sequences, an active ingredient not included in any previously registered product.

The application was approved on May 29, 1996, as CryIA(b) Form of the B.t.k. Insect Control Protein for seed propagation (EPA Registration Number 524–492). The chemical was amended to read ''*Bacillus thuringiensis* delta-endotoxin as produced by the CryIA(b) gene and the genetic material necessary for its production (PV-ZMCT01) in corn.''

A conditional registration may be granted under section 3(c)(7)(C) of FIFRA for a new active ingredient where certain data are lacking, on condition that such data are received by the end of the conditional registration period and do not meet or exceed the risk criteria set forth in 40 CFR 154.7; that use of the pesticide during the conditional registration period will not cause unreasonable adverse effects; and that use of the pesticide is in the public interest.

The Agency has considered the available data on the risks associated with the proposed use of *Bacillus thuringiensis* delta-endotoxin as produced by the CryIA(b) gene and the genetic material necessary for its production (PV-ZMCT01) in corn, and information on social, economic, and environmental benefits to be derived from such use. Specifically, the Agency has considered the nature and its pattern of use, application methods and rates, and level and extent of potential exposure. Based on these reviews, the Agency was able to make basic health and safety determinations which show that use of *Bacillus thuringiensis* delta-endotoxin as produced by the CryIA(b) gene and the genetic material necessary for its production (PV-ZMCT01) in corn during the period of conditional registration will not cause any unreasonable adverse effect on the environment, and that use of the pesticide is in the public interest.

This product is conditionally registered in accordance with FIFRA section 3(c)(7)(C). If the conditions are not complied with the registration will be subject to cancellation in accordance with FIFRA section 6(e).

Consistent with section 3(c)(7)(C), the Agency has determined that this conditional registration is in the public interest. Use of the pesticides are of significance to the user community, and appropriate labeling, use directions, and other measures have been taken to ensure that use of the pesticides will not result in unreasonable adverse effects to man and the environment.

More detailed information on this conditional registration is contained in an EPA Pesticide Fact Sheet on *Bacillus thuringiensis* CryIA(b) delta-endotoxin and the genetic material necessary for its production in corn.

A copy of the fact sheet, which provides a summary description of the chemical, use patterns and formulations, science findings, and the Agency's regulatory position and rationale, may be obtained from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161.

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are available for public inspection in the Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Rm. 1132, CM #2, Arlington, VA 22202 (703–305–5805). Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of Information Office (A-101), 401 M St., SW., Washington, D.C. 20460. Such requests should: (1) Identify the product name and registration number and (2) specify the data or information desired.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: July 30, 1997.

Janet L. Andersen,

Director, Biopesticides and Pollution Prevention Division, Office of Pesticide Programs.

[FR Doc. 97–21146 Filed 8-8-97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–755; FRL–5736–1]

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–755, must be received on or before September 10, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7506C), Information Resources and Services Division, 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: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
George LaRocca (PM 13).	Rm. 204, CM #2, 703–305–6100, e-mail: larocca.george@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Mary Waller, Acting (PM 21).	Rm. 265, CM #2, 703–308–9354, e-mail: waller.mary@epamail.epa.gov.	Do.

Product Manager	Office location/telephone number	Address
James Tompkins, Acting (PM 25).	Rm. 239, CM #2, 703–305–5697, e-mail: tompkins.jim@epamail.epa.gov.	Do.

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–755] (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–755] 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: August 1, 1997.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. Bayer Corporation

PP 7E4825

EPA has received a pesticide petition (PP 7E4825) from Bayer Corporation, 8400 Hawthorn Road, Kansas City, MO 64120, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing import tolerances for residues of the fungicide Tolylfluanid in or on the raw agricultural commodities apples and grapes at 5.0 parts per million (ppm), hops at 30 ppm and tomatoes at 1.0 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Plant metabolism studies were conducted using radiolabeled tolylfluanid applied to apples, grapes, and strawberries. Unchanged parent tolylfluanid was the major metabolite identified in these studies.

2. *Analytical method*. Bayer has developed an analytical method for the determination of tolylfluanid residues in raw agricultural and processed commodities of apples, grapes, tomatoes, and hops. Samples are analyzed by gas chromatography using thermionic nitrogen-phosphorus detector or flame photometric detector following extraction, filtration, and cleanup procedures. The limit of quantitation is 0.02 mg/kg for all matrices, except it is 0.05 mg/kg for raisins and wet apple pomace, 0.5 mg/kg for green hop cones, and 1.0 mg/kg for dried hop cones.

