mammals and that there is also no evidence to suggest that FWAs exhibit estrogenic properties.

C. Aggregate Exposure

The petitioner believes that 2,2′-(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate and stilbene FWAs in general are extensively used as optical brighteners both domestically and internationally. Approximately 30 million pounds are used annually in the United States alone with the majority being used in detergents to enhance the color of laundered clothing. Other uses of stilbenes include incorporation into textiles, paper, paint, and plastics (some of which are used in the food industry). In comparison, the proposed use of 2,2′-(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate as an inert ingredient in pesticide formulations is not expected to exceed 250,000 pounds annually (< 1% of the total FWA use in the United States).

The petitioner believes that potential routes of non-occupational exposure to FWAs currently include non-dietary (i.e., potential dermal exposure via contact with laundered clothing) and dietary sources (i.e., potential consumption in drinking water, in fish, and as residue of detergents adhering to dishes and cutlery) and consequently, the proposed inert ingredient use of 2,2′(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate is not expected to significantly increase the aggregate exposure to FWAs.

D. Cumulative Effects

The petitioner believes that data show that the diaminostilbene-disulfonic acid class of FWAs is relatively non-toxic to mammals and, in addition, the proposed use of 2,2′-(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate, a member of this class of chemistry, will not significantly increase the US population's exposure to FWAs. Thus, the petitioner believes that there is no expectation of significant incremental risk due to the use of 2,2′-(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate as an inert ingredient in pesticide formulations.

E. Safety Determination

The petitioner considered that toxicology studies conducted with 2,2′-(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate and other compounds in the stilbene class of chemistry show there is reasonable certainty that no harm to the U.S. population will result from aggregate exposure to FWA residue including all anticipated dietary exposures and all other non-occupational exposures for which there is reliable information. Experimental investigations show that the likelihood of FWAs constituting a danger to human health is so minimal as to be completely negligible.

The petitioner notes that there is no information available to indicate that children or infants would be more sensitive than adults to any toxic effect associated with exposure to 2,2′-(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate.

F. International Tolerances

There are no Codex maximum residue levels established for residues of 2,2′-(1,2-ethenediyl)bis[5-[[4-[bis(2-hydroxyethyl)amino]-6-phenylamino]-1,3,5-triazin-2-yl]amino]-benzenesulfonate on food or feed crops.

[FR Doc. 97–12914 Filed 5–15–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–733; FRL–5717–6]

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–733, 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: Jim Tompkins, Acting 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. 229, CM #2, 1921 Jefferson Davis Highway, Arlington, VA. 22202, (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 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–733] (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–733] 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 8, 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. BASF Corporation

PP 9F3804

BASF has submitted a pesticide petition (PP 9F3804) proposing tolerances for residues of the pesticide, sethoxydim, [2-(1-(ethoxyimino)butyl-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one] and its metabolites containing the 2-cyclohexen-1-one moiety (calculated as the herbicide) in or on the raw agricultural commodities, apricots, cherries (sweet and sour), nectarines, and peaches, at 0.2 parts per million (ppm).

A. Residue Chemistry

1. *Plant and animal metabolism*. The qualitative nature of the residues in plants and animals is adequately understood for the purposes of registration. Metabolic pathways in apricots, cherries (sweet and sour), nectarines, and peaches are similar. Analytical methods for detecting levels of sethoxydim and its metabolites in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances was submitted to EPA.

2. *Analytical method*. The proposed analytical method involves extraction, partition, and clean-up. Samples are then analyzed by gas chromatography with sulfur-specific flame photometric detection. The limit of quantitation is 0.05 ppm.

3. *Magnitude of the residues*. Peach samples from eleven trials in six states (CA, GA, SC, NJ, WA, WV) were analyzed for residues of sethoxydim and its metabolites. In none of the trials did the total residue in treated samples exceed 0.10 ppm of sethoxydim equivalents. Preharvest intervals (PHIs) ranged from 10 to 89 days with most samples harvested at a 10 to 20 day PHI. The treatment program included multiple applications at rates varying from 0.5 to 2.0 lb active ingredient (a.i.)/acre. Most samples received three applications of 0.5 lb a.i./acre. BASF is proposing a tolerance of 0.2 ppm to account for loss of residue during the first 30 days of frozen storage.

