mutagenic activity was associated with AVG in cultures of mouse lymphoma cells with or without metabolic activation. In an *in vivo* rat bone marrow cell micronucleus test, there was no indication that AVG was genotoxic.

3. *Developmental toxicity*. In a developmental toxicity study in rats by oral gavage, a no observable adverse effect level (NOAEL) of 1.77 mg a.i./kg bwt day was determined for both developmental and maternal toxicity. Two-generation reproduction study (rat) data are pending, as a condition of the section 3 registration. Interim data on the first generation have been submitted to the Agency.

4. *Subchronic toxicity*. A reference dose (RfD) of 0.002 mg a.i./kg bwt/day was derived from a 90 day feeding study in rats in which there was decreased food consumption, body weight and food efficiency (body weight gain/food consumption), and fatty changes in kidney and liver at dosage levels of 9 mg a.i./kg bwt/day or higher. The NOAEL in this study was assigned as 2.2 mg a.i./kg bwt/day. In a 21 day dermal toxicity study in rats, the NOAEL was greater than 1,000 mg a.i./kg/day. In a 28 day dietary immunotoxicity study in rats with a NOAEL of 5 mg a.i./kg/day, decreases in several immune response parameters are considered secondary to the decreased food consumption, body weight, and food efficiency in the treated rats.

D. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Expected dietary exposure from residues of AVG may occur through the current uses on apple and pear, and the proposed uses on stone fruit. Residue studies conducted with peaches indicate that at proposed label rates, AVG residue levels, if detectable, are below the level of quantitation at harvest. Because of the low rate of application and rapid decline rate, residues in or on treated stone fruit commodities are considered negligible, if detectable at all. However, for risk assessment purposes, maximum anticipated residues were assigned as the limit of quantitation.

ii. *Drinking water*. Residues of AVG are unlikely to occur in drinking water based on its use pattern, low application rates, and expected microbial degradation. There are no registered applications of AVG to water. However, for risk assessment purposes, worst-case assumptions of drift and persistence were incorporated to account for exposure through water consumption.

2. *Non-dietary exposure*. The only non-dietary exposure expected is to applicators. Exposure to AVG resulting from its application according to label directions is not expected to present risks of adverse health or environmental effects, based on its toxicology profile and occupational risk assessment. Non-occupational exposures (home/garden uses) are not applicable to this experimental use permit (EUP).

E. Cumulative Exposure

AVG is a structurally unique biochemical compound and is a naturally-occurring L-amino acid. It does not exhibit a toxic mode of action in its target crops. It is used to regulate the growth and development of the crop. It is used at low application rates and is derived from a naturally-occurring soil microbe. No risks from cumulative exposure have been identified for AVG.

F. Safety Determination

1. *U.S. population*. Based on a NOAEL of 2.2 mg/kg bwt/day from the subchronic toxicity study and an uncertainty factor of 1,000, the U.S. EPA established an RfD of 0.002 mg/kg/day to assess the current time-limited tolerance. For the proposed temporary tolerance on stone fruit, theoretical dietary exposure analyses were conducted using the current RfD and conservative assumptions, such as peach residue values at the LOQ, and 100% of all stone fruit treated. In addition, conservative assumptions of drift and exposure through potable water were included to address water consumption. Results indicated a reasonable certainty of no harm from the use of AVG on stone fruit. The addition of stone fruit to the existing uses on apple and pear totals 5.7% of the RfD for the general U.S. population. The addition of potable water brings the aggregate RfD for the general U.S. population to 7.7%.

2. *Infants and children*. The risks to infants and children have been evaluated based on a developmental study in rats as well as the use of a 10-fold uncertainty factor. Results indicate that there is a reasonable certainty of no harm to infants and children from the use of AVG on stone fruit. Stone fruit plus the existing uses on apple and pear totals 43.8% of the RfD for the most highly exposed sub-population, non-nursing infants less than 1-year old.

G. Effects on the Immune and Endocrine Systems

Abbott Laboratories has no information to suggest that AVG will adversely affect the immune or endocrine systems.

H. Existing Tolerances

U.S. EPA has established a time-limited tolerance to expire April 1, 2001, for the residues of aminoethoxyvinylglycine at a level of 0.08 ppm in apple, and pear commodities, as noted in 40 CFR 180.502.

