of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [OPP–30446]. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pest, Product registration.

Dated: December 29, 1997.

Donald R. Stubbs,

Acting Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 98–554 Filed 1–8–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPP–30409A; FRL–5763–5]

DowElanco; Approval of Pesticide Product Registrations

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces Agency approval of applications submitted by DowElanco, to conditionally register the pesticide products FirstRate Herbicide and Cloransulam-methyl Technical containing a new active ingredient not included in any previously registered products pursuant to the provisions of section 3(c)(7)(C) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended.

FOR FURTHER INFORMATION CONTACT: By mail: James Tompkins, Product Manager (PM) 25, Registration Division (7505C), Office of Pesticide Programs, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 239, CM #2, Environmental Protection Agency, 1921 Jefferson Davis Hwy, Arlington, VA 22202, 703–305–5697; e-mail: tompkins.james@epamail.epa.gov.

SUPPLEMENTARY INFORMATION:

Electronic Availability: Electronic copies of this document and the Fact Sheet are available from the EPA home page at the Environmental Sub-Set entry for this document under ''Laws and Regulations'' (http://www.epa.gov/fedrgstr/).

EPA issued a notice, published in the **Federal Register** of May 1, 1996 (61 FR 19279; FRL–5365–5), which announced that DowElanco, 9330 Zionsville Road, Indianapolis, IN 46268–1054, had submitted applications to register the pesticide products FirstRate Herbicide and Cloransulam-methyl Technical (EPA File Symbols 62719–ETL and 62719–ETU), containing the active ingredient cloransulam-methyl: *N*-(2-carbomethoxy-6-chlorophenyl)-5-ethoxy-7-fluoro (1,2,4) triazolo-[1,5-c]pyrimidine-2-sulfonamide at 84 and 97.5 percent respectively, an active ingredient not included in any previously registered products.

The applications were approved on October 29, 1997, for one end-use and one technical product listed below:

1. FirstRate Herbicide for broadleaf weed control in soybeans (EPA Registration Number 62719–275).

2. Cloransulam-methyl Technical for manufacturing use only (EPA Registration Number 62719–274).

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

The Agency has considered the available data on the risks associated with the proposed use of cloransulam-methyl: *N*-(2-carbomethoxy-6-chlorophenyl)-5-ethoxy-7-fluoro (1,2,4) triazolo-[1,5-c]pyrimidine-2-sulfonamide, and information on social, economic, and environmental benefits to be derived from such use. Specifically, the Agency has considered the nature and its pattern of use, application methods and rates, and level and extent of potential exposure. Based on these reviews, the Agency was able to make basic health and safety determinations which show that use of cloransulam-methyl: *N*-(2-carbomethoxy-6-chlorophenyl)-5-ethoxy-7-fluoro (1,2,4) triazolo-[1,5-c]pyrimidine-2-sulfonamide during the period of conditional registration will not cause any unreasonable adverse effect on the environment, and that use of the pesticide is, in the public interest.

These products are conditionally registered in accordance with FIFRA section 3(c)(7)(C). If the conditions are not complied with the registrations will be subject to cancellation in accordance with FIFRA section 6(e).

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

More detailed information on these conditional registrations is contained in an EPA Pesticide Fact Sheet on cloransulam-methyl: *N*-(2-carbomethoxy-6-chlorophenyl)-5-ethoxy-7-fluoro (1,2,4) triazolo-[1,5-c]pyrimidine-2-sulfonamide.

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

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are available for public inspection in the Public Information and Records Intregrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Rm. 1132, CM #2, Arlington, VA 22202 (703–305–5805). Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of Information Office (A-101), 401 M St., SW., Washington, D.C. 20460. Such requests should: (1) Identify the product name and registration number and (2) specify the data or information desired.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: December 18, 1997.

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 98–555 Filed 1–8–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–786; FRL–5762–6]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–786, must be received on or before February 9, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically 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: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224, e-mail: miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Marion Johnson (PM 10)	Rm. 217, CM #2, 703–305–6788, e-mail: johnson.marion@epamail.epa.gov.	Do.
Cynthia Giles-Parker (PM 22).	Rm. 229, CM #2, 703–305–7740, e-mail: giles-parker.cynthia@epamail.epa.gov.	Do.

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–786] (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 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [PF–786] 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: December 17, 1997.

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

1. BASF Corporation

PP 7F4881

EPA has received a pesticide petition (PP 7F4881) from BASF Corporation, Agricultural Products, P.O. Box 13528, Research Triangle Park, NC 27709, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of Pridaben, [2 tert-butyl-5(4-tert-butylbenzylthio)-4-chloropyridazin-3(2*H*)-one] in or on the raw agricultural commodity. 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, partition, clean-up and detection of residues by gc/ecd.

3. *Magnitude of residues*. Eleven peach residue trials to determine residues in peaches and nectarines were conducted in eight states. Residues of pyridaben were measured by gc/ecd. The method of detection had a limit of detection of 0.05 parts per million (ppm). Residues ranged from <0.05 to 2.36.

Eight plum residue trials to determine residues in plums and prunes were conducted in four states. Residues of pyridaben were measured by gc/ecd. The method of detection had a limit of detection of 0.05 ppm. Residues ranged from <0.05 to 0.683 ppm.

No residues trials were conducted to determine residues in cherries and apricots. Only postharvest applications are requested. No residues will occur since pyridaben is not systemic and does not translocate. The limit of detection of 0.05 ppm is proposed as the tolerance.

Fifteen grape residue trials were conducted in six states. Residues of pyridaben were measured by gc/ecd. The method of detection had a limit of detection of 0.05 ppm. Residues ranged from 0.168 to 1.38 ppm.

Six pecan residue trials were conducted in four states to complete requirements for a group tolerance for nut crops. BASF Corporation was granted a tolerance of 0.05 ppm for use on almonds. Residues of pyridaben were measured by gc/ecd. The method of detection had a limit of detection of 0.05ppm. There were no residues above 0.05 ppm.

