statement that this chemical demonstrates the properties associated with chemicals detected in groundwater, the Registrant is not aware of imidacloprid being detected in any wells, ponds, lakes, streams, etc. from its use in the United States. In studies conducted in 1995, imidacloprid was not detected in seventeen wells on potato farms in Quebec, Canada. In addition, groundwater monitoring studies are currently underway in California and Michigan. Therefore, contributions to the dietary burden from residues of imidacloprid in water would be inconsequential.

4. *Non-dietary exposure*— a. *Residential turf*. Bayer Corporation has conducted an exposure study to address the potential exposures of adults and children from contact with imidacloprid treated turf. The population considered to have the greatest potential exposure from contact with pesticide treated turf soon after pesticides are applied are young children. Margins of safety (MOS) of 7,587 - 41,546 for 10 year old children and 6,859 - 45,249 for 5 year old children were estimated by comparing dermal exposure doses to the imidacloprid no-observable effect level of 1,000 mg/kg/day established in a 15-day dermal toxicity study in rabbits. The estimated safe residue levels of imidacloprid on treated turf for 10 year old children ranged from 5.6 - 38.2 g/cm^2 and for 5 year old children from 5.1 - 33.3 g/cm^2. This compares with the average imidacloprid transferable residue level of 0.080 g/cm^2 present immediately after the sprays have dried. These data indicate that children can safely contact. Bayer Corporation has conducted an exposure imidacloprid-treated turf as soon after application as the spray has dried.

b. *Termiticide*. Imidacloprid is registered as a termiticide. Due to the nature of the treatment for termites, exposure would be limited to that from inhalation and was evaluated by EPA's Occupational and Residential Exposure Branch (OREB) and Bayer Corporation. Data indicate that the Margins of Safety for the worst case exposures for adults and infants occupying a treated building who are exposed continuously (24 hours/day) are 8.0 x 10^7 and 2.4 x 10^8, respectively, and exposure can thus be considered negligible.

c. *Tobacco smoke*. Studies have been conducted to determine residues in tobacco and the resulting smoke following treatment. Residues of imidacloprid in cured tobacco following treatment were a maximum of 31 ppm (7 ppm in fresh leaves). When this tobacco was burned in a pyrolysis study only two percent of the initial residue was recovered in the resulting smoke (main stream plus side stream). This would result in an inhalation exposure to imidacloprid from smoking of approximately 0.0005 mg per cigarette. Using the measured subacute rat inhalation NOEL of 5.5 mg/m^3, it is apparent that exposure to imidacloprid from smoking (direct and/or indirect exposure) would not be significant.

d. *Pet treatment*. Human exposure from the use of imidacloprid to treat dogs and cats for fleas has been addressed by EPA's Occupational and Residential Exposure Branch (OREB) who have concluded that due to the fact that imidacloprid is not an inhalation or dermal toxicant and that while dermal absorption data are not available, imidacloprid is not considered to present a hazard via the dermal route.

D. Cumulative Effects

No other chemicals having the same mechanism of toxicity are currently registered, therefore, there is no risk from cumulative effects from other substances with a common mechanism of toxicity.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, it can be concluded that total aggregate exposure to imidacloprid from all current uses including those currently proposed will utilize little more than 15% of the RfD for the U.S. population. EPA generally has no concerns for exposures below 100% of the RfD, because the RfD represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of imidacloprid, the data from developmental studies in both rat and rabbit and a 2-generation reproduction study in the rat have been considered. The developmental toxicity studies evaluate potential adverse effects on the developing animal resulting from pesticide exposure of the mother during prenatal development. The reproduction study evaluates effects from exposure to the pesticide on the reproductive capability of mating animals through two generations, as well as any observed systemic toxicity.

FFDCA Section 408 provides that the EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for imidacloprid relative to pre- and post-natal effects is complete. Further for imidacloprid, the NOEL of 5.7 mg/kg/bwt from the 2-year rat feeding/carcinogenic study, which was used to calculate the RfD (discussed above), is already lower than the NOELs from the developmental studies in rats and rabbits by a factor of 4.2 to 17.5 times. Since a 100-fold uncertainty factor is already used to calculate the RfD, it is surmised that an additional uncertainty factor is not warranted and that the RfD at 0.057 mg/kg/bwt/day is appropriate for assessing aggregate risk to infants and children. Using the conservative exposure assumptions described above, EPA has concluded that the TMRC from use of imidacloprid from published uses is 0.008358 mg/kg/bwt/day utilizing 14.7% of the RfD for the general population. For the most highly exposed subgroup in the population, non-nursing infants (less than 1 year old), the TMRC for the published tolerances is 0.01547 mg/kg/day. This is equal to 27.1% of the RfD. Therefore, dietary exposure from the existing uses including the currently proposed tolerances will not exceed the reference dose for any subpopulation (including infants and children).

F. International Tolerances

No CODEX Maximum Residue Levels (MRLs) have been established for residues of imidacloprid on any crops at this time.