3. *Magnitude of residues*. Bayer has conducted over 90 residue field trials in seven countries on apples, grapes, tomatoes, and hops. Residues of tolylfluanid in or on grapes harvested 14, 21 or 35 days following treatment according to recommended practices ranged from 0.03 mg/kg to 3.45 mg/kg, except residues of tolylfluanid were 5.08 mg/kg in one sample from a trial conducted in Spain. Residues of tolylfluanid ranged from 0.03 mg/kg to 0.66 mg/kg in tomatoes harvested 3 or 7 days following multiple applications with tolylfluanid. Residues of tolylfluanid ranged from 0.14 to 2.31 mg/kg in or on apples harvested 7 days after multiple applications with tolylfluanid. Residues of tolylfluanid in or on hops harvested 14 days following multiple applications ranged from 3.31 mg/kg to 27.0 mg/kg (dried cone) and ranged from 3.8 mg/kg to 17.6 mg/kg (green cone).

Studies have also been conducted to evaluate the potential for concentration of tolylfluanid residues during the processing of apples, grapes, and tomatoes. Tolylfluanid does not have the potential to concentrate in the EPA required processed commodities consumed by humans for apples, grapes and tomatoes. Residues of tolylfluanid may have the potential to concentrate in wet apple pomace, an animal feed item.

B. Toxicological Profile

1. *Acute toxicity*. Tolylfluanid exhibits low acute oral, dermal, and inhalation toxicity (LD_{50s} >5,000 mg/kg b.w.). An acute neurotoxicity study showed no specific evidence of neurotoxicity; non-specific signs of toxicity were observed in this study (in females only) at doses at and greater than 150 mg/kg b.w. Tolylfluanid is a severe dermal irritant, moderately irritating to the eye, and a skin sensitizer. Tolylfluanid showed no systemic toxicity following subacute dermal administration, but did cause dermal irritation. Effects seen in the acute as well as subacute inhalation

study indicate tolylfluanid is a strong respiratory irritant.

2. *Genotoxicity*. The genotoxic potential of tolylfluanid was assessed in several *in vivo* and *in vitro* studies. The weight-of-the-evidence indicates that tolylfluanid is not genotoxic.

3. *Reproductive and developmental toxicity*. Tolylfluanid showed no evidence of developmental toxicity based on two rat developmental toxicity studies. Tolylfluanid showed evidence of developmental effects in rabbits but only at a maternally toxic dose level.

Two complete 2-generation reproductive toxicity studies in rats and one supplementary 2-generation reproductive toxicity rat study have been conducted on tolylfluanid. Reproductive toxicity (decreased body weight development in pups and decreased number of pups born, birth weight, litter size, and lactation index) was noted only in the presence of parental toxicity (decreased body weight gain, organ weight changes, and hyperostosis of the crania).

4. *Subchronic toxicity*. Subchronic toxicity studies have been done with tolylfluanid in rats and dogs. Decreased body weight gain, decreased liver enzymes, slightly increased relative liver weights, and thyroid toxicity were noted in a subchronic rat dietary study (no correlating histopathological findings). Decreased body weight gain, increased liver enzyme activity, slightly increased relative liver weights, and increased PAS staining in the liver occurred in a subchronic dietary dog study. A subchronic neurotoxicity study in rats showed no evidence of neurotoxicity.

5. *Chronic toxicity*. Chronic toxicity studies on tolylfluanid were done in the rat, mouse and dog. Tolylfluanid was tested in two rat chronic dietary studies. Increased growth of the incisors of the upper jaw and skeletal changes (hyperostosis in the skull and ribs) resulted from the high fluorine content of the compound. Hepatotoxicity and renal toxicity were seen in rats, mice, and dogs. Hepatotoxicity was evidenced by hepatocellular cytoplasmic changes, vacuolation, and focal fatty changes in rats, hepatocellular hypertrophy and single cell necrosis in mice, decreased liver enzymes in rats, and increased liver enzymes in mice and dogs. Renal toxicity (microscopic kidney lesions, increased relative kidney weights, effects on urinalysis parameters) was probably attributable to the effects of fluoride on renal tubules. A second chronic toxicity study in dogs is currently ongoing (results not yet available).

6. *Oncogenicity*. Tolylfluanid showed no evidence of direct oncogenic activity in rats or mice. In rats tolylfluanid altered thyroid hormone levels and an increased incidence of hyperplastic and neoplastic lesions of the thyroid (primarily adenomas) in rats was observed. The thyroid neoplasia is considered to be a secondary (thresholdable) effect to altered thyroidal iodine metabolism and does not suggest a direct oncogenic effect. No treatment-related neoplasms were seen in the mouse oncogenicity study.