I. International Tolerances

No international or CODEX MRLs or exemptions have been established for aminoethoxyvinylglycine.

[FR Doc. 99–5817 Filed 3–9–99; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–862; FRL–6063–3]

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–862, must be received on or before April 9, 1999.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 119 at the address

given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: Mary L. Waller, Fungicide Branch, 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. 249, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–6117; e-mail:waller.mary@epamail.epa.gov.

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–862] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket control number [PF–862] 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: February 22, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summary of each petition was prepared by the petitioner and represents the views of the petitioner. 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. American Cyanamid Company

PP 7F4816

EPA has received a pesticide petition (PP 7F4816) from American Cyanamid Company, P.O. Box 400 Princeton, NJ 08543-0400 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of dimethomorph, (E,Z)4-[3-(4-chlorophenyl)-3-(3,4-dimethoxyphenyl)-1-oxo-2-propenyl]morpholine in or on the raw agricultural commodity cereal grains (Crop Group 15) and forage of cereal grain crops (Crop Group 16) at 0.05 parts per million (ppm) and fodder and straw of cereal grain crops (Crop Group 16) at 0.10 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of dimethomorph in plants is adequately understood for the purposes of these tolerances. A rotational crop study showed the potential for indirect or inadvertent residues of dimethomorph in or on commodities of cereal crops.

2. *Analytical method*. There is a practical method for measuring 0.050 ppm of dimethomorph in or on commodities of cereal crops. This gas chromatography method with nitrogen-phosphorus detection (M3112) is appropriate for enforcement purposes. Confirmation of residues is provided by liquid chromatography/mass spectroscopy of the final extract of this method.

3. *Magnitude of residues*. The magnitude of residue studies were conducted for wheat as a rotational crop to potatoes treated at 1.4 x the maximum labeled rate. Residues found in these studies were below the level of quantitation (LOQ) in the forage and grain samples from all six trials and in the hay, and straw samples from four of the trials. The maximum observed residue (sample means) was 0.057 ppm for hay, and 0.086 ppm for straw for the other two trials. Therefore, at the maximum labeled rate, residues of dimethomorph in or on hay are expected to be below the LOQ (< 0.05 ppm) and residues in or on straw are expected to be less than 0.10 ppm.

B. Toxicological Profile

1. *Acute toxicity*. An acute oral toxicity study in the Sprague-Dawley rat for dimethomorph technical with a LD_{50} of 4,300 milligram per kilogram bodyweight (mg/kg bwt) for males and 3,500 mg/kg bwt for females. Based upon EPA toxicity criteria, the acute oral toxicity category for dimethomorph technical is Category III or slightly toxic. Oral LD_{50} studies were conducted on the two isomers (E and Z) alone: An acute oral toxicity study in the Wistar rat for the E-isomer with a LD_{50} greater than 5,000 mg/kg bwt for males and approximately 5,000 mg/kg bwt for females. An acute oral toxicity study in the Wistar rat for the Z-isomer with a LD_{50} greater than 5,000 mg/kg bwt for both males and females. An acute dermal toxicity study in the Wistar rat for dimethomorph technical with a dermal LD_{50} greater than 5,000 mg/kg bwt for both males and females. Based on the EPA toxicity category criteria, the Acute dermal toxicity category for dimethomorph is Category IV or relatively non-toxic. A 4-hour inhalation study in Wistar rats for dimethomorph technical with a LC_{50} greater than 4.2 milligram per liter (mg/L) for both males and females. Based on the EPA toxicity category criteria, the acute inhalation toxicity category for dimethomorph technical is Category IV or relatively non-toxic.