Sour cherry samples from six trials in five states (MI, PA, OR, UT, WI) and sweet cherry samples from six trials in four states (WA, OR, MI, CA) were analyzed for residues of sethoxydim and its metabolites. In only one of the trials did the total residue in treated samples exceed 0.10 ppm of sethoxydim equivalents. The maximum residue found in this sample was only 0.13 ppm. PHIs ranged from 7 to 17 days with the exception of one sweet cherry sample which had a PHI of 43 days. The treatment program included multiple applications at rates varying from 0.3 or 0.5 lb a.i./acre. Most samples received two applications of 0.5 lb a.i./acre. BASF is proposing a tolerance of 0.2 ppm to account for loss of residue during the first 30 days of frozen storage.

One apricot sample and one nectarine sample from separate trials in California were analyzed for residues of sethoxydim and its metabolites. The apricot sample showed a total residue of less than 0.10 ppm of sethoxydim equivalents. The nectarine sample contained a total of 0.11 ppm of sethoxydim equivalents. The PHI was 17 days for the apricot sample and 21 days for the nectarine sample. The treatment program was two applications of 0.5 lb a.i./acre. BASF is proposing a tolerance of 0.2 ppm to account for loss of residue during the first 30 days of frozen storage.

B. Toxicological Profile

1. *Acute toxicity testing.* Based on the available acute toxicity data, sethoxydim does not pose any acute dietary risks. A summary of the acute toxicity studies follows.

i. Acute oral toxicity, rat: Toxicity Category III; LD_{50}=3,125 mg/kg (male), 2,676 mg/kg (female).

ii. Acute dermal toxicity, rat: Toxicity Category III; LD_{50}>5,000 mg/kg (male and female).

iii. Acute inhalation toxicity, rat: Toxicity Category III; LC_{50} (4-hour)=6.03 mg/L (male), 6.28 mg/L (female).

iv. Primary eye irritation, rabbit: Toxicity Category IV; no irritation.

v. Primary dermal irritation, rabbit: Toxicity Category IV; no irritation.

vi. Dermal sensitization, guinea pig: Waived because no sensitization was seen in guinea pigs dosed with the end-use product Poast (18 percent a.i.).

2. *Subchronic toxicity testing.* A summary of the subchronic toxicity data follows.

A 21–day dermal study in rabbits with a no-observed-adverse-effect-level (NOAEL) of >1,000 mg/kg/day (limit dose). The only dose-related finding was slight epidermal hyperplasia at the dosing site in nearly all males and females dosed at 1,000 mg/kg/day. This was probably an adaptive response.

3. *Chronic toxicity testing.* A summary of the chronic toxicity studies follows.

i. A 1–year feeding study with dogs fed diets containing 0, 8.86/9.41, 17.5/19.9, and 110/129 milligrams (mg)/kilogram (kg)/day (males/females) with a no-observed-effect-level (NOEL) of 8.86/9.41 mg/kg/day (males/females) based on equivocal anemia in male dogs at the 17.5-mg/kg/day dose level.

ii. A 2–year chronic feeding/carcinogenicity study with mice fed diets containing 0, 40, 120, 360, and 1,080 ppm (equivalent to 0, 6, 18, 54, and 162 mg/kg/day) with a systemic NOEL of 120 ppm (18 mg/kg/day) based on non-neoplastic liver lesions in male mice at the 360-ppm (54 mg/kg/day) dose level. There were no carcinogenic effects observed under the conditions of the study. The maximum tolerated dose (MTD) was not achieved in female mice.

iii. A 2–year chronic feeding/carcinogenic study with rats fed diets containing 0, 2, 6, and 18 mg/kg/day with a systemic NOEL greater than or equal to 18 mg/kg/day (highest dose tested). There were no carcinogenic effects observed under the conditions of the study. This study was reviewed under current guidelines and was found to be unacceptable because the doses

used were insufficient to induce a toxic response and an MTD was not achieved.

iv. A second chronic feeding/carcinogenic study with rats fed diets containing 0, 360, and 1,080 ppm (equivalent to 18.2/23.0, and 55.9/71.8 mg/kg/day (males/females). The dose levels were too low to elicit a toxic response in the test animals and failed to achieve an MTD or define a lowest effect level (LEL). Slight decreases in body weight in rats at the 1,080-ppm dose level, although not biologically significant, support a free-standing no-observed-adverse-effect-level (NOAEL) of 1,080 ppm (55.9/71.8 mg/kg/day (males/females)). There were no carcinogenic effects observed under the conditions of the study.

v. In a rat metabolism study, excretion was extremely rapid and tissue accumulation was negligible.