Based on the chronic toxicity data, Bayer believes the RfD for tolylfluanid is 0.08 mg/kg, based on the no observed adverse effect level (NOAEL) of 8 mg/kg b.w./day for parental and reproductive toxicity identified in the second 2-generation rat reproductive toxicity study (Pinckel and Ricke, 1995) and an uncertainty factor of 100. No unique concern for toxicity to infants and children was identified, therefore an additional safety factor is not warranted. (Note there is a seven-fold difference between the NOAEL and lowest effect level (LEL).

Using the Guidelines for Carcinogenic Risk Assessment published in September 1986, we believe the Agency will classify tolylfluanid as a Group C carcinogen (possible human carcinogen) based on benign thyroid tumors seen in the chronic rat studies). Mechanistic studies with tolylfluanid have shown that these tumors are induced through a nonlinear threshold mechanism similar to that discussed in EPA's thyroid policy document. Therefore, tolylfluanid should be regulated using the margin of exposure approach.

7. *Animal metabolism*. Metabolism studies were conducted using hens and goats. No residues of parent tolylfluanid were detected in any tissues, organs, milk, or eggs. Tolylfluanid is metabolized and excreted rapidly and efficiently in mammals.

C. Aggregate Exposure

1. *Dietary exposure*. Food and drinking water/non-dietary exposure.

2. *Food*. A chronic dietary exposure analysis was conducted for tolylfluanid. The reference dose (RfD) was 0.08 mg/kg/day based on a NOEL of 8 mg/kg/day and an uncertainty factor of 100. The no observed effect level (NOEL) was obtained from the rat reproduction study and the effect was decreased pup viability and decreased body weights.

The RfD could change based on the NOEL from a repeat chronic dog toxicity study which is currently ongoing (doses tested: 5, 20, and 80 mg/kg/day). The final report for this study is expected to be completed in the second part of 1997. If necessary, revising the RfD will be addressed at that time.

Tolylfluanid does not have the potential to concentrate in processed commodities consumed by humans. The proposed MRLs for the respective crops were used for the raw agricultural and processed commodities for grapes (5 mg/kg), tomatoes, (1 mg/kg), and hops (30 mg/kg). The anticipated residue level for fresh apples and apple juice was calculated by adjusting the proposed MRL for apples (5 mg/kg) for the percentage of fresh apples (4.8%) and apple juice (59.7%) consumed in the U.S. that are imported. No adjustments were made for the anticipated residue levels for grapes, tomatoes and hops.

The results of the chronic dietary exposure analysis for the overall U.S. population and the three most highly exposed population subgroups are summarized as follows.. The exposure estimate was compared against the RfD of 0.08 mg/kg. The theoretical maximum residue contribution (TMRC) as percentage of the RfD, was 9.53% for the U.S. population, 53.36% for non-nursing infants, 38.02% for nursing infants (0-1 yr old), and 26.16% for children (1-6 yrs old). The anticipated residue contribution (ARC) as percentage of the RfD was 5.97% for the U.S. population, 23.29% for non-nursing infants, 15.41% for nursing infants and 15.10% for children. As seen above, chronic dietary exposure to tolylfluanid is less than 24% of the RfD for even the most highly exposed subgroup. In addition, these exposure estimates greatly over estimate the anticipated risk for the following reasons: (1) a relatively small percentage of these crops will be treated with tolylfluanid; (2) a small percentage of the treated crops are imported to the U.S.; (3) a small percentage of the total U.S. consumption of these crops are imported products; and (4) the actual residues in the imported commodities will likely be below the proposed MRLs.

3. *Drinking water*. Tolylfluanid residue levels in tap water, non-tap water, and water in commercially prepared food were assumed to be zero because tolylfluanid is not registered for use in the United States and therefore, the only exposure is from the importation of tolylfluanid-treated commodities.

4. *Non-dietary exposure*. Tolylfluanid is not registered in the United States, therefore there is no non-occupational, structural or residential exposure.

D. Cumulative Effects

Tolylfluanid is a fungicide that is somewhat structurally similar to

Captan, and appears to share a common mechanism of fungicidal action with this product. However, tolylfluanid does not show a similar mammalian toxicity profile to Captan, which has been reported to produce mouse gastrointestinal tumors and male rat kidney tumors. No significant cumulative toxicity to mammals based on a common mechanism of action to that of Captan is anticipated for tolylfluanid.