B. Toxicological Profile

1. *Acute toxicity—Acute toxicity testing.* i. Acute Oral Toxicity (rat): LD_{50} = 1,100 mg/kg in males; 570 mg/kg in females. Tox Category: III.

ii. Acute Oral Toxicity (mouse): LD_{50} = 424 mg/kg in males; 383 mg/kg in females. Tox Category: II.

iii. Acute Dermal Toxicity (rat): LD_{50} = >2000 mg/kg in males and females. Tox Category: III.

iv. Acute Inhalation Toxicity (rat): LC_{50} = 0.66 mg/l in males; 0.62 mg/l in females. Tox Category: III.

v. Primary Eye Irritation (rabbit): Pyridaben is a slight ocular irritant. Tox Category: III.

vi. Primary Dermal irritation (rabbit): Pyridaben is not a dermal irritant. Tox Category: IV.

vii. Dermal Sensitization (guinea pig): Pyridaben is not a dermal sensitizer.

viii. Acute Neurotoxicity (rat): Rats were dosed once with 0, 50, 100 and 200 milligram/kilogram (mg/kg). The No Observed Effect Level (NOEL) for systemic toxicity was determined to be 50 mg/kg for both males and females. The Lowest Observed Effect Level (LOEL) for systemic effects was determined to be 100 mg/kg in both sexes based on decreased food consumption, decreased body weight gain and increased clinical signs. The LOEL for neurobehavioral effects was determined to be 200 mg/kg in males and >200 mg/kg in females.

2. Mutagenicity testing—Ames testing: Negative *In vitro* cytogenicity (Chinese hamster lung cells): Negative *In vivo* micronucleus assay (mouse): Negative DNA damage/repair (*E. coli*): Negative.

3. *Reproductive and developmental toxicity—* i. *Developmental toxicity testing (rat).* Sprague Dawley rats were dosed with 0, 2.5, 5.7, 13 and 30 mg/kg/day pyridaben in the diet from days 6 through 15 of gestation. The Maternal NOEL was determined to be 4.7 mg/kg/day and the maternal LOEL was determined to be 13 mg/kg/day based on decreased body weight gain, and decreased food consumption during the dosing period. The developmental NOEL was determined to be 13 mg/kg/day and the developmental LOEL was determined to be 30 mg/kg/day based on decreased fetal body weight and an increase in incomplete ossification in selected bones.

ii. *Developmental toxicity (rabbit).* New Zealand white rabbits were dosed with 0, 1.5, 5, and 15 mg/kg/day pyridaben in the diet from days 6 through 19 of gestation. The Maternal NOEL was determined to be 5 mg/kg/day and the maternal LOEL was determined to be 15 mg/kg/day based on decreased body weight gain, and decreased food consumption during the dosing period. The developmental NOEL was determined to be >15 mg/kg/day and the developmental LOEL was determined to be >15 mg/kg/day.

iii. *Developmental toxicity (rabbit).* Himalayan rabbits were dosed, by dermal application, with 0, 70, 170 and 450 mg/kg/day pyridaben from days 6 through 19 of gestation. The Maternal systemic NOEL was determined to be 70 mg/kg/day and the maternal LOEL was determined to be 170 mg/kg/day based on decreased body weight gain, and decreased food consumption during the dosing period. The developmental NOEL was determined to be 170 mg/kg/day and the LOEL determined to be 450 mg/kg/day based on decreased ossification of the skull.

iv. *Reproductive toxicity testing, multigeneration reproduction (rat).* CD rats were dosed with 0, 10, 28 and 80 ppm pyridaben in the diet. The Parental/Systemic NOEL was determined to be 28 ppm in both sexes (equivalent to 2.20 mg/kg/day in males and 2.41 mg/kg/day in females). The Parental/Systemic LOEL was determined to be 80 ppm (equivalent to 6.31 mg/kg/day in males and 7.82 mg/kg/day in females) based on decreased body weight, decreased body weight gain and decreased food efficiency. The reproductive NOEL and LOEL were both determined to be >80 ppm in males and females.

4. *Subchronic toxicity—* i. *A 21-day dermal (rat).* Rats were repeatedly dosed with pyridaben at 0, 30. 100, 300 and 1,000 mg/kg/day for 21 days. The NOEL was determined to be 100 mg/kg/day and the LOEL 300 mg/kg/day based on decreased body weight gain in females.

ii. *A 90-day rodent (rat).* CD rats were dosed with pyridaben at 0, 30, 65, 155 and 350 ppm in the diet for 13 weeks. The NOEL was determined to be 65 ppm (4.94 mg/kg/day) for males and 30 ppm (2.64 mg/kg/day) in females. The LOEL for males was determined to be 155 ppm (11.55 mg/kg/day) based on reduced body weight gain, reduced food consumption, reduced food efficiency, and altered clinical pathology parameters. The LOEL for females was determined to be 65 ppm (5.53 mg/kg/day) based on reduced body weight gain and reduced food efficiency.

iii. *A 90-day non-rodent (dog).* Beagle dogs were dosed with pyridaben at 0, 0.5, 1,4, and 16 mg/kg/day in the diet for 13 weeks. The NOEL was determined to be 1 mg/kg/day and the LOEL determined to be 4 mg/kg/day based on reduced body weight gain and an increase in clinical signs in both sexes.

iv. *A 90-day neurotoxicity (rat).* Rats were dosed with pyridaben at 0, 30, 100, and 350 ppm in the diet for 13 weeks. The systemic NOEL was determined to be 100 ppm (equivalent to 8.5 mg/kg/day in males and 9.3 mg/kg/day in females). The systemic LOEL was determined to be 350 ppm (equivalent to 28.8 mg/kg/day in males and 31.1 mg/kg/day in females) based on decreased body weight gain, decreased food consumption and decreased food efficiency. No neuropathological effects were noted in the study.