4. *Developmental toxicity testing.* A developmental toxicity study in rats fed dosages of 0, 50, 180, 650, and 1,000 mg/kg/day with a maternal NOAEL of 180 mg/kg/day and a maternal LEL of 650 mg/kg/day (irregular gait, decreased activity, excessive salivation, and anogenital staining); and a developmental NOAEL of 180 mg/kg/day, and a developmental LEL of 650 mg/kg/day (21 to 22 percent decrease in fetal weights, filamentous tail, and lack of tail due to the absence of sacral and/or caudal vertebrae, and delayed ossification in the hyoids, vertebral centrum and/or transverse processes, sternebrae and/or metatarsals, and pubes).

A developmental toxicity study in rabbits fed doses of 0, 80, 160, 320, and 400 mg/kg/day with a maternal NOEL of 320 mg/kg/day and a maternal LOEL of 400 mg/kg/day (37 percent reduction in body weight gain without significant differences in group mean body weights and decreased food consumption during dosing); and a developmental NOEL greater than 400 mg/kg/day (highest dose tested).

5. *Reproductive toxicity testing.* A 2–generation reproduction study with rats fed diets containing 0, 150, 600, and 3,000 ppm (approximately 0, 7.5, 30, and 150 mg/kg/day) with no reproductive effects observed under the conditions of the study.

6. *Mutagenicity testing.* Ames assays were negative for gene mutation in Salmonella typhimurium strains TA98, TA100, TA1535, and TA 1537, with and without metabolic activity.

A Chinese hamster bone marrow cytogenetic assay was negative for structural chromosomal aberrations at doses up to 5,000 mg/kg in Chinese hamster bone marrow cells *in vivo*.

Recombinant assays and forward mutations tests in *Bacillus subtilis*, *Escherichia coli*, and *S. typhimurium* were all negative for genotoxic effects at concentrations of greater than or equal to 100 percent.

C. Threshold Effects

Based on the available chronic toxicity data, EPA has established the Reference Dose (RfD) for sethoxydim at 0.09 mg/kg bw/day. The RfD for sethoxydim is based on a 1–year feeding study in dogs with a threshold NOEL of 8.86 mg/kg/day and an uncertainty factor of 100.

D. Non-Threshold Effects

A repeat chronic feeding/carcinogenicity study in rats was submitted to EPA in November of 1995 and is awaiting review. The Agency will reassess sethoxydim tolerances based on the outcome of the rat chronic feeding/carcinogenicity study. In the interim, there is little risk from establishment of the proposed tolerances since available studies in rats and mice indicate no carcinogenic effects, there are adequate data to establish a RfD, existing tolerances and the proposed tolerances do not exceed the RfD, and the proposed tolerances utilize less than 1 percent of the RfD. Thus, a cancer risk assessment is not necessary.

E. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure, BASF has estimated aggregate exposure based on the Theoretical Maximum Residue Contribution (TMRC) from the tolerances of sethoxydim on: apricots at 0.2 ppm, cherries at 0.2 ppm, nectarines at 0.2 ppm, and peaches at 0.2 ppm. (The TMRC is a ''worst case'' estimate of dietary exposure since it is assumed that 100 percent of all crops for which tolerances are established are treated and that pesticide residues are at the tolerance levels.) The TMRC from existing tolerances for the overall US population is estimated at approximately 37 percent of the RfD. Dietary exposure to residues of sethoxydim in or on food from these proposed tolerances increases the TMRC by less than 1 percent of the RfD for the overall US population. BASF estimates indicate that dietary exposure will not exceed the RfD for any population subgroup for which EPA has data [ref. Proposed Rule at 60 FR 13941 March 15, 1995]. This exposure assessment relies on very conservative assumptions-100 percent of crops will contain sethoxydim residues and those residues would be at the level of the tolerance-which results in an overestimate of human exposure.

