quickly undergoes ring closure forming homosaccharin; the end product. Hydroxylation of the 5 position of the pyrimidine ring forms a hydroxyl metabolite which can also be cleaved to form sulfonamide. An alternative pathway is the direct cleavage of the C-N bond in the parent to sulfonamide. One side reaction may lead to the formation of a free acid metabolite. CBTS previously concluded that due to the very low level of total residue, the small percentage of the hydroxyl and free acid metabolites present, and no expressed concerns over the low levels of residue in rice plants for homosaccharin, sulfonamide, and the desmethyl metabolite, the only residue of concern in rice plants (grain and straw) was the parent herbicide, bensulfuron methyl. In consideration of PP 5F4490 CBTS has again concluded that the only residue of concern is the parent, bensulfuron methyl.

2. *Analytical method*. There is an adequately validated practical analytical method available using HPLC-UV with column and eluent switching, to measure levels of bensulfuron methyl in or on rice with a limit of quantitation that allows monitoring of rice grain and straw at or above tolerance levels. EPA has provided information on this method to the Food and Drug Administration for future publication in PAM II.

3. *Magnitude of the residue*. Crop field trial residue data from a 60–day PHI study shows that the established bensulfuron methyl tolerance on rice grain of 0.02 ppm will not be exceeded when Londax* is used as directed, and the tolerance need not be changed. An adequate amount of geographically representative crop field trial residue data support the amended registration request and show that with the 60–day PHI, bensulfuron methyl residues will not exceed the proposed tolerance of 0.3 ppm on rice straw. An adequate bensulfuron methyl rice processing study using rice bearing detectable residues following an exaggerated 5X application shows that bensulfuron methyl does not concentrate in rice bran, hulls, and polished rice; thus no tolerances on these commodities are required.

*B. Toxicological Profile*

1. *Acute toxicity*. Bensulfuron methyl technical has been placed in EPA 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 (highest dose tested). Bensulfuron methyl has been placed in Category IV for the remaining acute toxicity tests based on the following: a rat acute oral study with an $LD_{50}$ of > 5,000 mg/kg; a rat acute inhalation study with an $LC_{50}$ of > 5.0 mg/l; and primary eye and dermal irritation tests that demonstrated no significant irritation in the rabbit. A dermal sensitization test with bensulfuron methyl technical in guinea pigs demonstrated no significant effects. Based on these results, DuPont believes that bensulfuron methyl represents a minimal acute toxicity risk.

2. *Genotoxicity*. Bensulfuron methyl technical was negative (non-mutagenic) in the Ames microbial mutation assay using four strains of *Salmonella typhimurium* and in a hypoxanthine-guanine phosphoribosyl transferase gene mutation assay using Chinese hamster ovary cells. In an *in vivo* bone marrow chromosome study in which rats were dosed with 0, 500, 1,500 or 5,000 mg/kg of bensulfuron methyl technical, no dose related toxicity or effects on mitotic index or chromosome aberrations were observed. In an *in vitro* sister chromatid exchange assay Chinese hamster ovary cells were dosed with bensulfuron methyl technical at concentrations ranging from 0.135 to 2.7 mM. A slight (1.4 fold) increase in sister chromatid exchanges was observed in the nonactivated system at the maximum concentration; however, a negative response was observed in the activated system at the same concentration. In an *in vitro* assay to assess unscheduled DNA synthesis in primary rat hepatocytes, bensulfuron methyl technical was negative. Based on the weight of these data, DuPont

believes that bensulfuron methyl 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, 50, 750, or 7,500 ppm of bensulfuron methyl failed to reveal any evidence suggestive of an adverse effect on reproductive potential. A reproductive NOEL was demonstrated at the highest dose tested of 7,500 ppm (309 and 405 mg/kg/day in males and females respectively). In a developmental toxicity study with bensulfuron methyl technical, pregnant rats were administered oral doses of 0, 50, 500 or 2,000 mg/kg/day on gestation days 7–16. There were no indications of compound related teratogenicity or maternal effects at any dose. Fetuses from the 200 mg/kg group exhibited signs of minimal toxicity, which included an increased incidence of minor skeletal variations. These consisted of extra ossification centers in the lumbar region and incompletely ossified sternebrae and hyoid. The fetal NOEL was 500 mg/kg/day based on these observations at the high dose. In a developmental toxicity study with bensulfuron methyl technical, pregnant rabbits were administered oral doses of 0, 30, 300 or 1,500 mg/kg/day on gestation days 7–19. Clinical signs of maternal toxicity and some decrease in fetal weight gain at the high dose defined maternal and fetotoxic NOEL's at 300 mg/kg/day. There were no dose related fetal malformations or variations. A teratogenic NOEL of 1,500 mg/kg/day was defined. Based on the weight of these data, DuPont believes that bensulfuron methyl was not a reproductive toxicant. Developmental effects observed in the absence of maternal toxicity were minimal, were only observed in the rat and had a clearly defined NOEL. This NOEL, 500 mg/kg/day, far exceeds any expected human occupational or consumer exposure.