[FR Doc. 97–28663 Filed 10–28–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–771; FRL–5749–7]

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–771, must be received on or before November 28, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), 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 to: opp-docket@epamail.epa.gov. Follow 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: Sidney C. Jackson, Registration Division (7505C), Office of Pesticide Programs, 401 M St., SW., Washington, DC 20460. Office location and telephone number, Rm. 274, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA., 703–305–7610, e-mail: jackson.sidney@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–771] (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 6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF-771] 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: October 22, 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. IR-4 Project

PP 2E4044 and 3E4164

EPA has received pesticide petitions (PP 2E4044 and 3E4164) from the Interregional Research Project number 4 (IR-4), 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 tolerances for residues of Triadimefon, 1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1*H*-1,2,4-triazol-1-yl)-2-butanone, and its metablolites containing chlorophenoxy and triazole moieties expressed as the fungicide in or on the raw agricultural commodities artichoke, globe at 0.6 parts per million (ppm) and pome fruits group (Crop Group 11) at 0.2 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition. This notice includes a summary of each petition prepared by the Bayer Corporation(Bayer), the registrant.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue in plants and animals is adequately understood. The residue of concern is triadimefon and its triazole and chlorophenoxy metabolites. Triadimefon is rapidly absorbed by plants and translocated systemically in the young growing tissue.

2. *Analytical method*. Adequate analytical methods are available for analysis of triadimefon and its triazole and chlorophenoxy metabolites in or on artichokes. These methods are available in PAM II as Method I.

3. *Magnitude of residues*. Three separate residue trials have been conducted on globe artichokes and submitted to the EPA. The EPA has determined that these data show that residues of triadimefon and its metabolites containing chlorophenoxy and triazole moieties (expressed as the fungicide) in the raw agricultural commodity artichokes, globe will not exceed the proposed tolerance of 0.6 ppm.

For pome fruits and as part of the reregistration requirements for triadimefon, Bayer has submitted nine trials on apples and six trials on pears to the EPA. EPA's Chemistry Branch Tolerance Support has concluded that these data are adequate to support the requested crop group tolerance for triadimefon and its metabolites containing chlorophenoxy and triazole moieties expressed as the fungicide in or on pome fruit at 0.2 ppm.

There are no livestock feed stuffs from globe artichokes and pome fruits, therefore, secondary residues in meat, milk, poultry and eggs are not expected.

B. Toxicological Profile

1. *Acute toxicity*. A rat acute oral study resulted in a lethal dose (LD_{50}) of 568 ± 61 milligrams (mg)/kilogram (kg)

for males and 363 ± 41 mg/kg for females. In a rabbit acute dermal study a LD_{50} of >2,000 mg/kg was determined. A rat acute inhalation study produced a lethal concentration (LC_{50}) of >3.570 mg/liter(l). A primary eye irritation study in the rabbit showed practically no irritation. A primary dermal irritation study showed practically no irritation and a primary dermal sensitization study indicated that triadimefon is a skin sensitizer.

2. *Genotoxicity*. Triadimefon has been found to be negative in the Ames reverse mutation test and in the Structural Chromosome Aberration Test.

3. *Reproductive and developmental toxicity*. A rat developmental toxicity study showed a maternal systemic no-observed-effect level (NOEL) of 30 mg/kg/day and the lowest-observed-effect level (LOEL) 90 mg/kg/day. The NOEL for developmental toxicity was 30 mg/kg/day and the LOEL was 90 mg/kg/day.

In the developmental toxicity study in rabbits, the maternal systemic NOEL was 50 mg/kg/day and the LOEL 120 mg/kg/day. The NOEL for developmental toxicity was 20 mg/kg/day and the LOEL was 50 mg/kg/day. Effects seen at the developmental lowest effect level(LEL) in the rabbit study were irregular spinous process and ossification of various bones.

A 3-generation rat reproduction study showed decreases in maternal body weight gain, fertility, and in litter size, pups survival during the lactation phase, and pups weights. The maternal NOEL was 300 ppm and the reproductive NOEL was 50 ppm.

A 2-generation rat reproductive study showed reductions in litter size, pups viability, birth and lactational weights. The reproductive NOEL was 50 ppm.

4. *Subchronic toxicity*. A 3-month feeding study in the rat produced a NOEL of 2,000 ppm based on decreased body weight gain and food consumption attributed to palatability. A rat 30-day feeding study showed a NOEL of 10 mg/kg. A 13-week dog-feeding study resulted in a NOEL of 2,400 ppm based on decreased body weight gain and food consumption due to palatability. Test results also showed a decreased hematocrit, RBC count, hemoglobin volume and microsomal induction. A 28-day rabbit dermal study produced a NOEL >250 mg/kg and a 21-day inhalation study in rats showed a NOEL of 78.7 mg/cubic meters(m^3)/6 hrs. per day/ 15 exposures.

5. *Chronic toxicity*. A 2–year rat chronic feeding study defined a NOEL for systemic effect as 300 ppm (males = 16.4 mg/kg/day; females = 22.5 mg/kg/day). The systemic LOEL was 1,800 ppm (males = 114.0 mg/kg/day; females = 199.0 mg/kg/day) based on neoplastic and systemic effects. A dog feeding study showed only minimal toxic effects decrease in body weight, increase in liver weight and in hepatic N-demethylase activity, and an increase in serum alkaline phosphatase activity. The NOEL was established at 100 ppm. A mouse oncogenicity study showed hepatocellular adenomas in both sexes of NMRI mice. The NOEL was established for males at 50 ppm. No NOEL was reached for females. A mouse carcinogenicity study using CF1-W74 mice was negative for carcinogenicity.