2. *''Other'' exposure*. Other potential sources of exposure of the general population to residues of pesticides are residues in drinking water and exposure from non-occupational sources. Based on the available studies submitted to EPA for assessment of environmental risk, BASF does not anticipate exposure to residues of sethoxydim in drinking water. There is no established Maximum Concentration Level (MCL) for residues of sethoxydim in drinking water under the Safe Drinking Water Act (SDWA).

BASF has not estimated non-occupational exposure for sethoxydim. Sethoxydim is labeled for use by homeowners on and around the following use sites: flowers, evergreens, shrubs, trees, fruits, vegetables, ornamental groundcovers, and bedding plants. Hence, the potential for non-occupational exposure to the general population exists. However, these use sites do not appreciably increase exposure. Protective clothing requirements, including the use of gloves, adequately protect homeowners when applying the product. The product may only be applied through hose-end sprayers or tank sprayers as a 0.14 percent solution. Sethoxydim is not a volatile compound so inhalation exposure during and after application would be negligible. Dermal exposure would be minimal in light of the protective clothing and the low application rate. Post-treatment (re-entry) exposure would be negligible for these use sites as contact with treated surfaces would be low. Dietary risks from treated food crops are already adequately regulated by the established tolerances. The additional usesapricots, cherries, nectarines, and peacheswill not increase the non-occupational exposure appreciably, if at all. The potential for non-occupational exposure to the general population is, thus, insignificant.

F. Cumulative Exposure

BASF also considered the potential for cumulative effects of sethoxydim and other substances that have a common mechanism of toxicity. BASF is aware of one other active ingredient which is structurally similar, clethodim. However BASF believes that consideration of a common mechanism of toxicity is not appropriate at this time. BASF does not have any reliable information to indicate that toxic effects produced by sethoxydim would be cumulative with clethodim or any other chemical; thus BASF is considering

only the potential risks of sethoxydim in its exposure assessment.

G. Safety Determination

1. *U.S. population*. Reference Dose (RfD), using the conservative exposure assumptions described above, BASF has estimated that aggregate exposure to sethoxydim will utilize <38 percent of the RfD for the US population. EPA generally has no concern for exposures below 100 percent of the RfD. Therefore, based on the completeness and reliability of the toxicity data, and the conservative exposure assessment, BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of sethoxydim, including all anticipated dietary exposure and all other non-occupational exposures.

2. *Infants and children*. Developmental toxicity was observed in a developmental toxicity study using rats but was not seen in a developmental toxicity study using rabbits. In the developmental toxicity study in rats a maternal NOAEL of 180 mg/kg/day and a maternal LEL of 650 mg/kg/day (irregular gait, decreased activity, excessive salivation, and anogenital staining) was determined. A developmental NOAEL of 180 mg/kg/day and a developmental LEL of 650 mg/kg/day (21 to 22 percent decrease in fetal weights, filamentous tail and lack of tail due to the absence of sacral and/or caudal vertebrae, and delayed ossification in the hyoids, vertebral centrum and/or transverse processes, sternebrae and/or metatarsals, and pubes). Since developmental effects were observed only at doses where maternal toxicity was noted, the developmental effects observed are believed to be secondary effects resulting from maternal stress.

3. *Reproductive toxicity*. A 2–generation reproduction study with rats fed diets containing 0, 150, 600, and 3,000 ppm (approximately 0, 7.5, 30, and 150 mg/kg/day) produced no reproductive effects during the course of the study. Although the dose levels were insufficient to elicit a toxic response, the Agency has considered this study usable for regulatory purposes and has established a free-standing NOEL of 3,000 ppm (approximately 150 mg/kg/day) [ref. Proposed Rule at 60 FR 13941].

4. *Reference dose*. Based on the demonstrated lack of significant developmental or reproductive toxicity BASF believes that the RfD used to assess safety to children should be the same as that for the general population, 0.09 mg/kg/day. Using the conservative exposure assumptions described above, BASF has concluded that the most sensitive child population is that of children ages 1 to 6. BASF calculates the exposure to this group to be <75 percent of the RfD for all uses (including those proposed in this document). The proposed tolerances in apricots, cherries, nectarines, and peaches represent an exposure to this group of <1 percent of the RfD. Based on the completeness and reliability of the toxicity data and the conservative exposure assessment, BASF concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the residues of sethoxydim, including all anticipated dietary exposure and all other non-occupational exposures.