Tolylfluanid alters the thyroid hormone balance, but: (1) no data exist showing specifically how tolylfluanid causes thyroid changes; (2) tolylfluanid is not known to be structurally similar to other thyroid tumorigens; (3) no common mechanism has been established or proposed and (4) even if it is eventually determined that the mechanism for thyroid tumorigenesis may be similar to other classes of pesticides, this endpoint is seen with tolylfluanid only at very high exposure levels. If an RfD for tolylfluanid were based on dose levels at which thyroid hormone levels were altered, a very low impact on a cumulative risk cup would be anticipated because the potency of tolylfluanid is very low.

Endocrine effects. Endocrine-related effects of tolylfluanid exposure appear to be limited to the thyroid. No evidence of estrogenic or anti-estrogenic activity was present in the available animal studies. The developmental toxicity and reproductive toxicity studies showed no effects suggesting endocrine disruption, (e.g., change in fetal sex ratios, change in estrous cycles or mating performance, change in fertility, or malformed or altered reproductive organ development).

E. Safety Determination

1. *U.S. population*. A chronic dietary exposure analysis was conducted for tolylfluanid. The chronic dietary exposure to tolylfluanid is 5.97% of the RfD for the U.S. population, using the ARC.

2. *Infants and children*. A chronic dietary exposure analysis was conducted for tolylfluanid. The chronic dietary exposure to tolylfluanid is 23.29% of the RfD for non-nursing infants, the most highly exposed group, using the ARC.

F. International Tolerances

The current Codex tolerances for tolylfluanid are based on residues of parent only. The Codex tolerances are: 5 mg/kg for currents (black, red, and white), 2 mg/kg for Gherkins, 1 mg/kg for head lettuce, 5 mg/kg for pome fruits, 3 mg/kg for strawberries, and 2 mg/kg for tomatoes. (Mary Waller)

2. DowElanco

PP 5E4571

EPA has received a pesticide petition (PP 5E4571) from DowElanco, 9330 Zionsville Road, Indianapolis, IN 46268-1054, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the herbicide, tebuthiuron and its related metabolites in or on the food commodities refined sugar and molasses at 0.05 parts per million (ppm) from treatment of sugarcane outside of the United States with tebuthiuron. The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by gas liquid chromatography using flame photometric detection. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of tebuthiuron has been investigated in grasses. The residues of concern are the parent compound and its metabolites 103 (OH) N-[5-(2-hydroxy-1,1-dimethylethyl)-1,3,4-thiazol-2-yl]-N,N'dimethylurea, 104 N-[5-(1,1-dimethylethyl)-1,3,4-thiazol-2-yl]-N-methylurea, and 109 N-[5-(1,1-dimethylethyl)-1,3,4-thiazol-2-yl]-N'-hydroxymethyl-N-methylurea. Tebuthiuron and its metabolites 104 and 109 have been identified in sugarcane.

2. *Analytical method*. The method for enforcement of plant commodities tolerances is a GLC method with flame photometric detection. The stated detection limit for the parent compound and metabolites 103 (OH), 104 and 109 is 0.01 ppm.

Enforcement methods for milk and meat have been developed by DowElanco and have been submitted to the Agency as part of reregistration. An adequate method (GC/flame photometric detection) exists to determine tebuthiuron and some metabolites (104, 106, and 109) in milk and ruminant tissue. The new enforcement method is needed to determine additional metabolites of toxicological concern.

3. *Magnitude of residues*. Commercial sugarcane samples were collected from two major Brazilian sugarcane growing regions. Tebuthiuron had been applied at rates ranging from 750 to 1,500 g ai/ha. Most of the samples were collected approximately 12 months after treatment. Analysis for tebuthiuron and metabolites 104 and 109 occurred within 60 days of sample collection. No residues of tebuthiuron were found above the LOQ (0.01 ppm). In many samples there was no detection of metabolites. In samples at one site treated with 1,250 g ai/ha, however, there were residues of the combined metabolites at the LOQ.

B. Toxicological Profile

1. *Acute toxicity*. Tebuthiuron is classified as a Moderate (Category II) acute toxicant based upon the acute oral LD_{50} value in the rat (387-477 mg/kg) and rabbit (286 mg/kg. The LD_{50} for the dermal toxicity in the rabbit was greater than the limit dose of 5,000 mg/kg. The acute inhalation LC_{50} in the rabbit was greater than 3.696 mg/L. Tebuthiuron produced slight irritation (slight conjunctival hyperemia at 1 hour post-treatment; Category IV) and was not a dermal irritant (Category IV) or dermal sensitizer. The following table summarizes the acute toxicity profile of tebuthiuron.