5. *Chronic toxicity—* i. *A 1-year non-rodent (dog).* Two studies were run. In the first, beagle dogs were dosed with pyridaben at 0, 1, 4, 16 and 32 mg/kg/day in the diet for 1-year. In the second, beagle dogs were dosed with pyridaben at 0 and 0.5 mg/kg/day in the diet for 1-year. The NOEL was determined to be <0.5 ppm and LOEL determined to be 0.5 mg/kg/day based on increased clinical signs and decreased body weight gain in both sexes.

ii. *Combined rodent chronic toxicity/carcinogenicity (rat).* Wistar rats were fed 0, 4, 10, 28 and 80 ppm pyridaben in the diet to assess carcinogenicity and 0, 4, 10, 28 and 120 ppm in the diet to assess chronic toxicity for 104 weeks. The NOEL was determined to be 28 ppm in both sexes (equivalent to 1.13 mg/kg/day in males and 1.46 mg/kg/day in females). The LOEL was determined

to be 120 ppm in both sexes (equivalent to 5.0 mg/kg/day in males and 6.52 mg/kg/day in females) based on decreased body weight gain in both sexes and decreased ALT levels in males. Pyridaben was not carcinogenic under the conditions of the test.

iii. *Carcinogenicity in the rodent (mouse).* CD-1 mice were fed 0, 2.5, 8.0, 25 and 80 ppm pyridaben in the diet for 78 weeks. The NOEL was determined to be 25 ppm in both sexes (equivalent to 2.78 mg/kg/day in both sexes). The LOEL was determined to be 80 ppm in both sexes (equivalent to 8.88 mg/kg/day in males and 9.74 mg/kg/day in females) based on decreased body weight gain, decreased food efficiency and changes in organ weights and histopathology. Pyridaben was not carcinogenic under the conditions of the test.

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

7. *Threshold effects.* Based on the available chronic toxicity data, EPA has established the Reference Dose (RfD) for pyridaben at 0.005 mg/kg/day. The RfD for pyridaben is based on a 1-year feeding study in dogs with a threshold Lowest-Observed Effect Level (LOEL) of 0.5 mg/kg/day based on increased clinical signs and decreased body weight gain in both sexes and an uncertainty factor of 100.

8. *Non-threshold effects.* Using its Guidelines for Carcinogenic Risk Assessment, EPA has classified pyridaben as Group ''E'' for carcinogenicity (no evidence of carcinogenicity) based on the results of carcinogenicity studies in two species. There was no evidence of carcinogenicity in an 18-month feeding study in mice and a 2-year feeding study in rats at the dosage levels tested. The doses tested were adequate for identifying a cancer risk. Thus, a cancer risk assessment is not necessary.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Since Pyridaben is regulated based upon non-carcinogenic chronic toxicity, BASF conducted a DRES analysis based on anticipated residue levels determined by the tolerance support branch of HED. The anticipated residue levels were derived from the average residue levels from field trials conducted at the maximum proposed use rate and minimum pre-harvest interval, and a correction factor of 2.3 to account for all organosoluble residues as determined by EPA HED. This analysis demonstrates that the exposure to non-nursing infants <1 year, the most sensitive sub-population is approximately 128.6% of the RfD and to the general population exposure is approximately 18.6%. Assuming a conservative pyridaben market share of 65% of all crop uses, then the most sensitive sub-population is approximately 83.8% of the RfD and to the general population exposure is approximately 12.1%.

ii. *Drinking water.* Other potential sources of exposure of the general population to residues of pesticides are residues in drinking water and exposure from non-occupational sources. Based on the studies submitted to EPA for assessment of environmental risk, BASF does not anticipate exposure to residues of pyridaben in drinking water. There is no established Maximum Concentration Level for residues of pyridaben in drinking water under the Safe Drinking Water Act. BASF has not estimated non-occupational exposure for pyridaben since the current registration for pyridaben is limited to commercial greenhouse use for non-food ornamental plants and the only other use will be for commercial apple/pear and citrus production The potential for non-occupational exposure to the general population is considered to be insignificant.

D. Cumulative Effects

BASF also considered the potential for cumulative effects of pyridaben and other substances that have a common mechanism of toxicity. BASF has concluded that consideration of a common mechanism of toxicity is not appropriate at this time since there is no reliable information to indicate that toxic effects produced by pyridaben would be cumulative with those of any other chemical compounds.

E. Safety Determination

1. *U.S. population*. Reference Dose (RfD), using the exposure assumptions described in section III, above, BASF concludes that aggregate exposure to pyridaben will utilize approximately 12.1% the RfD for the U.S. population. EPA generally has no concern for exposures below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of pyridaben, including all anticipated dietary exposure and all other non-occupational exposures.

2. *Infants and children*. Developmental toxicity (delayed ossification) was observed in developmental toxicity studies using rats and rabbits. The No-Observed Effect Level's (NOEL's) for developmental effects were established at 13 mg/kg/day in the rat study and 15 mg/kg/day in the rabbit study. The developmental effect observed in these studies is believed to be a secondary effect resulting from maternal stress (decreased body weight gain and food consumption).

In a 2-generation reproduction study in rats, pups from the high dose group, which were fed diets containing 80 ppm (equivalent to 6.31 and 7.82 mg/kg/day in male and females, respectively) gained less weight beginning on lactation day 14. Parental systemic toxicity including decreased body weights, body weight gains and food efficiency in males, and slightly decreased body weights and body weight gains in females during lactation was also observed in the high dose group. The results of this study indicate that the loss in weight gain in pups from the high dose group was affected by nursing.

No clear scientific consensus yet exists to define the most appropriate endpoints for assessing risk in children. However, in consideration of the data that show both developmental and reproductive toxicity were effects secondary to parental toxicity, BASF believes that the established Reference Dose (RfD) of 0.005 mg/kg/day is the most conservative approach for assessing risk in children. Using the exposure assumptions described in section 5, above, BASF has concluded that the percent of the RfD, when adjusted for a conservative market share of 65%, that will be utilized by aggregate exposure to residues of pyridaben from the proposed use in citrus, apples, pears, almonds, peaches, plums, and grapes is approximately 83.8% for non-nursing infants (< 1 year), the most sensitive sub-population. 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 pyridaben, including all anticipated dietary exposure and all other non-occupational exposures.