H. Other Considerations

The nature of the residue is adequately understood, and practical and adequate analytical methods are available for enforcement purposes. Enforcement methods for sethoxydim are listed in the Pesticide Analytical Manual, Vol. II (PAM II). Enforcement methods have also been submitted to the Food and Drug Administration for publication in PAM II.

There is no reasonable expectation that secondary residues will occur in milk, eggs or meat of livestock and poultry from the proposed uses of sethoxydim on apricots, cherries, nectarines, and peaches; there are no livestock feed items associated with these commodities.

I. International Tolerances

A maximum residue level has not been established for sethoxydim in apricots, cherries (sweet and sour), peaches, and nectarines by the Codex Alimentarius Commission.

2. Monsanto Company

PP 8F2128

Monsanto Company has submitted pesticide petition (PP 8F2128) proposing the establishment of tolerances for residues of the herbicide triallate (*S*-2,3,3, trichloroallyl diisopropyl thiocarbamate) and its metabolite 2,3,3,-trichloro-2-propene sulfonic acid (TSCPA) expressed as the parent equivalent, in on on the raw agricultural commodities sugarbeet roots at 0.1 ppm and sugarbeet foliage at 0.5 ppm.

A. Toxicological Profile

Monsanto has submitted numerous toxicology studies in support of triallate. The following are summaries of key toxicology studies.

1. Several acute toxicology studies place technical triallate in acute toxicity category III for acute oral and dermal toxicity, primary eye and dermal irritation, and in toxicity category IV for acute inhalation toxicity. Triallate is not a skin sensitizer. The NOEL for acute oral toxicity in rats is 50 mg/kg with a LOEL of 100 mg/kg based on flat-footed appearance of the hindlimbs observed at the 100 mg/kg dose level.

2. A more thorough acute neurotoxicity study in rats was conducted in which the observers were unaware of treatment level. In this acute neurotoxicity study rats were administered gavage dosage levels of 0, 60, 300, or 600 mg/kg. The LOEL and NOEL of this study was determined to be 300 mg/kg and 60 mg/kg, respectively. The LOEL was based on a transient decrease in motor activity detected at the time of peak effect (7 hr, postdosing). No gross pathological findings were present; neurohistopathological examinations did not reveal any treatment-related lesions in either the central or peripheral nervous systems. Abnormal behavioral effects were detected at the 600 mg/kg dose but not at any of the lower dose levels.

3. A subchronic neurotoxicity study in rats exposed for 13–weeks through the diet to 0, 100, 500 or 2,000 ppm triallate (0,6.38, 32.9, or 128.8 mg/kg/day, males, respectively; 0, 8.14, 38.9, or 146.6, females, respectively).The LOEL for systemic toxicity and neurotoxicity was 500 ppm (mg/kg/day: 32.9, males; 38.9, females); the NOEL was 100 ppm (mg/kg/day: 6.38, males; 8.14, females). The LOEL was based on treatment-related lesions in the spinal cord and peripheral nervous systems. Abnormal behavioral effects were detected at the 2,000 ppm level but not at any of the lower dose levels.

4. A 2–year feeding study with dogs fed dosage levels of 0, 1.275, 4.25 and 12.75 milligrams/kilograms/day (mg/kg/day) with a no-observed effect level (NOEL) of 1.275 mg/kg/day and a LEL of 4.25 mg/kg/day based on increased liver weight, elevated serum alkaline phosphate values, and increased hemosiderin deposition. The RfD for triallate is 0.013 mg/kg/day based on the NOEL of 1.275 mg/kg/day and an uncertainty factor of 100 for intra- and inter-species variation. Cholinesterase activity in plasma, erythrocytes and brain was not inhibited after 1.5, 3, 6, 12, 18 and 24 months of exposure.

5. A second chronic dog study was conducted in which dogs were administered gelatin capsules containing doses of 0, 0.5, 2.5, or 15 mg triallate/kg/day for 1–year. The LEL based on an increase in serum alkaline

phosphatase level was 15 mg/kg/day and the NOEL was 2.5 mg/kg/day.