6. *Animal metabolism*. In a general rat metabolism study triadimefon was initially converted by reduction of its carbonyl group. This conversion was more rapid in males. The major metabolites were the acid and alcohol of triadimefon. In males radioactivity was found mainly in feces, whereas, in females, radioactivity was equally distributed between urine and feces. No radioactivity was recovered in the expired air. Peak tissue levels were found in 2 to 4 hours and were highest in fat, liver and kidney.

7. *Endocrine effects*. No special studies investigating potential estrogenic or endocrine effects of triadimefon have been conducted. However, the standard battery of required studies has been completed. These studies include an evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure. No adverse effects were noted in any of the studies with either triadimefon or its metabolites.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure from food under the proposed tolerances, the EPA estimates exposure based on the Theoretical Maximum Residue Contribution (TMRC). The TMRC is obtained by using a model which multiplies the tolerance level residue for each commodity by consumption data which estimates the amount of each commodity and products derived from the commodities that are eaten by the U.S. population and various population subgroups. The model uses a reference dose (RfD) which the EPA has determined to be 0.04 milligrams(mg)/ kilogram(kg)/day. This RfD is based on a 2–year dog feeding study with a NOEL of 11.4 mg/kg/day and an uncertainty factor of 300. An uncertainty factor of 300 was applied to account for inter-species extrapolation (10), intra-species variability (10), and the lack of an adequate reproduction study (3). Decreased food intake, depression in weight gain, and significantly (p >0.05) increased alkaline phosphatase activity in both sexes were the effects observed at the lowest effect level (LEL). This assessment assumes 100% of all commodities will contain triadimefon residues, and those residues would be at the level of the tolerance for estimating potential human exposure.

2. *Food*. Using assumptions discussed above, it was determined that the TMRC for existing tolerances plus the proposed uses on globe artichokes and pome fruits. For globe artichokes, the TMRC is equivalent to 17% of the RfD for the US general population (48 states) and 74% of the RfD for the highest population subgroup (non-nursing infants >1 year old).

For pome fruits, the TMRC for triadimefon derived from the previously established tolerances plus the proposed 0.2 ppm tolerance for this crop group (pome fruit) would be 0.003782 mg/kg body weight(bwt)/day (9.5% of the RfD) for the U.S. population 48 states and 0.009549 mg/kg bwt/day (23.9% of the RfD) for the most highly exposed population subgroup, children (1-6 year old). Therefore, Bayer concludes that dietary exposure from the existing and proposed uses will not exceed the reference dose for any subpopulation including infants and children.

For globe artichoke, the estimated acute dietary exposure is based on a maternal NOEL of 10 mg/kg/day. The calculated Margin of Exposure (MOE) for the general US population is 100 (at the 99th percentile); for infants (>1 year old) 100 (at the 95th percentile); for children (1-6 year old) 200 (at the 96th percentile); and for both females (13+ years) and males (13+ years) 333 (at the 99th percentile). These values are all at or above the MOE level EPA considers to provide an adequate safety margin (100).

3. *Drinking water*. Available data show that triadimefon and its metabolites are mobile and persistent and have the potential to leach into groundwater. There is no established Maximum Concentration Level for residues of triadimefon in drinking water. No drinking water health advisory levels have been issued for triadimefon or its metabolite triadimefon. The ''Pesticides in Groundwater Database'' (EPA 734–12–92–001, September 1992) indicated that triadimefon was monitored for in 14 wells in California from 1984 to 1989. There were no detectable residues (limit of detection was not stated).

Previous experience with more persistent and mobile 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 maximum residue levels (MCL's). Best scientific judgement from available data suggests that the potential exposure from residues of triadimefon in drinking water, added to the current dietary exposure, will not result in an exposure which exceeds the RfD.

4. *Non-dietary exposure*. Triadimefon is currently registered for use on turf and ornamentals. Studies were conducted by Bayer designed to measure the upper bound acute exposure potential of adults and children from contact with triadimefon treated turf. The population considered to have the greatest potential exposure from contact with pesticide treated turf soon after pesticides are applied are young children. The estimated safe residue levels for triadimefon on treated turf for 10-year old children ranged from 1.3 - 6.4 micro gram(μg)/centimeter(cm)^2 and for 5-year old children from 1.1 - 5.6 μg/cm^2. This compares with the average triadimefon transferable residue level of 1.0 μg/cm^2 present immediately after the sprays have dried. Bayer concludes from these studies that children can safely contact triadimefon-treated turf as soon after application as the spray has dried.

D. Cumulative Effects

At this time, the Agency has not made a determination that triadimefon and other substances that may have a common mode of toxicity would have cumulative effects. For purposes of this tolerance, only the potential risks of triadimefon in its aggregate exposure are being considered.

E. Safety Determination

1. *U.S. population*. Using the exposure assumptions described above under aggregate exposure and based on the toxicity data, Bayer concludes that aggregate dietary exposure to triadimefon from the previously established tolerances plus the proposed use on globe artichoke will utilize 17% of the RfD for the U.S. population (48 states) and 74% of the RfD for the most highly exposed population subgroup (non-nursing infants >1 year old). In comparison, pome fruit will vitilize 9.5% and 23.9% of the RfD for the same U.S. population and for children (1-6 yrs), respectively. There is generally no concern for exposures below 100 percent of the RfD because the RfD represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. Bayer concludes that there is a reasonable certainty that no harm will result from aggregate exposure to triadimefon.