2. *Genotoxicty*. *Salmonella* reverse gene mutation assays (2 studies) were negative up to a limit dose of 5,000 μg/plate. Chinese hamster lung cells were negative in V79 cells up to toxic doses in 2 studies. Two Chinese hamster lung structural chromosomal studies were

reportedly positive for chromosomal aberrations at the highest dose tested (HDT) (160 µg/ml/-S9; 170 µg/ml/+S9). Dimethomorph induced only a weak response in increasing chromosome aberrations in this test system. These results were not confirmed in two micronucleus tests under *in vivo* conditions. Structural Chromosomal Aberration studies were weakly positive, in human lymphocyte cultures, but only in S9 activated cultures treated at the HDT (422 µg/ml) which was strongly cytotoxic. Dimethomorph was negative in the absence of activation at all doses and the positive in human lymphocyte cultures was only in S9 activated cultures treated at the HDT (422 µg/ml) which was strongly cytotoxic. Dimethomorph was negative in the absence of activation at all doses and the positive clastogenic response observed under the *in vitro* conditions was not confirmed in two *in vivo* micronucleus assays. Micronucleus assay (2 studies) indicated that dimethomorph was negative for inducing micronuclei in bone marrow cells of mice following i.p. administration of doses up to 200 mg/kg or oral doses up to the limit dose of 5,000 mg/kg. Thus, dimethomorph was found to be negative in these studies for causing cytogenic damage *in vivo*. Dimethomorph was negative for inducing unscheduled DNA synthesis in cultured rat liver cells at doses up to 250 µ/ml, a weak cytotoxic level. Dimethomorph was negative for transformation in Syrian hamster embryo cells treated in the presence and absence of activation up to cytotoxic concentrations (265 µg/ml/+S9; 50 µg/ml-S9).

3. *Reproductive and developmental toxicity*. A rat developmental toxicity study with a maternal toxicity lowest-observed-adverse-effect Level (LOAEL) of 160 mg/kg/day and a maternal toxicity no-observed adverse-effect level (NOAEL) of 60 mg/kg/day. The NOAEL for developmental toxicity is 60 mg/kg/day. Dimethomorph is not teratogenic in the Sprague-Dawley rat. A rabbit development toxicity study with parental LOAEL for systemic toxicity of 80 mg/kg/day, and a NOAEL of 24 mg/kg/day. The NOAEL for fertility and reproductive function was 80 mg/kg/day, the HDT.

4. *Subchronic toxicity* A 90-day dog dietary study in Sprague-Dawley rats with a NOAEL of greater than or equal to 73 mg/kg/day in males and 82 mg/kg/day in females, the HDT. A 90-day dog dietary study with a NOAEL 15 mg/kg/day, and a LOAEL of 43 mg/kg/day.

5. *Chronic toxicity*. A 2-year oncogenicity study in Sprague-Dawley rats with a NOAEL for systemic toxicity of 9 mg/kg/day for males and 12 mg/kg/day for females. The LOAEL for systemic toxicity is 36 mg/kg/day for males and 58 mg/kg/day for females. A 1-year chronic toxicity study in dogs with a NOAEL of 14.7 mg/kg/day and a LOAEL of 44.6 mg/kg/day. A 2-year oncogenicity study in Sprague-Dawley rats with a NOAEL for systemic toxicity of 9 mg/kg/day for males and 11 mg/kg/day for females. The LOAEL for system toxicity was 34 mg/kg/day for males and 46 mg/kg/ day for females. There was no evidence of increased incidence of neoplastic lesions in treated animals. The NOAEL for oncogenicity is 95 mg/kg/day for males and 132 mg/kg/day for females, the HDT. A 2-year oncogenicity study in mice with a NOAEL for systemic toxicity of 100 mg/kg/day, and LOAEL of 1,000 mg/kg/day. There was no evidence of increased incidence of neoplastic lesions in treated animals. The NOAEL for oncogenicity is 1,000 mg/kg/day, the HDT.

6. *Animal metabolism*. Results from livestock and rat metabolism studies show that orally administered dimethomorph was rapidly excreted by the animals. The principal route of elimination is the feces.

7. *Metabolite toxicology*. There were no metabolites identified in plant or animal commodities which require regulation.

8. *Endocrine disruption*. There is no evidence of effects of dimethomorph on the endocrine system. There were no changes noted in organ weights for the pituitary, thyroid, ovaries or testes. There was no increased incidence of mammary tumors observed. No effects on fertility or reproduction were noted and there was no evidence of related histopathological changes in reproductive or endocrine system organs.