F. Other Considerations

The qualitative nature of the residues in plants and animals is adequately understood. Residues of the parent molecule, pyridaben are the only residues of concern. Residues of pyridaben do not concentrate in the processed commodities apple and citrus juice. There is a practical analytical method for detecting and measuring levels of pyridaben in or on food with

a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances. Endocrine effects. No specific tests have been conducted with pyridaben to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there were no significant findings in other relevant toxicity studies, i.e., teratology and multi-generation reproductive studies, which would suggest that pyridaben produces endocrine related effects.

G. International Tolerances

A maximum residue level has not been established for pyridaben by the Codex Alimentarius Commission. (PM 10)

2. GMJA Specialties

PP 7G4891

EPA has received a pesticide petition (PP 7G4891) from GMJA Specialties, 1001 13th Avenue East, Bradenton, FL 34208, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of PT807-HCl in or on the raw agricultural commodity oranges at 0.01 ppm. The proposed analytical method is extracting PT807-HCl from whole oranges, juice, and dried pulp using organic solvents has been validated. Extracted PT807-HCl residues are analyzed using high performance liquid chromatography (HPLC) with a UV detector. The limit of quantitation (LOQ) of the method is 0.01 part 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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

Plant metabolism. The metabolism of PT807-HCl in plants and animals is understood. In plants (oranges), unchanged parent is the only residue identified in fruit. Valencia orange trees were treated with ^{14}C PT807-HCl at a nominal rate of 1,000 ppm (approximately 60x the maximum recommended application rate). Fruit from the previous season's crop present on the tree at the time of application was harvested 50 days after treatment (DAT) and mature fruit (not present on the tree at application) was harvested 370 DAT. Total radioactive residue (TRR) levels were 0.538 ppm in 50 DAT orange samples and were 0.051 ppm in 370 DAT orange samples. Most of the radioactivity was present on the peel (88.63% TRR or 0.475 ppm in the 50 DAT fruit, and 64.19% TRR or 0.033 ppm in the 370 DAT fruit). Unchanged parent PT807-HCl was detected in 50 DAT mature fruit using organic solvents has been validated. Extracted PT807-HCl residues are analyzed using HPLC with a UV detector. The LOQ of the method is 0.01 ppm.

B. Toxicological Profile

1. *Acute toxicity*. A battery of acute toxicity studies has been conducted and the results indicate that PT807-HCl exhibits low acute oral, dermal, and inhalation toxicity. PT807-HCl also has low potential as a skin or eye irritant and is not a skin sensitizer.

2. *Genotoxicity*. The genotoxic potential of PT807-HCl has been assessed in an Ames *Salmonella* assay, a CHO HGPRT gene mutation assay, a mouse micronucleus assay, and an *in vitro* CHO assay for chromosomal aberrations. The *in vitro* chromosomal aberration assay was positive with and without metabolic activation; however, all of the remaining assays were negative, indicating very low genotoxic potential of PT807 weakened by the negative finding in an *in vivo* study (mouse micronucleus) measuring a similar endpoint.

3. *Reproductive and developmental toxicity*. A 2-generation reproductive toxicity study of PT807-HCl is ongoing.

4. *Analytical method*. An analytical method capable of extracting PT807-HCl from whole oranges, juice, and dried pulp using organic solvents has been validated. Extracted PT807-HCl residues are analyzed using high performance liquid chromatography (HPLC) with a UV detector. The limit of quantitation (LOQ) of the method is 0.01 ppm.

5. *Magnitude of residues*. Seventeen field trials were conducted using various varieties of oranges in California (4 trials), Florida (12 trials), and Texas (1 trial). Two of the trials (1 in California and 1 in Florida) were decline studies with sampling intervals of 0, 7, 14, 30, and 60 days after application. For all other trials, oranges were harvested at the earliest possible time for normal commercial harvest after a single application with PT807-HCl at the maximum recommended application rate (6 g a.i./A). At some of the test sites (depending on the variety of oranges), the previous season's crops was present on the tree at application for these trials, oranges were collected 0 to 68 days after treatment (DAT). In all other trials, fruit were not present on the trees at applications and mature oranges were collected at normal harvest (197 to 359 DAT). Samples were analyzed for residues of PT807-HCl by HPLC with UV detection. Residues of PT807-HCl were nondetectable (<0.01 ppm) in all treated and control samples.

A processing study was conducted using oranges treated at 5x the maximum application rate in California. The harvested oranges were from the previous season's crop and were on the tree at the time of application. Therefore the application represents the maximum possible residues. No detectable residues were measured in whole oranges, juice, or oil. Residues of PT807-HCl were detectable in dried pulp at 0.015 and 0.017 ppm (average 0.016 ppm). Correcting the measured residues for the exaggerated application rate, no detectable residues are likely in any processed product of oranges.

Residues of PT807-HCl were determined to be stable in whole orange fruit, oil, juice, and dried pulp stored frozen up to 113 days.

6. *Subchronic toxicity*. Subchronic toxicity studies have been conducted with PT807-HCl in mice, rats, and dogs. In dietary studies in rats and dogs, the most notable findings include decreased food consumption and a consequent decrease in bodyweight gain.(resulting primarily from poor palatability of the test material). Dogs also showed a trend toward anemia, and males showed arrested or delayed sexual maturation at the high dose (equivalent to approximately 222 mg/kg/day). Marked weight loss and decreased weight gain was observed at this dose and this dose level is considered to have exceeded a MTD. Rats dosed by gavage showed signs of neurotoxic effects (tremors, incoordination, changes in activity) at doses 3-- mg/kg/day. In mice treatment-related decreased food consumption and body weight gain were seen in males at 7,000 (HDT). No treatment-related toxicity was evident at dietary doses up to 3,500 ppm (479 and 635 mg/kg/day for males and females, respectively).

7. *Chronic toxicity*. Chronic toxicity studies of PT807-HCl in rats and doges are currently ongoing.

8. *Animal metabolism*. ^{14}CPT807-HCl was extensively metabolized and readily eliminated in the urine and feces following oral administration to a lactating goat. The efficient elimination processes resulted in negligible to modest retention of radioactive residues in milk and tissues (<0.2 % of the administered dose). No residues of unchanged parent were identified in tissues or milk. The rapid elimination of the PT807-HCl and its metabolites

coupled with the highly exaggerated dose (approximately 3,600x the dietary burden) clearly indicates that no detectable residues of PT807-HCl will accumulate in milk and tissues.