Bayer estimated acute dietary exposure using the maternal NOEL of 10 mg/kg/day and determined that the calculated MOE for each population group is at or above the MOE level EPA considers to provide an adequate safety margin.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of triadimefon, the data from developmental studies in both rat and rabbit and a 2-generation reproduction study in the rat were considered. The developmental toxicity studies evaluate any potential adverse effects on the developing animal resulting from pesticide exposure of the mother during prenatal development. The reproduction study evaluates any effects from exposure to the pesticide on the reproductive capability of mating animals through 2-generations, as well as any observed systemic toxicity.

Results of a rat and rabbit developmental toxicity studies and a 2-generation and 3-generation rat reproduction studies conducted with triadimefon have been reviewed. Maternal and developmental toxicity NOELs of 30 mg/kg/day were determined in the rat developmental toxicity studies. In the rabbit developmental toxicity study, the maternal NOEL was 50 mg/kg body weight(bwt)/day and the developmental NOEL was 20 mg/kg bwt/day. The rat reproduction studies were inconclusive.

FFDCA Section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal effects and the completeness of the toxicity database. Therefore, EPA has incorporated an additional 3-fold uncertainty factor into the calculation of the RfD because of the absence of an acceptable reproduction study.

There is approximately a two-fold difference between the developmental NOEL of 20 mg/kg/day from the rabbit developmental toxicity study and the NOEL of 11.4 mg/kg/day from the 2–year dog feeding study which was the basis of the RfD. It is further noted that in the rabbit developmental toxicity study, the developmental NOEL of 20 mg/kg/day is lower than the maternal systemic NOEL of 50 mg/kg/day, suggesting the possibility of increased sensitivity for the pre-natal child.

The TMRC value for the most highly exposed infant and children subgroup (non-nursing infants >1 year old) occupies 74% of the RfD. However, this calculation also assumes 100% crop treated and uses tolerance level residues for all commodities. Refinement of the dietary risk assessment by using percent of crop treated and anticipated residue data would likely greatly reduce the dietary exposure estimate and result in an anticipated residue contribution (ARC) which would occupy a percent of the RfD that is substantially lower than the currently calculated TMRC value.

Should an additional uncertainty factor be deemed appropriate, when considered in conjunction with a refined exposure estimate, Bayer believes it is unlikely that the dietary risk will exceed 100 percent of the RfD. Due to the completeness and reliability of the toxicity data and the exposure assessment, Bayer believes there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to triadimefon residues.

Bayer estimated acute dietary exposure using the maternal NOEL of 10 mg/kg/day and determined that the calculated MOE for infants and children population groups is at or above the MOE level EPA considers to provide an adequate safety margin.

F. International Tolerances

There are no CODEX, Canadian, or Mexican MRLs for triadimefon residues in/on globe artichokes. A CODEX MRL for triadimefon residues in/on pome fruits has been established at 0.5 ppm.

2. IR-4 Project

PP 6E4652

EPA has received a pesticide petition (PP 6E4652) from the Interregional Research Project number 4 (IR-4), 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 the combined residues of quizalofop-p ethyl ester [ethyl (*R*)-(2-[4-((6-chloroquinoxalin-2-yl) oxy)phenoxy])-propanoate), and its acid metabolite quizalofop-p [*R*-(2-[4-((6-chloroquinoxalin-2-yl)oxy)phenoxy]) propanoic acid), and the S enantiomers of both the ester and the acid, all expressed as quizalofop-p ethyl ester in or on the raw agricultural commodities spearmint tops and peppermint tops at 3.0 parts per million(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 support granting of the petition. Additional data may be needed before EPA rules on the petition. This notice includes a summary of the petition prepared by the DuPont Agricultural Products(DuPont), the registrant.

A. Residue Chemistry

1. *Plant metabolism*. The registrant has provided plant metabolism studies for soybeans, cotton, tomatoes, potatoes, and sugar beets. These studies have been previously reviewed in PP 3F4268. In summary, quizalofop-p ethyl ester is metabolized by cleavage at three sites as follows: (a) Primary pathway is hydrolysis of the ethyl ester to form the quizalofop-p acid, then (b) cleavage of the enol ether linkage in the acid, between the phenyl and quinoxalinyl rings, to form phenols, and (c) cleavage of the ether linkage between the isopropanic group and the phenyl ring to form a phenol.

The plant metabolism data show that quizalofop-p ethyl ester does not translocate, but is rapidly hydrolyzed to the corresponding acid; then the phenols conjugate with the plant sugars. Metabolism studies in soybeans using the racemic mixture quizalofop ethyl ester and the resolved D+ isomer show nearly identical pathways.

The nature of the quizalofop-p ethyl ester residue in cottonseed, potatoes, tomatoes, soybeans, and sugar beets is adequately understood. The residues of concern are quizalofop-p ethyl ester and its acid metabolite, quizalofop-p, and the S enantiomers of both the ester and the acid, all expressed as quizalofop-p ethyl ester. EPA is translating these data to mint.