C. Aggregate Exposure

1. *Dietary exposure*. Dietary exposure should be based upon the Theoretical Maximum Residue Concentration (TMRC) from the established tolerances for residues of dimethomorph at 0.05 ppm in or on potato; for the proposed tolerances for residues of dimethomorph at 2.0 ppm in or on grapes; and 0.15 ppm on potatoes wet peel; for the proposed tolerances for indirect and inadvertent residues of dimethomorph at 0.05 ppm in or on cereal grains, and in or on fodder and straw of cereal grain crops, and from the time-limited tolerances (i.e. at 1.0 ppm for cantaloupes, cucumbers, squash, and watermelons) which were established under Section 18 emergency exempt ions and which are not due to expire at or near completion of this regulatory action.

i. *Food*. The goat and poultry metabolism studies demonstrate that there is no reasonable expectation of transfer of residues to meat, milk, poultry, or eggs from potato, grape, and cereal crop commodities. Therefore, no consumption data associated with meat, milk, poultry or eggs should be included in the calculation of the TMRC. Except for the permanent tolerances on potato tubers, there are no other permanent U.S. tolerances for dimethomorph.

ii. *Drinking water*. The predicted dimethomorph surface and ground water concentrations are well below the drinking water level of concern. Using the SCI-GROW model to generate the Estimated Environmental Concentration (EEC) of dimethomorph residues in ground water, the projected EEC is 0.26 parts per billion (ppb). Using the Generic Estimated Environmental Concentration (GENEEC) model to estimate acute and chronic EECs of dimethomorph residues in surface water, the projected EEC ranged from a peak of 28 ppb to a 56 day concentration of 24 ppb. The level of concern for chronic exposure to residues of dimethomorph range from 960 ppb for children 1-6 years old to 3,400 ppb for the U.S. population and males 13 years and older. Therefore, American Cyanamid believes that exposure from water is below the level of concern for all of the populations examined. In addition, American Cyanamid believes that the aggregate (food, and water) chronic exposure for infants, children, and adults does not exceed the level of concern and adverse health effects from chronic exposure to dimethomorph in food, and water are not expected in these populations.

2. *Non-dietary exposure*. In the United States, dimethomorph is registered only for use on potatoes. Thus, there is no potential for non-dietary exposure.

D. Cumulative Effects

There is no information to indicate that any toxic effects produced by dimethomorph would be cumulative with those of any other chemical. The fungicidal mode of action of dimethomorph is unique; dimethomorph inhibits cell wall formation only in Oomycete fungi. The result is lysis of the cell wall which kills growing cells and inhibits spore formation in mature hyphae. This unique mode of action and limited pest spectrum suggest that there is little or no potential for cumulative toxic effects in mammals. In addition, the toxicity studies submitted to support this

petition do not indicate that dimethomorph is a particularly toxic compound.

E. Safety Determination

1. *U.S. population*. The established reference dose (RfD) is 0.1 mg/kg bwt/day, based on a NOAEL of 10 mg/kg bwt/day from a 2-year dietary toxicity study in rats that demonstrated decreased bwt, and liver foci in females. The established RfD is also based on an uncertainty factor of 100. The TMRC from the established tolerances for residues in or on potato along with the current Section 18 time-limited tolerances (cantaloupes, watermelons, cucumbers, and squash, as well as expiring tolerances for tomato commodities) utilizes less than 4% of the RfD for all population subgroups. The TMRC for grapes and cereal grains is not expected to cause the RfD to be exceeded.

2. *Infants and children*. American Cyanamid believes that the results of the studies submitted to support this package provide no evidence that dimethomorph caused reproductive, developmental or fetotoxic effects. No such effects were noted at dose levels which were not maternally toxic. The NOAELs observed in the developmental and reproductive studies were 6 to 65 times higher than the NOAEL (10 mg/kg bwt/day) used to establish the RfD. There is no evidence to indicate that children or infants would be more sensitive than adults to toxic effects caused by exposure to dimethomorph.

F. International Tolerances

No Codex maximum residue levels (MRLs) have been established for dimethomorph to date.