9. *Metabolite toxicology*. The metabolism of PT807-HCl in oranges has been determined. The only significant metabolite is unchanged parent. No detectable residues of PT807-HCl are anticipated in oranges treated at the recommended application rate.

C. Aggregate Exposure

1. *Dietary exposure*. There are no anticipated dietary exposures to PT807-HCl outside of those requested in this temporary tolerance petition. The chronic dietary exposure from the consumption of oranges and its processed products treated with PT807-HCl is very low. The exposure is only 5.0 % of the RfD (0.00063 mg/kg/day) for the most highly exposed sub-population, children 1 to 6 years old. The dietary exposure is only 1.7% of the RfD (0.00021 mg/kg/day) for the U.S. population.

2. *Food*. The proposed temporary tolerance of 0.01 ppm was used for the residue level to calculate the dietary exposure from residues of PT807-HCl in or on oranges. Based on the processing study, there is no anticipated concentration of residues of PT807-HCl in processed products of oranges, therefore, the proposed temporary tolerance level for whole oranges was also used for the processed commodities. For the purpose of calculating a worst-case estimate, it was assumed that 100% of the oranges and their processed products were treated with PT807-HCl.

3. *Drinking water*. Based on the results of the GENEEC model, the 56-day chronic EEC (calculated from the lowest K_{oc} value measured for PT807-HCl) is 0.315 µg/L. Using the standard drinking water consumption scenarios of 2 liters per day for a 70-kg adult and 1 liter per day for a 10 kg child, the calculated consumption of PT807-HCl in drinking is 0.009 µg/kg/day for an adult and 0.032 µg/kg/day for a child. These consumption values correspond to 0.7% of the RfD for adults and 2.6% of the RfD for children. As discussed above, drinking water concentrations calculated by the GENEEC procedure represent very conservative screening level assessments of drinking water exposure. Finally, the above drinking water calculations use the water concentration calculated from the lowest K_{oc} value measured for PT807-HCl. Three of four soils tested gave K_{oc} values that are more than 10-fold higher, leading to correspondingly lower calculated water concentrations.

4. *Non-dietary exposure*. There are currently no registered uses for PT807-HCl, and therefore, there is no anticipated non-occupational exposure to the chemical.

D. Cumulative Effects

GMJA Specialities is not aware of any currently registered products that are structurally similar to PT-807-HCl or that would be likely to share a common mechanism of action. Therefore, no cumulative exposures are considered in the PT807-HCl dietary risk assessment.

E. Safety Determination

1. *U.S. population*. The chronic dietary exposure from the consumption of oranges and its processed products treated with PT807-HCl is very low. The exposure is only 5.0 % of the RfD (0.00063 mg/kg/day) for the most highly exposed sub-population, children 1 to 6 years old. The dietary exposure is only 1.7% of the RfD (0.00021 mg/kg/day) for the U.S. population.

2. *Infants and children*. The reference dose is conservatively calculated using a very high (10,000-fold) safety factor for children. Based on currently available data, PT807-HCl does not present a unique hazard to infants or children and there is no evidence that children are likely to be more sensitive to the toxic effects of PT807-HCl. A 2-generation reproductive toxicity study with PT807-HCl in rats is currently ongoing. PT807-HCl showed evidence of developmental effects in rats only at a severely maternally toxic dose level. No evidence of developmental toxicity was seen in rabbits.

F. International Tolerances

There are no Codex Alimentarius Commission (Codex Maximum Residue Levels (MRLs) for PT807-HCl. (PM 22)

3. Rohm & Haas Company

PP 3F4229

EPA has received a pesticide petition (PP 3F4229) from Rohm & Haas Company, Philadelphia, PA, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of oxyfluorfen in or on the raw agricultural commodities peanut meat, meal, vine, hay, crude oil, soap stock, and refined oil at 0.05 ppm and peanut hulls at 0.10 ppm. The proposed analytical method involves extraction from the raw agricultural commodity with methanol or acetonitrite. Extracts are refluxed in presence of NaOH and Al to reduce and or hydrolyze residues to 4-(2-chloro-4-(trifluoromethyl)-phenoxy)-2-ethoxybenzenenamine. The derivatives are partitioned into hexane and heptafluorobutyryl derivatives prepared. Following Florisel cleanup, residues are determined by electron capture GLC. 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 and animal metabolism*. The chemical identities of potential plant residues resulting from the use of oxyfluorfen have been elucidated. The principal residue in plants is parent oxyflurofen.

The chemical identities of potential animal residues resulting from consumption of oxyfluorfen-treated crops have been elucidated. Parent oxyfluorfen is the principal residue in animal tissues. Oxyfluorfen residues do not transfer to milk (concentration <0.01 ppm at 10x dose). Residues also do not appreciably transfer to cow muscle, liver and kidney (highest level 0.011 ppm at 10x dose). Residues are present in cow fat at low levels (less than 0.01 at 1x dose). Residues in eggs and hen liver are 0.02 ppm or less on average at a 1x dose, and less than 0.01 ppm in muscle at the 1x dose. Residues approach 0.2 ppm in hen fat at the 1x dose.

2. *Analytical method*. There is a practical analytical method for detecting and measuring levels of oxyfluorfen in or on food with a limit of detection that allows monitoring of food with residues at or above the levels in these proposed tolerances. EPA has provided information on this method to FDA. The method is available to anyone who is interested in pesticide residue enforcement from: By mail, Calvin Furlow, Public Response and Program Resources Branch, Field Operations Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Crystal Mall #2, Rm. 1132, 1921 Jefferson Davis Highway, Arlington, Virginia, 703-305-5805.