2. *Analytical method*. An adequately validated residue analytical method, LAN-1, was used to gather the magnitude of the quizalofop-p, its acid metabolite, and residue data on mint hay and mint oil. Samples were analyzed using MS30.00, an adaptation of Analytical Method for the Quantification of Quizalofop (IN-YE945) and Quizalofop-Ethyl (DPX-79379) in Raw and Processed Agricultural Commodities, Protocol No. Lan-1, Enviro-Test Laboratory. (Reference Method: Determination of DPX-79376, DPX-79376 Acid and Conjugates as DPX-79376 as Acid in Cottonseed and Fractions Treated with Assure (II Herbicide. DuPont Report No. AMR 1853–90).

3. *Magnitude of residues*. The maximum residues detected on fresh mint foliage at the proposed labeled level of DuPont's product, Assure, of 0.2 pounds(lbs) active ingredient(ai) acre (1x) applied 30 days before harvest were 0.22, 0.46, and 1.0 ppm for Indiana, Oregon and Washington, respectively. The largest residue found on fresh mint foliage, 2.6 ppm, was detected in a Washington sample treated with 0.4 lbs. acre (2x) 29 days before harvest, twice the maximum yearly rate allowed. At the Level of Quantitation of 0.05 ppm, there were no detectable residues in the mint oil, either at the proposed label rate of 0.2 lbs. ai/acre(A), or at the exaggerated rate of 0.4 lbs. ai/A, indicating that quizalofop-p ethyl and its acid metabolite are not concentrated during the oil distillation process.

Results of a freezer storage stability study demonstrated that the two compounds, quizalofop-p ethyl ester and quizalofop acid, were stable in frozen storage at -20 degrees centigrade for 592 to 593 days in mint hay, and 597 days in oil. Field samples were stored a maximum of 654 days.

The residues detected in this study are well below the proposed tolerances of 3.0 ppm for the raw agricultural commodity mint. The nature of the residues is adequately understood and an adequate analytical method is available for enforcement purposes. Based on the information presented above, Dupont believes the establishment of the proposed tolerance would protect the public health and would not expose man or the environment to unreasonable adverse effects.

B. Toxicological Profile

1. *Acute toxicity*. Several acute toxicology studies were conducted and the overall results placed technical grade quizalofop ethyl in toxicity Category III. These include the following studies in Category III: acute oral toxicity (LD_{50}s 1,480 and 1,670 for female and male rats, respectively) and eye irritation (mild effects; reversible within 4 days). Dermal toxicity (lethal dose) LD_{50} >5,000 milligram(mg)/kilogram(kg); rabbit), inhalation toxicity (lethal concentration) LC_{50} >5.8 mg/liter(L); rat) and dermal irritation were classified within Category IV. Technical quizalofop ethyl was not a dermal sensitizer.

2. *Genotoxicity*. Technical quizalofop ethyl was negative in the following genotoxicity tests: bacterial gene mutation assays with *E. coli* and *S. typhimurium*; gene mutation assays in Chinese hamster ovary(CHO) cells ; *in vitro* DNA damage assays with B. subtillis and in rat hepatocytes; and an *in vitro* chromosomal aberration test in CHO cells.

3. *Reproductive and developmental toxicity*. Studies supporting the registration include: A developmental toxicity study in rats administered dosage levels of 0, 30, 100, and 300 mg/kg/day (HDT). The maternal toxicity no-observed-effect level (NOEL) was 30 mg/kg/day and a developmental toxicity NOEL was greater than 300 mg/kg/day (HDT). The maternal NOEL was based on reduced food consumption and increased liver weights.

A developmental toxicity study in rabbits administered dosage levels of 0, 7, 20, and 60 mg/kg/day with no developmental effects noted at 60 mg/kg/day (HDT). The maternal toxicity NOEL was 20 mg/kg/day based on decreases in food consumption and body weight gain at 60/mg/kg/day (HDT).

A 2-generation reproduction study in rats fed diets containing 0, 25, 100 or 400 ppm (or approximately 1, 1.25, 5, and 20 mg/kg/day, respectively) with a developmental (systemic effects) NOEL of 1.25 mg/kg/day for F2B weanlings based on increased liver weights and increased incidence of eosinophilic changes in the livers at 5.0 mg/kg/day. These liver changes were considered to be physiological or adaptive changes to compound exposure among weanlings. When access to the mother's feed is available, it is a common observation that young rats will begin consuming chow prior to complete weaning at 21-days of age. Consumption could not be quantified; therefore, the maternal consumption was assumed as the NOEL (if normalized on a body weight basis, exposures to the weanling rats were likely higher). The parental NOEL of 5.0 mg/kg/day was based on decreased body weight and premating weight gain in males at 20 mg/kg/day (HDT).

4. *Subchronic toxicity*. A 90-day study was conducted in rats fed diets containing 0, 40, 128, 1,280 ppm (or approximately 0, 2, 6.4 and 64 mg/kg/day, respectively). The NOEL was 2 mg/kg/day. This was based on increased liver weights at 6.4 mg/kg.

A 90–day feeding study in mice was conducted with diets that contained 0, 100, 316 or 1,000 ppm (or approximately 0, 15, 47.4, and 150 mg/kg/day, respectively). The NOEL was >15 mg/kg/day Lowest Dose Tested (LDT) based on increased liver weights and reversible histopathological effects in the liver at the LDT. A 6-month feeding study in dogs was conducted with diets that contained 0, 25, 100 or 400 ppm (or approximately 0, 0.625, 2.5, and 10 mg/kg/day, respectively). The NOEL was 2.5 mg/kg/day based on increased blood urea nitrogen at 10 mg/kg/day. A 21–day dermal study was conducted in rabbits at doses of 0, 125,

500 or 2,000 mg/kg/day. The NOEL was 2,000 mg/kg/day (HDT).