6. A 2–year chronic feeding/carcinogenicity study in B6C3F1 mice fed dosage levels of 0, 3, 9, or 37.5 mg/kg/day resulted in a statistically significant increased incidence of hepatocellular carcinomas in males at 37.5 mg/kg/day and a positive trend and a borderline significant increase in females at 37.5 mg/kg/day. For chronic toxicity, the NOEL was 3 mg/kg/day and the LEL was 9 mg/kg/day. The LEL was based on increases in liver weights; the incidence of altered hepatic foci of the liver; splenic hematopoiesis and blood glucose levels in males at 60 and 250 ppm.

7. A 2–year chronic feeding/carcinogenicity study in male and female rats fed dose levels of 0, 0.5, 2.5, and 12.5 mg/kg/day resulted in an increased incidence in renal tubular cell adenoma above historical control levels. Although no absolute pair-wise statistical significance was found, renal tubular cell adenoma is considered a rare tumor type making this finding biologically significant. For chronic toxicity, the NOEL was 2.5 mg/kg/day and the LEL was 12.5 mg/kg/day. The LEL was based on decreased survival in high-dose males and females, decreased mean body weight in high-dose males, and increased adrenal weights in high-dose males.

8. A chronic/oncogenicity study of triallate was also conducted in hamsters at 50, 300, or 2,000 ppm for 79 (females) or 95 (males) weeks. The objective of this study was to see if triallate induces melanotic changes (nodular aggregated of melanocyte, possibly premalignant) in skin of hamsters similar to those induced by diallate, a compound structurally similar to triallate. There were no increases in either non-neoplastic or neoplastic lesions in any organs. For chronic toxicity, the NOEL was 300 ppm and LEL was 2,000 ppm based on a decrease in body weight gain and corresponding decrease in food consumption by males fed the 2,000 ppm diet during the first 13 weeks of the study but not thereafter.

9. A 2–generation reproduction study with rats fed dose levels of 0, 50, 150 or 600 ppm resulted in a reproductive NOEL of 150 ppm and a LEL of 600 ppm. Treatment-related reproductive effects were: reduced pregnancy rates; shortened gestation period; increased neonate mortality in the F2b litter; reduced pup weights at birth in the F2b litter; and reduced pup weights in late lactation in all litters. These effects were only observed in rats treated with the highest dose level which also caused maternal toxicity was manifested by an increase in mortality, decrease in body weight, increase in chronic nephritis, and head bobbing and circling. For maternal toxicity, the LEL was 600 ppm and NOEL was 150 ppm.

10. A developmental toxicity study in rats fed dose levels of 0, 10, 30, or 90 mg/kg/day during gestation days 6-21 resulted in a developmental toxicity NOEL greater than 90 mg/kg/day. For fetotoxicity, the LEL was 90 mg/kg/day and the NOEL was 30 mg/kg/day based on reduced body weight, reduced ossification of the skull, and malaligned sternebrae. For maternal toxicity, the LEL was 90 mg/kg/day and the NOEL was 30 mg/kg/day based on reduction in maternal body weight. The teratogenic NOEL was > 90 mg/kg/day.

11. A developmental toxicity study in rabbits fed doses of 0, 5, 15, and 45 mg/kg/day on gestation days 6 through 28 resulted in a developmental toxicity NOEL greater than 45 mg/kg/day. For fetotoxicity, the LEL was 15 mg/kg/day and the NOEL was 5 mg/kg/day based on an increase in fused sternebrae, increased number of bent hyoid arch bones, as well as decreased body weight. The NOEL was >45 mg/kg/day for teratogenicity.

12. Numerous mutagenicity assays have been conducted with triallate resulting in mixed results. Triallate gave a positive response for base pair conversions in Salmonella strains TA100 and TA1535 with and without activation and negative results without activation in Ames assays. Triallate was positive for mitotic recombination in *Saccharomyces cerevisiae* strain D3 but was negative for gene conversion in strain D4. The mouse lymphoma gene mutation assay produced both positive results for forward mutations at the $TK^{+/-}$ locus with and without activation and negative results at this locus. Triallate was nonmutagenic in a dominant lethal test with mice given a single intraperitoneal injection; this study however, was considered inadequate by current test guideline/standards. Triallate did not induce gene mutations (HGPRT) locus) in Chinese hamster ovary cells (CHO) with and without metabolic activation. It gave a positive response for sister chromatid exchanges (SCEs) in CHO cells both with and without metabolic activation. Triallate did not induce unscheduled DNA synthesis in rat hepatocytes. In an *in vivo* cytogenetic assay, no mutagenic response was seen in the bone marrow cells of hamsters. Overall, triallate is genotoxic in *in vitro* systems and negative in *in vivo* systems and is considered a genotoxic compound.