2. BASF Corporation

PP 7F4880

EPA has received a pesticide petition (7F4880) from BASF Corporation, 26 Davis Drive, Post Office Box 13528, Research Triangle Park, North Carolina 27709-3528, proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for combined residues of kresoxim-methyl (methyl (E)-2-methoxyimino-2-[2-(o-tolyloxymethyl) phenyl] acetate) and the glycoside conjugates of its metabolites 2-[o-(o-hydroxymethylphenoxymethyl) phenyl]-2-(methoxyimino) acetic acid and 2-[o-(p-hydroxy-o-methylphenoxymethyl) phenyl]-2-(methoxyimino) acetic acid in or on the raw agricultural commodities pome fruit, grapes and pecans at 0.30 parts per million (ppm) for pome fruit, 1.0 ppm for grapes, 0.15 ppm for pecans and 0.70 ppm for apple pomace. 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*. BASF Corporation notes that metabolism in plants is understood.

2. *Analytical method*. The proposed analytical method involves extraction, enzyme hydrolysis, partition, clean-up and detection of residues by high performance liquid chromotography using ultra-violet (HPLC/UV) detection.

3. *Magnitude of residues*. Twelve grape residue trials were conducted in six States. Residues of kresoxim-methyl and its two metabolites were measured by HPLC/UV. The analytical method had a limit of detection (LOD) of 0.05 ppm for each of the three analytes. Residues ranged from < 0.15 ppm to 0.79 ppm.

Nineteen apple residue trials were conducted in 12 States. Residues of kresoxim-methyl and its two metabolites were measured by HPLC/UV. The analytical method had a LOD of 0.05 ppm for each of the three analytes. Residue of parent and metabolites ranged from < 0.15 to 0.23 ppm.

Eight pear residue trials were conducted in five States. Residues of kresoxim-methyl and its two metabolites were measured by HPLC/UV. The analytical method had a LOD of 0.05 ppm for each of the three analytes. Residues of parent plus metabolites ranged from < 0.15 to 0.26 ppm.

Six pecan residue trials were conducted in five States. Residues of kresoxim-methyl and its two metabolites were measured by HPLC/UV. The analytical method had a LOD of 0.05 ppm for each of the three analytes. No residue of parent or metabolites was found in any sample above the LOD.

B. Toxicological Profile

1. *Acute toxicity*—Acute/subchronic toxicology. Based on available acute toxicity data, kresoxim-methyl does not pose any acute toxicity risks. Acute toxicology studies place technical-grade kresoxim-methyl in Toxicity Category IV for acute oral and Category III for acute dermal and acute inhalation toxicity. The material is not an eye irritant, a primary dermal irritant or a skin sensitizer. Additionally, in acute and subchronic neurotoxicity studies, kresoxim-methyl did not show any signs of neurotoxicity at dose levels up to and including 2,000, and 1,267 milligram/kilogram/day (mg/kg/day), respectively.

2. *Genotoxicty*. With regard to the liver tumors, kresoxim-methyl is not a genotoxic agent and is not an initiator of the carcinogenic process. The increased incidence of liver tumors in rats is the result of liver tumor promoting properties of the test substance.

3. *Reproductive and developmental toxicity*—i. *Reproductive toxicity*. The 2-generation reproduction study with rats resulted in a reproductive no-observed adverse effect level (NOAEL) of 1,625 mg/kg/day, and a maternal NOAEL of 100 mg/kg/day. These NOAEL values are significantly higher than the NOAEL from the 2 year feeding study in rats used to establish the reference dose (RfD).

ii. *Developmental toxicity*. The teratogenicity study in rats resulted in a developmental toxicity NOAEL of 1,000 mg/kg/day, and a maternal toxicity NOAEL of 1,000 mg/kg/day. These NOAEL values are significantly higher than the NOAEL from the 2 year feeding study in rats used to establish the RfD.