5. *Chronic toxicity*. An 18-month carcinogenicity study was conducted in CD-1 mice fed diets containing 0, 2, 10, 80 or 320 ppm (or approximately 0, 0.3, 1.5, 12, and 48 mg/kg/day, respectively). There were no carcinogenic effects observed under the conditions of the study at levels up to and including 12 mg/kg/day. A marginal increase in the incidence of hepatocellular tumors was observed at 48 mg/kg/day, the highest dose tested (HDT) which exceeded the maximum tolerated dose (MTD).

A 2–year chronic toxicity/carcinogenicity study was conducted in rats fed diets containing 0, 25, 100 or 400 ppm (or 0, 0.9, 3.7, and 15.5 mg/kg/day for males and 0, 1.1, 4.6, and 18.6 mg/kg/day for females, respectively). There were no carcinogenic effects observed under the conditions of the study at levels up to and including 18.6 mg/kg/day (HDT). The systemic NOEL was 0.9 mg/kg/day based on altered red cell parameters and slight/minimal centrilobuler enlargement of the liver at 3.7 mg/kg/day.

A 1–year feeding study was conducted in dogs fed diets containing 0, 25, 100 or 400 ppm (or approximately 0, 0.625, 2.5, and 10 mg/kg/day, respectively). The NOEL was 10 mg/kg/day (HDT).

The Carcinogenicity Peer Review Committee (CPRC) of the EPA has evaluated the rat and mouse cancer studies on quizalofop along with other relevant short-term toxicity studies, mutagenicity studies, and structure activity relationships. The CPRC concluded, after three meetings and an evaluation by the EPA Science Advisory panel, that the classification should be a Category D (not classifiable as to human cancer potential). No new cancer studies were required.

The first CPRC review tentatively concluded that quizalofop should be classified as a Category B2 (probable human carcinogen). That classification was based on liver tumors in female rats, ovarian tumors in female mice, and liver tumors in male mice. This classification was downgraded to a Category C (possible human carcinogen) at a second CPRC review. The change in classification was due to a reexamination of the liver tumors in female rats and ovarian tumors in female mice. The first peer review had found a statistically significant positive trend for liver carcinomas in female rats. Subsequent to this conclusion the tumor data were reevaluated, and the revaluation showed a reduced number of carcinomas. Although there remained a statistically significant positive trend for carcinomas in the study, the CPRC concluded that the carcinomas were not biologically significant given the few carcinomas identified (one at the mid-dose and two at the high dose). Noting that this level of carcinomas was within historical levels, the CPRC concluded that administration of quizalofop did not appear to be associated with the liver carcinomas.

As to the ovarian tumors in female mice, the CPRC had first attached importance to the fact that these tumors were statistically significant at the high dose as compared to historical control values although statistically significant when compared to concurrent controls. However, review of further historical control data showed that the level of ovarian tumors in the quizalofop study was similar to the background rate in several other studies. Given this information and that the quizalofop study showed no hyperplasia of the ovary, no signs of endocrine activity related to ovarian function, and no dose response relationship, the CPRC concluded that the ovarian tumors were probably not compound-related.

The findings of the second CPRC review were presented to EPA's Scientific Advisory Panel (SAP). The SAP concurred with the CPRC conclusion that the liver tumors in female rats and the ovary tumors in female mice showed no evidence of carcinogenicity. However, the SAP disagreed with CPRC's classification of quizalofop as a Category C based on the liver tumors in male mice. The SAP concluded that the mouse liver tumors did not support such a classification because the tumors occurred at a dose above the maximum-tolerated dose (MTD) and because they were not statistically significant if a ''p''value of less than 0.05. The SAP believed that such greater statistical rigor was appropriate for variable tumor endpoints such as male mouse liver tumors.

Following the SAP review, the CPRC changed the classification for quizalofop to Category D. The Category D classification is based on an approximate doubling in the incidence of male mice liver tumors between controls an the high dose. This finding was not considered strong enough to warrant the finding of a Category C (possible human carcinogen) since the increase was of marginal statistical significance, occurred at a high dose which exceeded the predicted MTD, and occurred in a study in which the concurrent control for liver tumors was somewhat low as compared to the historical controls, while the high dose control group was at the upper end of previous historical control-groups.

EPA has found the evidence on the carcinogenicity of quizalofop-p ethyl ester in animals to be equivocal and therefore concludes that quizalofop-p ethyl ester does not induce cancer in animals within the meaning of the Delaney clause. Important to this conclusion was the following evidence: (a) The only statistically significant tumor response that appears compound-related was seen at a single dose in a single sex in a single species; (b) the response was only marginally statistically significant; (c) the response was only significant when benign and malignant tumors were combined; (d) the tumors were in the male mouse liver; (e) the tumors were within historical controls; and (f) the mutagenicity studies were negative. Although in some circumstances a finding of animal carcinogenicity would be made despite any one, or even several, of the six factors noted, the combination of all of these factors here cast sufficient doubt on the reproducibility of the response in the high dose male mouse that EPA concludes the evidence on carcinogenicity is equivocal.