Test	Species	Category
Oral	Mouse	III
	Rat, Rabbit	II
Dermal	Rabbit	IV
Inhalation	Rat	III
Eye Irritation	Rabbit	IV
Dermal Irritation	Rabbit	IV
Dermal Sensitization	Guinea Pig	none

2. *Genotoxicity*. Results from a battery of assays in vitro indicate that tebuthiuron is not genotoxic. It was inactive in the Ames S. typhimurium reverse gene mutation assay with or without metabolic activation. In the mouse lymphoma assay, tebuthiuron was negative without metabolic activation and slightly positive (mutation index of 2) with metabolic activation at doses 700 mg/mL. In this assay, cytotoxicity was observed at doses 200 mg/mL. In Chinese Hamster Ovary cells, there were chromosomal aberrations and cytotoxicity at the highest doses tested with (1,550 mg/mL) and without (1,950 mg/mL) metabolic activation. There was no Unscheduled DNA Synthesis in primary rat hepatocytes at 800 mg/mL, while cytotoxicity was observed at 900 mg/mL.

3. *Reproductive and developmental toxicity*. In a 3-generation reproduction study in which rats were fed 28 or 56 mg tebuthiuron/kg/day, F_{1b} weanling pups had reduced mean body weight gains. No reproductive no observed effect level (NOEL) could be determined from this study.

In a 2-generation reproduction study, rats were fed tebuthiuron at dietary levels of 100, 200 or 400 ppm (7, 14, 28 mg/kg/day). There was a reduced rate of body weight gain in the F_1 females during the premating period at the 14 and 28 mg/kg/day dose levels. The systemic NOEL of this study was 7 mg/kg/day and the reproductive NOEL was the highest dose tested (28 mg/kg/day). The RfD for tebuthiuron was determined to be 0.07 mg/kg/day based upon the systemic NOEL of this 2-generation reproduction study with a Safety Factor of 100.

In a developmental toxicity study in which rats were fed 0, 15, 30, or 45 mg tebuthiuron/kg/day, the maternal NOEL was 30 mg/kg/day based upon reduced body weight gain and food consumption. There were no adverse developmental effects observed in this study. The developmental NOEL was the highest dose tested (45 mg/kg/day).

Rabbits were administered 0, 10, or 25 mg tebuthiuron/kg/day by oral gavage on gestation days 6-18. The maternal toxicity NOEL was the highest dose tested (25 mg/kg/day). Although there was an apparent decrease in fetal weights at the highest dose, this was probably the result of an increased number of fetuses per litter in the highest dose group (5.7 fetuses/litter versus 4.4 fetuses/litter in controls). Therefore, no treatment-related adverse affects were attributed to tebuthiuron.

These studies indicate that tebuthiuron is not a developmental or reproductive toxicant.

4. *Subchronic toxicity*. Rats were exposed to tebuthiuron in the diet at the exposure levels of 0, 20, 50, or 125 mg/kg/day for 90 days. The NOEL was determined to be 50 mg/kg/day based upon reduced body weight, increased relative liver, kidney, and gonad weights, and slight vacuolization of pancreatic acinar cells at 125 mg/kg/day. In addition, males also had increased relative spleen and prostate gland weights at the highest dose.

Dogs were exposed to tebuthiuron in the diet for 90 days at 0, 500, 1,000, or 2,500 ppm. The NOEL was determined to be 500 ppm (12.5 mg/kg/day) based upon anorexia, weight loss, increases in blood urea nitrogen and alkaline phosphatase activity, and increases in spleen and thyroid gland weights at the LOEL value of 1,000 ppm (25 mg/kg/day).

Rabbits were exposed dermally to 1,000 mg tebuthiuron/kg/day for 6 hours a day for 21 days. Slight erythema occurred in these rabbits and resolved by day 7. The NOEL was less than 1,000 mg/kg/day.

5. *Chronic toxicity*. Dogs were fed tebuthiuron in capsules at doses of 0, 12.5, 25, or 50 mg/kg/day for 1-year. The NOEL was determined to be 25 mg/kg/day based upon the clinical signs of anorexia, diarrhea, and emesis as well as increased thrombocyte count, alanine transferase, and alkaline phosphatase activity, and increased liver, kidney, and thyroid weights at the LOEL value of 50 mg/kg/day.