4. *Subchronic toxicity.* In a 90–day feeding study in rats conducted with bensulfuron methyl technical at dietary levels of 0, 100, 1,500, and 7,500 ppm, the NOEL was 1,500 ppm (93 and 111 mg/kg/day, M/F) and the LEL was 7,500 ppm (474 and 567 mg/kg/day, M/F) based on increased cholesterol, slight reductions in erythrocytes among males, slightly elevated liver weights, and reduced uptake of stain in the cytoplasm of liver cells fixed for histological evaluation in both sexes. The latter was not considered to be associated with an adverse effect. In a 90–day feeding study in mice conducted with bensulfuron methyl technical at dietary levels of 0, 300, 1,000, 3,000 and 10,000 ppm, the NOEL was 1,000 ppm (132 and 133 mg/kg/day, M/F) and the LEL was 3,000 ppm (387 and 407 mg/kg/day, M/F) based on fatty deposition in the cortico-medullary junction of the adrenals in females, and centrilobular hepatocyte swelling and increased liver weights in males and females. In a 90–day feeding study in dogs conducted with bensulfuron methyl technical at dietary levels of 0, 100, 1,000, and 10,000 ppm, the NOEL was 1,000 ppm (32.1 and 36.3 mg/kg/day, M/F) and the LEL was 10,000 ppm (340 and 360 mg/kg/day, M/F) based on elevated alkaline phosphatase and alanine aminotransferase (ALT or SGPT), elevated liver weights, gross liver enlargement and discoloration, and microscopic findings of gall bladder calculus, bile stasis, centrilobular hepatocyte swelling, and vacuolation of the seminiferous tubules at the highest dose tested.

5. *Chronic toxicity/oncogenicity.* A 1–year feeding study in dogs was conducted with bensulfuron methyl technical at dietary levels of 0, 50, 750, and 7,500 ppm. Very little toxicity and no mortality were observed in this study. Gross findings suggest that bensulfuron methyl may have directly irritated the oral mucosa, especially in the high dose males and females. The major target organ was the liver as demonstrated by elevated alkaline phosphatase and SGPT (ALT), elevated liver weights, and microscopic findings of brown pigment in the biliary canaliculi of the liver at the highest dose tested. The defined systemic NOEL is 750 ppm (21.4 and 19.9 mg/kg/day, M/F) and the systemic LEL is 7,500 ppm (237.3 and 222.6 mg/kg/day, M/F). A 2–year combined chronic toxicity and oncogenicity study in mice was conducted with bensulfuron methyl technical at dietary levels of 0, 10, 150, 2,500 and 5,000 ppm. Very little toxicity was observed in this study. There were no dose-related effects on mortality, clinical signs, body weights, food consumption, or food efficiency. The systemic NOEL was 2,500 ppm (226 and 227 mg/kg/day, M/F) and the systemic LEL was 5,000 ppm (455 and 460 mg/kg/day, M/F) based on reduced water consumption; increased alkaline phosphatase, SGOT, SGPT, and total cholesterol; enlarged liver, abdominal cavity ascites, and benign nodules and masses in the liver; increased liver weights; centrilobular hepatocyte swelling, focal hepatocellular necrosis, and increased brown pigment deposition of stellate cells in the liver. There were no oncogenic effects found at the maximum dose of 5,000 ppm (455 and 460 mg/kg/day, M/F). A 2–year combined chronic toxicity and oncogenicity study in rats was conducted with bensulfuron methyl technical at dietary levels of 0, 50, 750 and 7,500 ppm. Bensulfuron methyl caused little toxicity at the doses used in this study. The systemic NOEL was 750 ppm (30 and 40 mg/kg/day, M/F) and the systemic LEL was 7,500 ppm (309 and 405 mg/kg/day, M/F) based on decreased body weight gain in females, increased BUN and creatinine in males, diffuse fatty changes in male livers, and centrilobular hepatocellular hypertrophy and centrilobular hepatocyte cytoplasmic basophilia margination in both sexes. Although effects were minimal to mild for chronic feeding/oncogenicity studies with bensulfuron methyl, these studies have been found acceptable by EPA as noted in the New Chemical Standard Toxicology Chapter for DPX-F5384 (bensulfuron methyl) - ''because of the mild toxicity and lack of oncogenic response at substantial maximum doses in the chronic and subchronic studies in rats and mice. There was also a lack of an oncogenic response in structurally related chemicals.''