6. *Animal metabolism*. The metabolism of quizalofop ethyl in animals (rat, goat and poultry) is well understood. 14_C-phenyl and 14_C-quinoxaline quizalofop ethyl ester metabolism studies have been conducted in each species. There are similarities among these species with respect to metabolism. Quizalofop ethyl is rapidly and extensively metabolized and rapidly excreted by rats. The principal metabolites were the quizalofop-p acid and two dechlorinated hydroxylated forms of the acid. Tissue residues were minimal and there was no evidence of accumulation of quizalofop ethyl or its metabolites in the rat.

The primary pathway in ruminants is hydrolysis of the ethyl ester to form the quizalofop-p methyl ester. In poultry, the primary metabolic pathway is also the hydrolysis of the ethyl ester to form the quizalofop-p acid, then the methyl esterification to form the quizalofop methyl ester becomes a minor pathway.

The nature of the quizalofop ethyl ester residue in livestock is adequately understood. The residues of concern are quizalofop ethyl, quizalofop methyl, and quizalofop, all expressed as quizalofop ethyl.

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

C. Aggregate Exposure

1. *Dietary exposure.* An analysis of chronic dietary risk was conducted to determine the impact of the possible addition of peppermint and spearmint to the Assure label. A Reference Dose (RfD) of 0.009 mg/kg/day was used in the analyses. Consumption data were available for peppermint and spearmint from previous studies.

2. *Food.* The first step in the analysis was to run the TAS (Tolerance Assessment System) program using current tolerances with an RfD of 0.009 mg/kg/day. The Theoretical Maximum Residue Concentration (TMRC), based on the current tolerances, was 0.000288 mg/kg/day for the U.S. population (48 states) and 0.000759 mg/kg/day for the population subgroup with the highest estimated exposure (non-nursing infants >1 year old). For the U.S. population subgroup this represents approximately 3.2% of the RfD while for the most exposed population this represents approximately 8.4% of the RfD. Based on the risk estimates arrived at in this analysis, chronic dietary risk from the current uses of Assure is minimal.

Consumption data for peppermint and spearmint within the TAS database are available only for the entire U.S. population (48 states) and not for the population subgroups. For peppermint the consumption is listed as 0.000001 gram(g)/kg body weight(bw)/day for the raw commodity and 0.000255 for the flavoring oil. For spearmint the consumption is 0.000001 g/kg bw/day for the raw commodity and 0.000458 for the flavoring oil. The TMRC, based on the current tolerances and the potential peppermint and spearmint tolerances, was 0.000290 mg/kg/day for the U.S. population (48 states). Since no consumption data were available for population subgroups, Theoretical Maximum Residue Concentrations did not change and the sub group with the highest potential exposure had a TMRC of 0.000759 g/kg/day (non-nursing infants >1 year old). When expressed as a percentage of the RfD, the U.S. population (48 states) was approximately 3.2% and that of the population subgroup with the highest potential exposure, i.e. infants and children, was approximately 8.4%. These results indicate that predicted chronic exposure after the addition of a peppermint tolerance is well below the RfD. The lack of specific population sub-group data for these commodities should not be a problem since both peppermint and spearmint are not likely to be consumed in large quantities by any population subgroup and the difference between the TMRC and the RfD is so great.

3. *Drinking water.* Another potential source of dietary exposure to pesticides is residues in drinking water. There is no established Maximum Concentration Level (MCL) for quizalofop ethyl in water. Based on the low use rate of quizalofop ethyl, and a use pattern that is not widespread (since the current and proposed uses are on minor crops), DuPont does not anticipate residues of quizalofop in drinking water and exposure from this route is unlikely.

4. *Non-dietary exposure.* Quizalofop ethyl is not registered for any use which could result in non-occupational, non-dietary exposure to the general population.

D. Cumulative Effects

There is no evidence to indicate or suggest that quizalofop p-ethyl has any toxic effects on mammals that would be cumulative with those of any other chemicals.

E. Safety Determination

1. *U.S. population.* Using the exposure assumptions described above and based on the most sensitive species chronic NOEL of 0.9 mg/kg and a reference dose (RfD) of 0.009 mg/kg/day, the existing tolerances and proposed use of quizalofop ethyl on mint are expected to utilize 3.2% of the RfD for the general U.S. population. Generally, exposures below 100% of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to quizalofop ethyl resulting from proposed agricultural use on peppermint and spearmint.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of quizalofop ethyl, data were considered from developmental toxicity studies in the rat and rabbit, and a multi-generation reproduction study in rats. There were no developmental effects observed in the absence of maternal toxicity in the rat and rabbit developmental studies. Minimal adaptive or physiological effects were observed in livers of weanlings in the 2-generation rat reproduction study described earlier. However, this effect was only observed at a dose that far exceeds any expected human exposure. Further, the NOEL of 0.9 mg/kg/day from the 2–year rat study with quizalofop ethyl, which was used to calculate the RfD(discussed above), is already lower than any of the NOELs defined in the developmental and reproductive toxicity studies with quizalofop ethyl.