3. *Magnitude of the residues*. Residue studies have been conducted in accordance with the geographic distribution mandated by the EPA for peanuts. Oxyfluorfen residues were not detectable in nutmeat [NDR <LOQ = 0.01 mg/kg] or peanut hay [NDR<LOQ = 0.03 mg/kg]. Tolerances of 0.05 ppm in

nutmeat and hay are proposed based on these data. Residues were not measured in processed fractions of peanuts and tolerances are not proposed because residues are not likely to exceed the proposed 0.05 ppm tolerance level for nutmeat since the maximum theoretical concentration factor for process fractions is 2x.

B. Toxicological Profile

1. *Acute toxicity.* Oxyfluorfen is practically nontoxic by the oral, dermal, and respiratory routes of exposure, is nonirritating to the skin and moderately irritating to the eye.

2. *Genotoxicity. In vitro* tests in *salmonella* and mouse lymphoma cells have indicated the potential for genotoxicity. *In vivo* tests do not show a potential for adverse chromosal effects.

3. *Reproductive and developmental toxicity.* Maternal and developmental toxicity were noted at an oxyfluorfen dose of 183 mg/kg/day (NOEL of 18 mg/kg/day) in rats and at a dose of 30 mg/kg/day (NOEL of 10 mg/kg/day) in rabbits. Reductions in body weight of offspring and histopathologic alteration of the kidneys of parents were observed with a dose of oxyfluorfen of ~80 mg/kg/day (NOEL ~20 mg/kg/ day) in a rat 2-generation reproduction study.

4. *Subchronic and chronic toxicity.* Adverse effects on the liver marked the LOEL in all three chronic toxicity studies with NOELs of 2.5, 2.0, and 0.3 mg/kg/day seen in the dog, rat, and mouse studies respectively. A statistically significant positive dose-related trend for liver adenomas and carcinomas was observed in the chronic mouse study and oxyfluorfen is classified as a Group C chemical by EPA. A reference dose of 0.003 mg/kg/day and a $Q1^*$ of 0.128 $(mg/kg/day)^{-1}$ has been set by the Agency.

5. *Animal metabolism.* Animal metabolism studies have been conducted on farm animals using laying hens and lactating goats and in a laboratory animal (rat). These studies were reviewed and accepted by the Agency. EPA has concluded that the metabolism of oxyfluorfen in animals is adequately understood.

C. Aggregate Exposure

1. *Food.* To determine chronic (using the RfD) and cancer (using the $Q1^*$ approach) risks, refined dietary exposure estimates using percent of crop treated and anticipated residues were utilized for registered uses of oxyfluorfen with established tolerances on the following food and/or animal feed items: dates, figs, guava, loquats, olives, papaya, persimmon, pomegranate, plantains, kiwi, cocoa butter, coffee, artichokes, taro-roots and greens, garlic, shallots, cauliflower, bok-choy, and other Chinese variety cole crops, dry beans, crabapples, quince, blackberry, raspberry, Brazil nut, cashew, chestnuts, hazelnuts, hickory nuts, macadamias, pecans, horseradish, peppermint, spearmint, pistachio nuts, cotton, cherries, nectarines, plums, prunes, almonds, walnuts, bananas, broccoli, cabbage, apricots, nutmeat, milk, onions, soybeans, apples, pears, peaches, grapes, and corn. Actual residues are expected to be quite low because the majority of the use patterns direct sprays onto weeds or soil and away from the crop. There are long preharvest intervals for sprays which are directly applied to crops.

Acute dietary exposure (food only) was calculated using the TMRC (worst case) assumptions.

2. *Drinking water.* The Agency has reviewed environmental fate data which indicate that oxyfluorfen is persistent but nonmobile. There is no established Maximum Concentration Level (MCL) for residues of oxyfluorfen in drinking water. No health advisory levels for oxyfluorfen in drinking water have been established. As noted in ''Pesticides in Groundwater Database'' EPA 734-12-92-001, September 1992, 188 wells were monitored in Texas in 1987 and 1988. No detectable residues of oxyfluorfen were found in any of the samples.

While EPA has not yet pinpointed the appropriate bounding figure for consumption of contaminated water, the ranges the Agency is continuing to examine are all below the level that would cause oxyfluorfen to exceed the RfD if the tolerance being considered in this document were granted. In addition, chronic exposure to oxyfluorfen residues resulting from potential water exposure would not increase the total cancer risk so that it exceeds the Agency's level of concern. The potential exposures associated with oxyfluorfen in water, even at the higher levels the Agency is considering as a conservative upper bound for RfD exposure considerations, would not prevent the Agency from determining that there is a reasonable certainty of no harm if the tolerance is granted.

Despite the potential for acute exposure to oxyfluorfen in drinking water, it is not expected that the aggregate acute exposure will exceed the Agency's level of concern if the tolerance being considered in this document were granted. The potential acute term exposures associated with oxyfluorfen in water, even at the higher levels the Agency is considering as a conservative upper bound, would not prevent the Agency from determining that there is a reasonable certainty of no harm if the tolerance is granted.

3. *Non-dietary exposure.* Oxyfluorfen is registered for outdoor residential use. Acceptable, reliable data are not currently available with which to assess acute risk. However, based on the available residential exposure data and the best professional judgment of scientists who have worked with the available occupational exposure data, 5% of the risk for outdoor residential uses is a reasonable, protective default assumption for this pesticide. Chronic exposure to oxyfluorfen residues resulting from potential outdoor residential exposure would not increase the total chronic or cancer risks so that they exceed the Agency's level of concern.

Theoretically, it is also possible that a residential, or other non-dietary, exposure could be combined with the acute total dietary exposure from food and water. However, the Agency does not believe that aggregating multiple exposure to large amounts of pesticide residues in the residential environment via multiple products and routes for a one-day exposure is a reasonably probable event. It is highly unlikely that, in one day, an individual would have multiple high-end exposures to the same pesticide by treating their lawn and garden, treating their house via crack and crevice application, swimming in a pool, and be maximally exposed in the food and water consumed.

D. Cumulative Effects

EPA does not have, at this time, available data to determine whether oxyfluorfen has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, oxyfluorfen does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that oxyfluorfen has a common mechanism of toxicity with other substances.