Tebuthiuron was fed to 40 Harlan (Wistar) rats/sex/group at concentrations 400, 800, or 1,600 ppm (20, 40, or 80 mg/kg/day) for 2 years. There were 60 control rats/sex. The systemic NOEL value was 40 mg/kg/day and the lowest observed effect level (LOEL) value was 80 mg/kg/day based upon a reduction in weight gain and elevated kidney weights. There were no treatment-related carcinogenic effects.

In another study, tebuthiuron was fed to 40 Harlan (ICR) mice/sex/group at 400, 800, or 1,600 ppm (57, 144, or 228 mg/kg/day) for 2 years. There were 60 control mice/sex. The systemic NOEL value was the highest dose tested (228 mg/kg/day). Although there were no compound-related carcinogenic effects, the dose levels were judged to be inadequate for carcinogenic testing. This study was considered to be supplemental to the rat study by the Health Effects Division (HED) and the Reference Dose (RfD) Committee, and that no additional study would be required. The HED RfD Committee has classified tebuthiuron as a Group D carcinogen (not classifiable as to human carcinogenicity).

6. *Animal metabolism*. The metabolism of tebuthiuron has been investigated in ruminants. The residues of concern in milk and meat are the parent compound and its metabolites 104, 106 N-[5-(1,1-dimethylethyl)-1,3,4-thiazol-2-yl]-urea, 108 [2-dimethylethyl)-5-amino-1,3,4-thiadiazole], and 109.

The metabolism of radiolabelled tebuthiuron was conducted in four laboratory species (rats, rabbits, dogs, and mice) using a single administration by gavage of 10 or 160 mg/kg. In all four species, tebuthiuron was readily absorbed, metabolized, and excreted. In rats, rabbits and dogs, elimination in the urine accounted for 84% to 95% of the administered dose (the parent compound accounting for 0.4% to 0.7% of the dose). Biliary excretion was demonstrated in the rat. Mice excreted less radioactivity in the urine (66%; with 23% as unchanged parent compound) and more in the feces (31%) as compared with the other species examined. At least seven major metabolites were excreted in the urine, and there was no unusual tissue distribution of metabolites.

C. Aggregate Exposure

Tebuthiuron currently is registered for treatment of forage grasses and hay, therefore, potential dietary exposure to humans is from secondary residues in milk and meat from livestock which have consumed treated grasses. A chronic dietary exposure analysis was conducted for tebuthiuron using the existing tolerances of 0.3 ppm in milk and 2.0 ppm in meat and the proposed tolerance of 0.05 ppm in cane sugar and molasses. The exposure assessment included the worst-case assumptions that all ruminants and horses were fed treated grasses, and sugar and molasses available to consumers came from treated sugarcane. As tebuthiuron was detected in ground water at 23 ppb in a small scale monitoring study under a high exposure scenario, this value was used in all water in the consumption survey. In this estimation, exposure to the U.S. population from water sources represented 1.1% of the RfD (about 24% of total exposure to tebuthiuron). The following table summarizes the results from the chronic aggregate exposure analysis.

	Dietary Exposure (mg/kg BW/day)	% of Rfd
All Infants	0.004971	7.1%
Nurs. Infants < 1 yr	0.001859	2.7%
Non-nurs. Inf. < 1 yr	0.006429	9.2%
Children 1-6 yrs	0.005732	8.2%
Children 7-12 yrs	0.004376	6.3%
Females 13-50 yrs	0.002394	3.4%

As the RfD was based upon decreased body weight gains in the reproduction toxicity study, the subpopulations shown above represent the groups with the highest potential impact from this endpoint. This is a worst-case estimate based upon tolerance values and the assumption that all water sources will have the residue concentration that was found in the monitoring study. Even with these worst-case estimations, aggregate exposure levels were less than 10% of the RfD for any subpopulation.

D. Cumulative Effects

The potential for cumulative effects of tebuthiuron and other substances that have a common mechanism of toxicity was considered. The mammalian toxicity of tebuthiuron is well defined. However, the biochemical mechanism of toxicity of this compound is not well known. No reliable information exists to indicate that toxic effects produced by tebuthiuron would be cumulative with those of any other chemical compounds. Therefore, consideration of a common mechanism of toxicity with other compounds is not appropriate. Thus only the potential risks of tebuthiuron are considered in the aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Based upon maximum expected residues in meat, milk, and refined sugar and molasses from sugarcane, DowElanco concludes that there is a reasonable certainty of no harm resulting from aggregate exposure of tebuthiuron to the general population.