4. *Subchronic toxicity*—i. *Acute/subchronic toxicology*. Based on available acute toxicity data, kresoxim-methyl does not pose any acute toxicity risks. Acute toxicology studies place technical-grade kresoxim-methyl in Toxicity Category IV for acute oral and Category III for acute dermal and acute inhalation toxicity. The material is not an eye irritant, a primary dermal irritant or a skin sensitizer. Additionally, in acute and subchronic neurotoxicity studies, kresoxim-methyl did not show any signs of neurotoxicity at dose levels up to and including 2,000 and 1,267 mg/kg/day, respectively.

ii. *Subchronic toxicology*—a. *Teratology - Rat*. A teratogenicity study in the rat with doses at 100, 400, and 1,000 mg/kg/day by gavage was performed with a maternal NOAEL of 1,000 mg/kg/day and fetal NOAEL of 1,000 mg/kg/day.

b. *Teratology - Rabbits*. A teratogenicity study in the rabbit with doses at 100, 400, and 1,000 mg/kg/day by gavage was performed with a maternal NOAEL of 1,000 mg/kg/day and fetal NOAEL of 1,000 mg/kg/day.

c. *Mutagenicity*. Modified Ames Test (2 studies; point mutation): Negative; *In Vitro* chinese hamster ovary hypoxanthine guanine phophoribosyl transferase (CHO/HGPRT) (point

mutation): Negative; *In Vitro* Cytogenetics Chromosome Damage Human Lymphocytes: Negative; *In Vivo* Chromosome Mouse Micronucleus: Negative; *In Vitro* DNA Damage & Repair Rat Hepatocytes: Negative; UDS *ex Vivo* DNA Damage & Repair Wistar Rats (Single Oral Dose): Negative; UDS *ex Vivo* DNA Damage & Repair Wistar Rats (3 Week Feeding): Negative.

5. *Chronic toxicity*—i. *Threshold effects*. Based on review of the available data, BASF believes the RfD for kresoxim-methyl will be based on the 2 year feeding study in rats with a threshold NOAEL of 36 mg/kg/day in males, and 47 mg/kg/day in females. Using an uncertainty factor of 100, the RfD is calculated to be 0.36 mg/kg/day.

ii. *Non-threshold effects - carcinogenicity*. Kresoxim-methyl was shown to be non-carcinogenic in mice. In the rat carcinogenicity study, a statistically significant increase in liver tumors was observed in both male and female animals at 370 and 746 mg/kg/day, and 503 and 985 mg/kg/day dose levels, respectively. Kresoxim-methyl is not a genotoxic agent and mechanistic studies have shown that the increased incidence of liver tumors in rats is the result of liver tumor promoting properties of the test substance. Kresoxim-methyl is not an initiator of the carcinogenic process. Based on the available data, the mechanism of promotion is the induction of liver cell proliferation of the test substance. The data available also indicate that dose levels which do not induce liver toxicity neither induce cell proliferation nor enhance the carcinogenic process. Therefore, a threshold for liver carcinogenicity in rats can be defined to be approximately 40 mg/kg/day.

Based on the results of the carcinogenicity study in mice, the results of genotoxicity testing, the results of the 24 month chronic feeding/oncogenicity study in rats; and auxiliary mechanistic data showing that kresoxim-methyl is not an initiator of the carcinogenic process, BASF believes that the threshold approach to regulating kresoxim-methyl is appropriate.

C. Toxicity Data Supporting Kresoxim-methyl Tolerances

1. *Chronic feeding*—i. *Nonrodent*. A 12 month feeding study in the dog with doses of 29, 142, and 738 mg/kg/day was performed with a NOAEL of 138 mg/kg/day for males, and 761 mg/kg/day for females. The only effect observed was reduced body weights (bwt) in male dogs at the highest dose tested (HDT).

ii. *Chronic feeding/oncogenicity - Rats*. A 24 month chronic feeding/oncogenicity study in the rat with doses at 9, 36, 370, and 746 mg/kg/day for males and 12, 48, 503, and 985 mg/kg/day for females was performed with a NOAEL of 36 mg/kg/day in males, and 47 mg/kg/day in females. Reduced bwt changes were observed in male, and female rats in the highest two dose groups. Histopathologically, changes in the liver were observed in either or both of the highest two dose groups for male, and female rats. These changes consisted of increased liver weight, increased hepatocellular hypertrophy, increased incidence and severity of eosinophilic foci of hepatocellular alterations, and increased incidence and degree of severity of bile duct proliferation. Associated with the liver, an increase of serum-gamma-glutamyltransferase values was observed. A statistically significant increase in liver tumors was observed in both male, and female animals at 370 mg/kg/day and 985 mg/kg/day, respectively. With regard to the liver tumors, kresoxim-methyl is not a genotoxic agent and is not an initiator of the carcinogenic process. The increased incidence of liver tumors in rats is the result of liver tumor promoting properties of the test substance. Based on the available data, the mechanism of promotion is the induction of liver cell proliferation of the test substance. The data available also indicate that dose levels which do not induce liver toxicity neither induce cell proliferation nor enhance the carcinogenic process. Therefore, a threshold for liver carcinogenicity in rats can be defined to be approximately 40 mg/kg/day.