E. Endocrine Effects

The toxicity studies required by EPA for the registration of pesticides measure numerous endpoints with sufficient sensitivity to detect potential endocrine-modulating activity. No effects have been identified in subchronic, chronic, reproductive, or developmental toxicity studies to indicate any endocrine-modulating activity by oxyfluorfen.

More importantly, the multi-generation reproduction study in rodents is a complex study design which measures a broad range of endpoints in the reproductive system and in developing offspring that are sensitive to alterations by chemical agents. Oxyfluorfen has been tested in two separate multi-generation studies and each time the results demonstrated that oxyfluorfen is not a reproductive toxin.

F. Safety Determination

1. *U.S. population*— i. *Chronic RfD and cancer risk.* Using the refined dietary exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, it is concluded that aggregate dietary exposure (food only) to oxyfluorfen will utilize 0.04% of the RfD for the general United States population. EPA has no concern generally for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to oxyfluorfen in drinking water and from the 5% default-level contribution from non-dietary, nonoccupational exposure, it is not expected the aggregate exposure will exceed 100% of the RfD. As noted above, oxyfluorfen has been classified as a Group C chemical by the Agency based on liver adenomas and carcinomas in the 20-month mouse feeding study. The Agency recommends using the $Q1^*$ approach to assess cancer risk. A value of 0.067 $(mg/kg/day)^{-1}$ is recommended.

The refined dietary assumptions for existing oxyfluorfen tolerances plus those proposed for peanuts result in an Anticipated Residue Contribution (ARC) that is equivalent to a risk of 8.0×10^{-8} (food only). Actual residues are expected to be quite low because the majority of the use patterns direct sprays onto weeds and away from the crop and there are long preharvest intervals for sprays which are directly applied to crops. Environmental fate data indicate that oxyfluorfen strongly adheres to soil, does not leach into groundwater and has not been detected in sampled groundwater. Based on this information, occurrence of oxyfluorfen in drinking water is unlikely. Outdoor residential uses of oxyfluorfen are limited and exposure is expected to be low. Oxyfluorfen is toxic to lawn grasses and certain ornamental plants, and use is generally limited to spot treatments for nonselective weed control. Chronic exposure to oxyfluorfen residues resulting from potential residential and/or water exposure would not increase the total cancer risk so that it exceeds the Agency's level of concern. There is a reasonable certainty that no harm will result from chronic aggregate exposure to oxyfluorfen residues.

ii. *Acute risk.* The acute dietary exposure endpoint of concern for oxyfluorfen is fused sternebrae in developing pups which was observed in the rabbit developmental study. The population subgroup of concern is females 13+ years old (women of childbearing age). For this subgroup, the calculated MOE at the high end exposure is greater than 5,000. The Agency considers dietary (food) MOEs of greater than 100 to be acceptable for oxyfluorfen. Acute dietary exposure (food only) was calculated using the TMRC (worst case) assumptions.

In the absence of data for drinking water exposure, the ranges of exposure being considered by the Agency for consumption of contaminated water will be reserved for drinking water. The aggregate MOE level of concern for dietary plus the addition of upperbound estimates for drinking water is not likely to raise the MOE level of concern above 150. Despite the potential for acute exposure to oxyfluorfen in drinking water, it is not expected that the aggregate exposure will exceed the Agency's level of concern if the tolerance being considered in this document were granted. It is therefore concluded that the potential acute exposure associated with oxyfluorfen in water, even at the higher levels the Agency is considering as a conservative upper bound, would not prevent the Agency from determining that there is a reasonable certainty of no harm if the tolerance is granted.

2. *Infants and Children*. The toxicology database is complete for oxyfluorfen relative to prenatal and postnatal toxicity. In the developmental toxicity study in rabbits, at the maternally toxic dose of 30 mg/kg/day, there were developmental anomalies (fused sternebrae) in the fetuses which demonstrated that prenatal toxicity should be evaluated by an acute dietary risk estimate. The acute dietary MOE for pregnant women 13+ years old is greater than 5,000 based on a developmental NOEL of 10 mg/kg/day. This MOE is much higher than the minimal acceptable MOE (100 for dietary-food only) and suggests that prenatal developmental risks to infants and children from exposure to oxyfluorfen dietary residues is not a concern. Additionally, the rabbit developmental NOEL of 10 mg/kg/day is 33 times greater than the NOEL of 0.3 mg/kg/day used to calculate the RfD. In the developmental toxicity study in rats, both the developmental and maternal NOEL and LOEL of 18 and 183 mg/kg/day, respectively, occurred at the same dose levels and demonstrates that there is no special sensitivity in infants and children exposed to oxyfluorfen. Although the developmental findings in the rat were severe effects, the developmental NOEL of 18 mg/kg/day is greater than the rabbit developmental NOEL of 10 mg/kg/day used to calculate acute dietary MOEs. Therefore, the acute dietary risk estimates calculated from the rabbit developmental NOEL are lower than acute dietary MOEs which could be calculated for the more severe effects occurring in rats above the NOEL of 18 mg/kg/day. By basing the acute dietary MOEs on the NOEL in the most sensitive species (rabbit), pregnant women are protected against both types of prenatal toxicity effects as seen in the rat and rabbit developmental toxicity studies. Therefore, there are no significant prenatal toxicity concerns for infants and children due to the high MOE for pregnant women 13+ years old. In the 2-generation reproductive toxicity study in rats used to assess the postnatal toxicity potential of infants and children, the NOEL and LOEL of 20 mg/kg/day and 80 mg/kg/day, respectively, for developmental/reproductive and systemic toxicity demonstrated that there are no pup toxicity effects in the absence of parental toxicity (NOEL and LOEL are the same for pups and parental animals). Therefore, there are no special postnatal sensitivities in infants and children which can be attributed to the findings of the 2-generation reproductive toxicity study in rats. Additionally, the developmental/reproductive NOEL of 20/mg/kg/day [which is the NOEL for decreased litter size at birth as well as decreased pup body weight] and the parental systemic NOEL of 20 mg/kg/day is 66 times greater than the NOEL of 0.3 mg/kg/day used to calculate the RfD.