2. *Infants and children*. The toxicological data indicate that tebuthiuron is not a developmental or reproductive toxicant, and that infants and children are not sensitive subpopulations. There is a reasonable certainty that no harm will result from aggregate exposure of tebuthiuron to infants and children.

F. International Tolerances

No Codex MRLs have been established or proposed for residues of tebuthiuron.

G. Endocrine Effects

An evaluation of the potential effects on the endocrine systems of mammals has not been determined; However, no evidence of such effects were reported in the chronic or reproductive toxicology studies described above. There was no observed pathology of the endocrine organs in these studies. There is no evidence at this time that tebuthiuron causes endocrine effects. (James Tompkins)

3. Merck Research Laboratories

PP 7F4844

EPA has received a pesticide petition (PP 7F4844) from Merck Research Laboratories, P.O. Box 450, Hillsborough Road, Three Bridges, NJ. The petition proposes, pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d). that EPA amend 40 CFR part 180 to establish tolerances for pesticide chemical residues consisting of the insecticide abamectin (avermectin B_1) and/or its delat 8,9- isomers in or on the following food items: grapes, raisins, and other grape-derived food items at 0.02 parts per million (ppm) and chili peppers at 0.01 ppm. Abamectin has been approved by EPA for use on many other food crops, including various tree fruits, nuts, and vegetables (including bell peppers), as well as hops and cotton. Tolerances corresponding to these uses are in effect for abamectin residues (including a tolerance for bell peppers at 0.01 ppm); the most recent rule, reissuing tolerances for abamectin on citrus and cotton under the FFDCA as amended by the Food Quality Protection Act (FQPA), was published in the **Federal Register** on March 24, 1997 (62 FR 13833). A notice of filing with regard to that rulemaking had earlier been published on December 10, 1996 (61 FR 65043). The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using UV detection. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of abamectin in plants is adequately understood and the residues of concern include the parent insecticide, abamectin or avermectin B_1 (which is a mixture of a minimum of 80% avermectin B_{1a} and a maximum of 20% avermectin B_{1b}) and the delta 8,9-isomer of the B_{1a} and of the B_{1b} components of the parent insecticide. Animal metabolism also has been studied but is not relevant to this petition, since the crops involved are not significant animal feed items.

2. *Analytical method*. Practicable analytical methods (HPLC-fluorescence methods) are available to detect residues that would exceed the proposed tolerances, and for enforcement. The methods are sufficiently sensitive to detect residues at or above the tolerances proposed. All methods have undergone independent laboratory validation as required by PR Notice 88-5.

3. *Magnitude of residues*. In residue field trials on grapes, the highest residue combined values in day 28 (or later) samples was 6.7 ppb for abamectin B_{1a} plus its delta 8,9- photoisomer; there were no detectable levels of abamectin b_{1b} residues in any of the day 28 (or later) samples. In the two raisin samples the levels for abamectin B_{1a} ranged from 8.6 to 11.8 ppb. The residues did not concentrate in grape juice. These data support the proposed tolerance of 0.02 ppm for total toxic residues of abamectin on the RACs grapes, grape juice, and raisins and the proposed 28-day PHI.

For chili peppers the primary B_{1a} component and its photoisomer, the residues recovered on day 7 were all either nonquantifiable (less than 5 ng/g, but equal or greater to 2 ng/g) or nondetectable (less than 2 ng/g). These data support the proposed tolerance of 0.01 ppm for total toxic residues of

abamectin on the RAC chili peppers and the proposed 7-day PHI.

B. Toxicological Profile

All the toxicity data on which this petition is based have previously been submitted to EPA in support of other petitions, and were summarized in the recent notice of filing (61 FR 65043). In the recent final rule (62 FR 13833) EPA concluded that acute dietary exposure risk evaluations should be based on a no observed effect level (NOEL) of 0.06 mg/kg bw/day (mouse pup NOEL in a developmental toxicity study using the delta 8,9-isomer of abamectin) and that a margin of exposure of 300 should be required. EPA determined that chronic dietary exposure risk evaluations should be based on a reference dose (RfD) of 0.0004 mg/kg bw/day, derived from a 2-generation rat reproduction study with a NOEL of 0.12 mg/kg/day and an uncertainty factor of 300.

This petition contains a supplemental a document setting forth new acute exposure and chronic exposure and risk analyses that corrects previously submitted analyses to reflect newly available residue data on chili peppers (the previously submitted report used data on bell peppers only) and to reflect current Agency preferences regarding the handling of blended foods. The results of the old and new analyses are substantially similar.