6. *Animal metabolism.* Disposition and metabolism of bensulfuron methyl were tested in male and female rats at oral doses of 16 an 2,000 mg/kg. Absorption of the radiolabelled test article from the gut was nearly total at both dose levels. The major elimination route was urine for the low- dose groups and feces for the high-dose groups. No measurable quantities of $CO_2$ or volatile metabolites were released from the lungs. Minute quantities of radioactivity (2.1%) were distributed to the body tissues, chiefly the gastrointestinal tract. Approximately half the administered radioactivity was eliminated by 24 hours in the low-dose groups, and 48 hours in the high dose groups. Nearly 99% was eliminated by the time of sacrifice at 96 hours. This study indicates that bensulfuron methyl has low toxicity and does not accumulate within the body. The major compound eliminated in urine and feces was ODS DPX-F5384 (desmethyl metabolite), formed by demethylation of the pyrimidine ring. The parent compound was found in feces but not in urine.

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

8. *Endocrine effects.* No special studies investigating potential estrogenic or other endocrine effects of bensulfuron 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 bensulfuron methyl 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 these tolerances, an estimate of aggregate exposure is made using the tolerance on rice grain at 0.02 ppm. The potential exposure is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of rice or rice products eaten by various population subgroups. Rice straw is fed to animals, thus exposure of humans to residues of rice straw might result if such residues are transferred to meat, milk, poultry, or eggs. However, based on the results of livestock metabolism studies in which no quantifiable residues were reported when feeding levels were approximately 500X the potential dietary burden from feeding bensulfuron methyl treated rice straw, the EPA has concluded that there is no reasonable expectation that measurable residues of bensulfuron methyl will occur in meat or milk. Rice straw is not a poultry feed item, thus no residues are expected in poultry or eggs. There are no other established tolerances or registered uses for bensulfuron methyl in the United States. Based on a NOEL of 750 ppm (21.4 and 19.9 mg/kg/day, M/F) from the chronic dog toxicity study and a hundredfold safety factor, the reference dose (RfD) is 0.20 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 bensulfuron methyl will utilize <0.01% of the RfD.

2. *Dietary exposure*--(ii) *drinking water*. Other potential dietary sources of exposure of the general population to residues of pesticides are residues in drinking water. There is no Maximum Contaminant Level established for residues of bensulfuron methyl. The petitioner has been advised by the EPA that all environmental fate data requirements for bensulfuron methyl have been satisfied and based on these studies and the conditions of use, the potential for finding significant bensulfuron methyl residues in water, with the exception of flooded rice fields, is minimal. However, for purposes of assessing a potential dietary exposure from water an estimated exposure may be made using information from a prior Experimental Use Permit (EUP) which has since been withdrawn without prejudice. Under this EUP bensulfuron methyl was evaluated as an aquatic vegetation management herbicide applied directly to water at a rate identical to it's current registered use in rice. With this prior EUP, a temporary tolerance for bensulfuron methyl residues in potable water of 0.1 ppm was established. Assuming this extreme case scenario with residues at this tolerance level and using a consumption figure of 2 liters per day of drinking water (consistent with the National Primary Drinking Water Regulations -- Synthetic Organic and Inorganic Chemicals, (56 FR 3526, January 30, 1991)), a theoretical maximum residue contribution (TMRC) of <0.000004 mg/kg/day was calculated (calculated and reported by the California Department of Food and Agriculture, Division of Pest Management, April, 1989). With the above assumptions which would now reflect an off-label use of bensulfuron methyl, and therefore clearly overestimate potential human exposure, dietary (drinking water) exposure to bensulfuron methyl would still only utilize <0.01% of the RfD.