B. Threshold Effects

1. *Chronic effects*. Based on a complete and reliable toxicity database, the EPA has adopted a reference dose (RfD) value of 0.013 mg/kg bwt/day using the NOEL of 1.275 mg/kg bwt/day from a 2–year dog feeding study and an uncertainty factor of 100. The endpoint effect in this study was increased liver weights and hemosiderin and serum alkaline phosphate (SAP) levels.

2. *Acute effects*. EPA has determined that the appropriate NOEL to use to assess safety of acute exposure is 5 mg/kg bwt/day from a developmental toxicity study in rabbits, based in increases in the incidences of skeletal malformations in rabbit fetuses. EPA has concluded that the subpopulation of concern for this endpoint are females older than 13 years old.

C. Non-Threshold Effects

Carcinogenicity. Triallate has been classified by EPA as Group C - possible human carcinogen. EPA based this classification on a statistically significant increase in hepatocellular tumors in male mice, with a positive trend and a borderline significant increase in females. In addition, the increased incidence of renal tubular cell adenoma, a rare tumor type, in male rats was considered by EPA to be biologically significant although no absolute pair-wise statistical significance was found. Triallate is considered genotoxic and has structural similarities to carcinogenic analogues. EPA is currently applying the extrapolation model approach for risk assessment and has calculated the upper bound potency factor Q_1* to be 0.08320 $(mg/kg/day)^{-1}$.

D. Aggregate Exposure

For purposes of assessing the potential dietary exposure, the theoretical maximum residue concentration (TMRC) and anticipated chronic dietary risk assessment based on exposure to all crops for which triallate is labelled is an appropriate estimate of aggregate exposure. EPA has notified the petitioner that these analyses include permanent tolerances of 0.05 ppm for peas, lentils, barley, and wheat, as established under 40 CFR 180.314. Tolerances are also established for canary grass; however, EPA's Dietary Risk Evaluation Section (DRES) does not have consumption figures for this RAC, and its contribution is expected to be negligible. Anticipated residues, and 100 percent of crop treated was used for sugarbeet sugar. Sugarbeet foliage is a potential animal feed item associated with this use. However, based on the

results of animal metabolism studies, EPA has concluded that secondary residues are not expected to occur in meat, milk, poultry, and eggs as a result of this proposed use.

EPA has also conducted an acute dietary exposure assessment. It is EPA policy to use ''high-end'' residue level estimates for acute exposure analyses; in this case, tolerance levels were used for all commodities.

Other potential sources of exposure of the general population to residues of pesticides are residues in drinking water and exposure from non-occupational sources. Based on the available studies used in EPA's assessment of environmental risk, triallate appears to be moderately persistent and immobile to highly immobile in different soils. EPA's ''Pesticides in Ground Water Database'' (EPA 734–122–92–001, September 1992), shows no detections for triallate in ground water, and it does not exceed the proposed criteria for establishing a pesticide as restricted use due to ground water concerns. It was not a target of EPA's National Survey of Wells for Pesticides, and is not listed as a unregulated contaminant for monitoring in drinking watersupplies under the Safe Drinking Water Act. No Maximum Contaminant Level or Health Advisory levels have been established for triallate.

Previous experience with persistent and immobile pesticides for which there have been available data to perform quantitative risk assessments have demonstrated that drinking water exposure is typically a small percentage of the total exposure when compared to the total dietary exposure. This observation holds even for pesticides detected in wells and drinking water at levels nearing or exceeding established MCLs. Based on this experience and considering the low fraction of a percent of the RfD (<.04 percent) occupied by dietary exposure to triallate, combined exposure from drinking water and dietary exposure would not be expected to result in an ARC that exceeds 100 percent of the RfD. Therefore, potential triallate residues in drinking water are not likely to pose a human health concern.