C. Aggregate Exposure

1. *Dietary exposure*. The March 1997 rule was based on an exposure analysis submitted by Merck that included exposure attributable to grapes and peppers. The exposure contribution for chili peppers was calculated using data on bell peppers. With the present petition, Merck is submitting new residue data on chili peppers and a revised acute and chronic risk assessment that incorporates that data; the exposure levels have not changed significantly. The chronic exposure for the U.S. population at large is estimated to be 0.000006 mg/kg bw/day, and for children aged 1-6, the highest exposure group, chronic exposure is estimated to be 0.000014 mg/kg bw/day. The estimated acute exposure (at the 99.9th percentile level) is for the U.S. population at large, 0.000025 mg/kg bw/day.

2. *Drinking water*. In the final rule EPA also concluded that drinking water exposure assumptions were not of concern.

3. *Non-dietary exposure*. In the final rule published on March 24, 1997, EPA concluded that there is no likelihood of significant exposure from the registered residential indoor and outdoor nonfat use of abamectin. Approval of tolerances for grapes and chili peppers would not change that conclusion.

D. Cumulative Effects

Abamectin is a member of the avermectin family of natural and semi-synthetic compounds. Ivermectin, another member of that family, is very closely similar to abamectin in structural standpoint; it is used as a human and animal drug. Emamectin, a proposed new pesticide, is made from abamectin but is less similar to abamectin than is avermectin. These compounds are all Merck products. Other companies product certain other drugs have certain structural similarities. Merck in not aware of any information indicating what, if any, cumulative effect would result from exposure to two or more of these compounds. The March 1997 rule discussed cumulative effects and stated that in view of the lack of information on how to evaluate possible common mechanisms, it would not assume that abamectin has a common mechanism of toxicity with any other substance.

E. Safety Determination

In the recently issued final rule (62 FR 13833, March 24, 1997) EPA discussed analyses of risks from chronic and acute exposure for all existing or pending tolerances. Those analyses included exposure to grapes and peppers, among other previously-approved and then-pending uses. In the final rule, EPA found the risks to be acceptable, with regard to both the general U.S. population and with regard to infants and children. As noted earlier, Merck now has submitted specific residue data on chili peppers, but the exposure analyses are not significantly affected thereby.

F. International Tolerances

Codex has not issued abamectin tolerances for grapes and chili peppers. (George LaRocca)

[FR Doc. 97–21147 Filed 8–8–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[FRL–5873–4]

Four Documents Required Under the Safe Drinking Water Act as Amended

AGENCY: Environmental Protection Agency.

ACTION: Notice of Availability.

SUMMARY: In this notice, the Environmental Protection Agency (EPA or the Agency) is publishing two documents, and announcing the public availability of three other documents. All the documents relate to provisions in the Safe Drinking Water Act, as amended in 1996 (SDWA), and were issued by the Agency on August 6, 1997.

The documents that can be obtained from the Agency are: (1) EPA 816–R–97–009, ''State Source Water and Assessment Guidance'' which is guidance for states to follow in developing state source water assessment and petition programs (SDWA sections 1453 and 1454); (2) EPA 816–R–97–010, ''Guidance for Future State Ground Water Protection Grants' which establishes procedures for application for state ground water protection program assistance and identifies key elements of state ground water protection programs (SDWA section 1429(b)); and (3) EPA–815–R–97–002, ''Small System Compliance Technology List for the Surface Water Treatment Rule'' which contains detailed information on the list of technologies published in this notice.

Published in this notice are the list of small system compliance technology that meets the Surface Water Treatment Rule (SWTR) for three population sizes of small drinking water systems as required by SDWA section 1412(b)(4)(E)(v) and alternative monitoring guidelines for states to follow in proposing alternative monitoring requirements for chemical contaminants as required by SDWA 1418(b)(2). The alternative monitoring guidelines are also available as a separate document, EPA 816–R–97–001.

DATES: The documents are available beginning August 6, 1997.

ADDRESSES: Copies of these documents are available from the Safe Drinking Water Act Hotline, telephone (800) 426–4791 or e-mail hotline-sdwa@epamail.epa.gov. Copies are also available from the Office of Water Resource Center (RC4100), U.S. EPA, 401 M Street, SW, Washington, DC 20460, (202) 260–7786. The Center is open from 8:30 a.m. until 5:00 p.m. Monday through Friday. The documents are available, as of August 6, 1997, on EPA's Web Site at the following address: ''http://www.epa.gov/OGWDW''.

SUPPLEMENTARY INFORMATION:

Table of Contents