3. *Non-dietary exposure*. Bensulfuron methyl is not registered for any use which could result in non-occupational, non-dietary exposure to the general population.

*D. Cumulative Effects*

Bensulfuron methyl belongs to the sulfonylurea class of compounds. Other compounds in this class are registered herbicides. However, the herbicidal activity of the sulfonylureas 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. There is no evidence to indicate or suggest that bensulfuron methyl has any toxic effects on mammals that would be cumulative with those of any other chemical.

*E. Safety Determination*

1. *U.S. population in general*. Based on a complete and reliable toxicity database, the EPA has adopted an RfD value of 0.20 mg/kg/day using the NOEL of 750 ppm (21.4 and 19.9 mg/kg/day, M/F) from the chronic dog toxicity study and a hundredfold safety factor. Using crop tolerance levels, assuming 100% of the crop being treated, a drinking water estimate which is clearly an overestimate based on off-label use, and a complete battery of toxicity data, it is concluded that aggregate exposure to bensulfuron methyl will utilize significantly less than 0.1% of the RfD for either the entire U.S. population or any of the population subgroups for which consumption data is available, including infants and children. 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, DuPont believes that there is a reasonable certainty that no harm will result from aggregate exposure to bensulfuron methyl residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of bensulfuron methyl, 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, DuPont believes that bensulfuron methyl is not a reproductive toxicant. Developmental effects observed in the absence of maternal toxicity were minimal, and were only observed in the rat and at a dose that far exceeds any expected human exposure. 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 bensulfuron methyl relative to pre- and post-natal effects for children is complete. Further, as the NOEL of 20 mg/kg/day from the 1–year dog study with bensulfuron methyl which was used to calculate the RfD (discussed above), is already lower than any of the NOEL's defined in the developmental and reproductive toxicity studies with bensulfuron methyl, an additional safety factor is not warranted. As stated above,

aggregate exposure assessments utilized significantly less than 0.1% of the RfD for either the entire U.S. population or any of the population subgroups for which consumption data was available, including infants and children. Therefore, DuPont believes that it may be concluded that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to bensulfuron methyl residues.

*F. International Tolerances*

There are no Canadian, Mexican, or Codex MRLs/ tolerances for bensulfuron methyl on rice straw. Compatibility is not a problem at this time.

[FR Doc. 97–12907 Filed 5–15–97; 8:45 am]
**BILLING CODE 6560–50–F**

## ENVIRONMENTAL PROTECTION AGENCY

**[PF–732; FRL–5717–4]**

### 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–732, must be received on or before June 16, 1997.

**ADDRESSES:** By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources 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:** By mail: Joanne Miller, PM 23, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 237, CM #2, 1921 Jefferson Davis Hwy, Arlington, VA 22202, 703–305–6224, e-mail: miller.joanne@epamail.epa.gov.

**SUPPLEMENTARY INFORMATION:** EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–732] (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–732] 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: May 7, 1997.

**James Jones,**

*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.

### FMC Corporation

*PP 6G4615*

EPA has received a pesticide petition (PP 6G4615) from FMC Corporation, 1735 Market St., Philadelphia, PA 19103, 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 establishing a temporary tolerance for the combined residue of the herbicide carfentrazone-ethyl (ethyl-α-2-dichloro-5-[4-(difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzene-propanoate) and its major wheat metabolites: carfentrazone-ethyl chloropropionic acid (α, 2-dichloro-5-[4-difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzenepropanoic acid), 3-hydroxymethyl-F8426-chloropropionic acid (α, 2-dichloro-5-[4-difluoromethyl)-4,5-dihydro-3-hydroxymethyl-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzenepropanoic acid) and 3-desmethyl- F8426 chloropropionic acid (α, 2-dichloro-5-[4-difluoromethyl)-4,5-dihydro-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzenepropanoic acid) in or on wheat raw agricultural commodities: 0.2 ppm in or on wheat hay, 0.2 ppm in or on wheat straw, 0.2 ppm in or on wheat grain; and establishing tolerance for combined residues of the herbicide carfentrazone-ethyl (ethyl-α-2-dichloro-5-[4-(difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzene-propanoate) and its two major corn metabolites: carfentrazone-