EPA consideration of a common mechanism of toxicity is not appropriate at this time since there is no information to indicate that toxic effects produced by triallate would be cumulative with those of any other chemical compound. Triallate is a thiocarbamate herbicide. Thiocarbamate herbicides are not applied to any significant degree in areas where triallate would be used to control wild oats in sugarbeet crops. Thiocarbamates are only used to a small extent in other crops. Hence, dietary exposure to thiocarbamate herbicides is expected to be minimal. Considering the low fraction of the percent of the RfD (<.04 percent) occupied by dietary exposure and the minimal exposure levels to other thiocarbamate herbicides through the diet; the combined exposure to other thiocarbamate herbicides would not be expected to pose a human health concern. There is also no data to indicate that there are similar mechanisms of toxicity between triallate and carbamate insecticides that inhibit cholinesterase activity. Triallate does not inhibit cholinesterase activity in plasma, erythrocytes and brain in dogs after chronic exposure to triallate. Triallate does not cause symptoms typical of cholinesterase inhibition in rats after acute or subchronic exposure to triallate.

E. Determination of Safety for U.S. Population and Sub-populations.

1. *Upper bound carcinogenic exposure*. Based on EPA's Q_1* value of 0.08320 $(mg/kg/day)^{-1}$, the upper bound cancer risk contributed by all the published uses, plus this new use on sugarbeets was calculated by EPA to be 1.7×10^{-7} for the U.S. Population in general; risks from the established uses contribute approximately 1×10^{-7} to this risk, and the proposed use on sugarbeets contributes approximately 0.7×10^{-7}. The sub-population with the highest exposure level were children (1 to 6 years old) which has an upper bound cancer risk was 4.2×10^{-7}. These levels of risk are below the level of risk generally considered to be of concern by EPA (1×10^{-6}). EPA has concluded that the dietary cancer risk posed by use of triallate is not considered to be of concern.

2. *Chronic dietary exposure*. Using anticipated residues and realistic estimates of percent of crop treated, the anticipated residue concentration (ARC) for the overall U.S. Population is calculated by EPA to be 0.000002 mg/kg bwt/day, representing 0.01 percent of the RfD, for established uses and this proposed use on sugarbeets. The ARCs for the U.S. Population and the 22 population subgroups all utilized <0.04 percent of the RfD, with the highest exposed subgroup, being children (1 to 6 years old), with 0.035 percent of the RfD utilized. EPA has concluded that the chronic dietary risk exposure from triallate appears to be minimal for this petition for use on sugarbeets, and does not exceed the RfD for any of the DRES subgroups.

3. *Acute dietary exposure*. EPA used ''high-end'' residue level estimates for acute exposure analyses; in this case, tolerance levels were used for all commodities. Since the endpoint used for risk assessment of the acute risk is derived from a rabbit developmental study, EPA concluded that the population subgroup of concern would be females (13+ years old). The MOE value calculated for this subgroup is 12,500, which is well above the level considered by EPA to be of concern (>100). EPA has concluded that there is little concern for acute effects due to dietary exposure to this chemical.

4. *Conclusion*. Based on the above risk assessments, there is a reasonable certainty that no harm will result from aggregate exposure to triallate residues.

F. Determination of Safety for Infants and Children

In assessing the potential for additional sensitivity of infants and children to residues of triallate, the developmental toxicity studies in the rat and rabbit and the 2–generation reproduction study in the rat should be considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity. The results of these studies indicate that triallate is not a specific teratogen or reproductive toxin. The only evidence of developmental toxicity occuring below maternally toxic doses was an increase in fused sternebrae, increase number of bent hyoid arch bones, as well as decreased body weight in rabbits. In most instances, fusion only involved two adjacent sternebrae and not the entire chain. Consequently, this type of skeletal defect is considered a minor anomaly rather than a major malformation. The incidence of bent hyoid arch bones was increased from control values but within the laboratory's historical control range. The LEL for fetotoxicity in rabbits was considered by EPA to be 15 mg/kg/day and the NOEL was 5 mg/kg/day.

The 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 completeness of the database or for significant developmental effects. The toxicological database relative to pre- and post-natal effects of triallate is complete. There are no developmental effects that are of substantial concern. Thus, an additional safety factor is not necessary.

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.

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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