iii. *Oncogenicity - Mice*. A mouse oncogenicity study using dosage levels at 60, 304, and 1,305 mg/kg/day for males, and 81, 410, and 1,662 mg/kg/day for females was performed with a NOAEL of 304 mg/kg/day for males, and 81 mg/kg/day for females, with no evidence of oncogenicity. Bwt changes were observed in both male, and female mice in the highest dose group and only in the females in the 410 mg/kg/day group. Histopathology was limited only to the highest dose group and consisted of increased incidence of renal papillary necrosis for both male, and female mice and increased incidence and higher degree of severity of liver amyloidosis in females only.

iv. *2-Generation reproduction - Rats*. A 2-generation reproductive study in the rat with doses at 5, 100, 407, and 1,625 mg/kg/day was performed with a NOAEL of 100 mg/kg/day for parental and developmental toxicity, and a NOAEL of 1,625 mg/kg/day for reproduction toxicity. Decreased body weight was seen in both the pups and parents. Reduced serum-gamma-glutamyltransferase was seen in F0 males and both sexes of the F1 generation, and reduced kidney weights were seen in the F1 generation at the 407 and 1,625 mg/kg/day dose levels. Decreased fat storage was observed in F0 and F1 male livers at the 407 and 1,625 mg/kg/day dose levels.

6. *Animal metabolism*. BASF Corporation notes that metabolism in animals is understood.

D. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential chronic dietary exposure, BASF has estimated aggregate exposure based on the Theoretical Maximum Residue Contribution (TMRC) from the proposed tolerance for kresoxim-methyl on pome fruit at 0.30 ppm, grapes at 1.0 ppm, and pecans at 0.15 ppm. The TMRC is a ''worse case'' estimate of dietary exposure since it is assumed that 100% of all crops for which tolerances are established are treated and that pesticide residues are always found at the tolerance levels.

i. *Food*. Dietary exposure to residues of kresoxim-methyl in or on food will be limited to residues on pome fruit, grapes, and pecans. Apple pomace is fed to animals; thus exposure of humans to residues in apple pomace might result if such residues carry through to meat, milk, poultry, or eggs. However, BASF has concluded that there is no reasonable expectation that measurable residues of kresoxim-methyl will occur in meat, milk, poultry, or eggs from this use. There are no other established U.S. tolerances for kresoxim-methyl, and there are no currently registered uses for kresoxim-methyl on food or feed crops in the U.S.

Dietary exposure to residues of kresoxim-methyl from the proposed tolerances on pome fruit, grapes, and pecans would account for less than 0.15% of the RfD (.36 mg/kg/day) for the general population of the U.S. The most highly exposed group in the subpopulation groups would be non-nursing infants < 1 year old, which uses 0.88% of the RfD.

ii. *Drinking water*. Other potential sources of exposure for the general population to residues of kresoxim-methyl are residues in drinking water and exposure from non-occupational sources. Based on the available studies, BASF does not anticipate exposure to residues of kresoxim-methyl in drinking water. There is no established Maximum Concentration Level (MCL) for residues of kresoxim-methyl in

drinking water under the Safe Drinking Water Act (SDWA).

2. *Non-dietary exposure*. Kresoxim-methyl is currently registered for use in greenhouses on ornamental plants. The potential for non-occupational exposure to the general population is not significant.

E. Cumulative Effects

BASF has considered the potential for cumulative effects of kresoxim-methyl and other substances that have a common mechanism of toxicity. No evidence or information exists to suggest that toxic effects produced by kresoxim-methyl would be cumulative with those of any other chemical compound.

F. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the completeness and the reliability of the toxicity data, BASF has estimated that aggregate exposure to kresoxim-methyl will utilize less than 0.15% of the RfD for the total U.S. population. BASF concludes that there is a reasonable certainty that no harm will result from the aggregate exposure to residues of kresoxim-methyl, including anticipated dietary exposure and non-occupational exposures.

2. *Infants and children*—i. *Developmental toxicity*. The teratogenicity study in rats resulted in a developmental toxicity NOAEL of 1,000 mg/kg/day, and a maternal toxicity NOAEL of 1,000 mg/kg/day. These NOAEL values are significantly higher than the NOAEL from the 2 year feeding study in rats used to establish the RfD.

The teratogenicity study in rabbits resulted in a developmental toxicity NOAEL of 1,000 mg/kg/day, and a maternal toxicity NOAEL of 1,000 mg/kg/day. These NOAEL values are significantly higher than the NOAEL from the 2 year feeding study in rats used to establish the RfD.

ii. *Reproductive toxicity*. The 2-generation reproduction study with rats resulted in a reproductive NOAEL of 1,625 mg/kg/day, and a maternal NOAEL of 100 mg/kg/day. These NOAEL values are significantly higher than the NOAEL from the 2 year feeding study in rats used to establish the RfD.

iii. *Reference Dose*. Since developmental and reproductive toxicity occurs at levels at or above the levels shown to exhibit parental toxicity and since these levels are significantly higher than those used to calculate the RfD, BASF believes the RfD of 0.36 mg/kg/day is an appropriate measure of safety for infants and children.

Using the conservative exposure assumptions described above, BASF has concluded that the portion of the RfD that will be utilized by aggregate exposure to residues of kresoxim-methyl resulting from the proposed tolerances will be less than 1% for all populations of infants and children. The most highly exposed group in the subpopulation groups would be non-nursing infant < 1-year old, which uses 0.88% 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 to infants and children from aggregate exposure to the residues of kresoxim-methyl, including all anticipated dietary exposure and all other non-occupational exposures.

G. International Tolerances

A maximum residue level has not been established for kresoxim-methyl by the Codex Alimentarius Commission.

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BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPP–181067; FRL 6066–3]

Bifenthrin; Receipt of Application for Emergency Exemption, Solicitation of Public Comment

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has received a specific exemption request from the Washington Department of Agriculture (hereafter referred to as the ''Applicant'') to use the pesticide bifenthrin (CAS 8657–04–3 *cis* and 83322–02–5 *trans*), formulated as Brigade WSB, to treat up to 8,500 acres of raspberries to control weevils. This is the seventh year this use has been requested, and it has been allowed under section 18 for the past 6 years. Since this request proposes a use which has been requested or granted in any 3 previous years, and a complete application for registration and petition for tolerance has not yet been submitted to the Agency, EPA is soliciting public comment before making the decision whether or not to grant the exemption, in accordance with 40 CFR 166.24(a)(6).

DATES: Comments must be received on or before March 25, 1999.

ADDRESSES: Three copies of written comments, bearing the identification notation (OPP–181067), should be submitted by mail to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring comments to: Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Follow the instructions under SUPPLEMENTARY INFORMATION. No Confidential Business Information (CBI) should be submitted through e-mail.

Information submitted in any comment concerning this notice may be claimed confidential by marking any part or all of that information as CBI. Information so marked 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 provided by the submitter for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. The docket is available for public inspection at the Virginia address given above, 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Andrea Beard, 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. 271, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, (703–308–9356); e-mail: beard.andrea@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: Pursuant to section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) (7 U.S.C. 136p), the Administrator may, at her discretion, exempt a state agency from any registration provision of FIFRA if she determines that emergency conditions exist which require such exemption. The Applicant has requested the Administrator to issue a specific exemption for the use of bifenthrin on raspberries to control weevils. Information in accordance with 40 CFR part 166 was submitted as part of this request.

According to the Applicant, this emergency exists because of the loss of the chlorinated hydrocarbon insecticides. Initially, raspberry growers obtained some relief through use of carbofuran under an exemption; however, that use was later disallowed due to groundwater concerns.