Using the exposure assumptions described above and based on the most sensitive species chronic NOEL of 0.9 mg/kg and a reference dose (RfD) of 0.009 mg/kg/day, the existing tolerances and proposed use of quizalofop ethyl on mint are expected to utilize 8.4% of the RfD for infants and children. Infants and children have a low potential for quizalofop ethyl exposure because of both the low levels of mint in the diet (mint is a low dietary intake crop used primarily as an oil for flavoring, and is diluted to a ratio of 1:250 or greater in the finished food product), and the absence of detectable residues in mint oil. The toxicology profile of quizalofop ethyl demonstrates low mammalian toxicity. Because there was no evidence that offspring were uniquely susceptible to the toxic effects of quizalofop ethyl, an additional 10-fold uncertainty factor should not be required to protect infants and children. Therefore, the registrant believes that the RfD of 0.009 mg/kg/day, which utilizes a 100-fold safety factor, is appropriate to assure a reasonable certainty of no harm to infants and children from aggregate exposure to quizalofop ethyl.

F. International Tolerances

Since there are no Mexican, Canadian, or Codex MRLs/tolerances, compatibility is not a problem at this time.

3. IR-4 Project

PP 6E4658

EPA has received a pesticide petition (PP 6E4658) from the Interregional Research Project Number 4 (IR-4), 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 to establish an exemption from the requirements of a tolerance for copper-ethylenediamine complex (Komeen) in or on the raw agricultural commodity (RAC) potatoes. 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 support granting of the petition. Additional data may be needed before EPA rules on the petition. This notice includes a summary of the petition prepared by the Griffin Corporation (Griffin), the registrant.

A. Residue Chemistry

1. *Analytical method*. A practical analytical method for copper-ethylenediamine complex is not required for crop use since it is expected that no residues will occur in RACs.

2. *Magnitude of residues*. Residues are not expected in the RAC (potatoes) since the potato tubers are underground and only the vines which are above ground are treated.

B. Toxicological Profile

The Agency does not require subchronic, chronic, reproductive or developmental toxicity studies for the copper salts.

Copper-ethylenediamine(Komeen) is slightly to moderately toxic upon acute oral, dermal and inhalation exposure, slightly irritating to the skin and moderately irritating to the eye.

Acute toxicity. The acute oral lethal dose LD_{50} (95% confidence limits) for Komeen was 498 milligram(mg)/kilogram(kg) (349–710 mg/kg).

The acute dermal LD_{50} for Komeen was determined to be >2,000 mg/kg.

The acute inhalation lethal concentration LC_{50} (95% confidence limits) for Komeen was 0.81 mg/liter(l) (0.26-1.37 mg/l).

Komeen was shown to be moderately irritating to the eye with all signs of ocular irritation cleared within 10 days of treatment.

C. Aggregate Exposure

1. *Dietary (food) exposure*. Based on the proposed used pattern of potato vine desiccation, no copper residues are expected to occur on potatoes and the dietary exposure would be negligible by comparison to the normal daily intake of copper. A single day's diet may contain 10 mg or more of copper. The daily recommended allowance of copper for adults nutritional needs is 2 mg.

2. *Drinking water*. Copper is ubiquitous in the environment and found in natural water. In 1991, the USEPA established a maximum contamination level (MCL) for copper in drinking water of 1.3 mg/l. No impact on copper levels found naturally in water would occur as a result of potato vine desiccant use for this product.

3. *Non-dietary exposure*. Copper is registered for use as an aquatic herbicide for outdoor residential sites. Any contributions to aggregate exposure from this use would not be expected to be significant.

4. *Potential for endocrine effects*. Since copper is required for homeostasis, low copper dietary exposures would not be expected result in any adverse endocrine effects.

D. Cumulative Effects

Griffin believes that no cumulative adverse effects are expected from long-term exposure to copper salts. No other elements are expected to produce cumulative toxicity with copper.

E. Safety Determination

Copper compounds such as copper sulfate pentahydrate are considered as Generally Recognized as Safe (GRAS) by the Food and Drug Administration and as such are exempt from the requirement of a tolerance when used as aquatic herbicides (40 CFR 180.1021). Copper compounds are also exempt from the requirement of a tolerance when applied to growing crops when used as a plant fungicide in accordance with good agricultural practices (40 CFR 180.1001(b)(1). Copper-ethylenediamine complex is registered as an aquatic herbicide under the trade name, Komeen.

1. *U.S. population*. Copper is a component of the human diet and an essential element. Use of copper-ethylenediamine complex is not expected to increase the amount of copper in the diet as a result of potato vine desiccation.

2. *Infants and children*. Infants and children also require copper in their diets and Griffin believes that no special sensitivity for this population subgroup would be expected as a result of the proposed use.

F. International Tolerances

No international tolerances have been established for copper-ethylenediamine complex.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–181049; FRL 5751–6]

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 California Department of Pesticide Regulations (hereafter referred to as the ''Applicant'') to use the pesticide bifenthrin (CAS #8657–04–3 *cis* and 83322–02–5 *trans*), formulated as Capture 2EC, to treat up to 22,000 acres of broccoli, cauliflower, cabbage, and rapini; and 40,000 acres of lettuce, to control silverleaf whitefly. An emergency exemption has been requested for this use for the previous 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 November 13, 1997.

ADDRESSES: Three copies of written comments, bearing the identification notation ''OPP–181049,'' 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. 1132, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically 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 as a comment concerning this document 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 submitted for inclusion in the public record. Information not marked confidential will be included in the public docket by EPA without prior notice. The public docket is available for public inspection in Rm. 1132 at the Virginia address given above, from 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: Crystal Mall #2, Rm. 267, 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