Based on the above, EPA concludes that reliable data support use of the standard hundredfold margin of exposure/uncertainty factor and that an additional margin/factor is not needed to protect the safety of infants and children.

i. *Chronic risk.* Using the refined exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, it is concluded that aggregate dietary exposure to oxyfluorfen will utilize 0.05% of the RfD for infants and 0.08% of the RfD for children. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at

or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to oxyfluorfen in drinking water and from non-dietary, nonoccupational exposure, the chronic aggregate exposure is not expected to exceed 100% of the RfD. There is a reasonable certainty that no harm will result to infants and children from chronic aggregate exposure to oxyfluorfen residues.

ii. *Acute risk.* As mentioned above, the acute dietary exposure endpoint of concern for oxyfluorfen is fused sternebrae in developing pups which was observed in the rabbit developmental study. The population subgroup of concern is females 13+ years old (women of childbearing age). For this subgroup, the calculated MOE at the high end exposure is greater than 5,000. The Agency considers dietary (food) MOEs of greater than 100 to be acceptable for oxyfluorfen. Acute dietary exposure (food only) was calculated using the TMRC (worst case) assumptions.

In the absence of data for drinking water exposure, the ranges of exposure being considered by the Agency for consumption of contaminated water will be reserved for drinking water. Based on the ranges under consideration, the aggregate MOE level of concern for dietary plus the addition of drinking water is not likely to raise the MOE above the Agency's level of concern. The large MOE calculated for this use of oxyfluorfen provides assurance that there is a reasonable certainty of no harm for infants and children.

G. International Tolerances

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRL's) established for residue of oxyfluorfen in or on raw agricultural commodities. (PM 23)

[FR Doc. 98–557 Filed 1–8–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–44645; FRL–5763–9]

TSCA Chemical Testing; Receipt of Test Data

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces EPA's receipt of test data on n-amyl acetate (CAS No. 628–63–7), and alkyl glycidyl ether (CAS No. 120547–52–6). These data were submitted pursuant to enforceable testing consent agreements/orders issued by EPA under section 4 of the Toxic Substances Control Act (TSCA). Publication of this notice is in compliance with section 4(d) of TSCA.

FOR FURTHER INFORMATION CONTACT: Susan B. Hazen, Director, Environmental Assistance Division (7408), Office of Pollution Prevention and Toxics, Environmental Protection Agency, Rm. E–543B, 401 M St., SW., Washington, DC 20460, (202) 554–1404, TDD (202) 554–0551; e-mail: TSCA-Hotline@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: Under 40 CFR 790.60, all TSCA section 4 enforceable consent agreements/orders must contain a statement that results of testing conducted pursuant to testing enforceable consent agreements/orders will be announced to the public in accordance with procedures specified in section 4(d) of TSCA.

I. Test Data Submissions

Test data for n-amyl acetate were submitted by RegNet Environmental Services on behalf of Union Carbide Corporation pursuant to a TSCA section 4 enforceable testing consent agreement/order at 40 CFR 799.5000. EPA received the data on October 29, 1997. The submission includes a final report entitled ''A 13-Week Inhalation Neurotoxicity Study By Whole-Body Exposure of n-Amyl Acetate Vapor in the Albino Rat.'' This chemical is primarily used as a solvent for nitrocellulose lacquers and paints. Other large uses are as extraction solvents in penicillin manufacture and electrostatic spray coatings for automobiles. Miscellaneous uses include a solvent in photographic film, leather polishes, dry cleaning preparations, and as a flavoring agent.

Test data for alkyl glycidyl ether were submitted by The Society of the Plastics Industry, Inc. Epoxy Resin Systems Alkyl Glycidyl Ether Task Force. The following companies comprise the Task Force: Air Products and Chemicals Inc.; Callaway Chemical Company; Ciba-Geigy Corporation; CVC Specialty Chemicals; and Shell Chemical Company. The submission includes three final reports entitled (1) ''Alkyl Glycidyl Ether: 2-Week Range Finding and 13-Week Repeated Dose Dermal Toxicity Study in Fischer 344 Rats, (2) ''A Dermal Developmental Toxicity Screening Study of Alkyl Glycidyl Ethers in Rats,'' and (3) ''Bacterial Reverse Mutation Assay with an Independent Repeat Assay.'' These reports were submitted in accordance with a TSCA section 4 enforceable testing consent agreement/order at 40 CFR 799.5000. The first two reports were received by EPA on November 12, 1977 and the third report was received by EPA on November 18, 1997. This chemical is used as an epoxy resin additive and as a modifier for other epoxides in flooring and adhesives.

EPA has initiated its review and evaluation process for this data submission. At this time, the Agency is unable to provide any determination as to the completeness of the submission.

II. Public Record

EPA has established a public record for this TSCA section 4(d) receipt of data notice (docket number OPPTS–44645). This record includes copies of all studies reported in this notice. The record is available for inspection from 12 noon to 4 p.m., Monday through Friday, except legal holidays, in the TSCA Nonconfidential Information Center (also known as the TSCA Public Docket Office), Rm. B–607 Northeast Mall, 401 M St., SW., Washington, DC 20460. Requests for documents should be sent in writing to: Environmental Protection Agency, TSCA Nonconfidential Information Center (7407), 401 M St., SW., Washington, DC 20460 or fax: (202) 260–5069 or e-mail: oppt.ncic@epamail.epa.gov.

Authority: 15 U.S.C. 2603.

List of Subjects

Environmental protection, Test data.

Dated: December 22, 1997.

Charles M. Auer,

Director, Chemical Control Division, Office of Pollution Prevention and Toxics.

[FR Doc. 98–560 Filed 1–8–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–42199B; FRL–5765–1]

Enforceable Consent Agreement Development for Maleic Anhydride; Solicitation of Interested Parties and Notice of Public Meeting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA is soliciting interested parties who want to monitor or participate in negotiations on an enforceable consent agreement (ECA) concerning the use of pharmacokinetics (PK) studies and mechanistic data to help meet testing requirements for maleic anhydride in the proposed hazardous air pollutants (HAPs) test