the comment date. Protests will be considered by the Commission in determining the appropriate action to be taken, but will not serve to make protestants parties to the proceeding. Any person wishing to become a party must file a motion to intervene. Copies of this filing are on file with the Commission and are available for public inspection.

Lois D. Cashell,

Secretary.

[FR Doc. 97–26665 Filed 10–7–97; 8:45 am]

BILLING CODE 6717–01–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–766; FRL 5746–9]

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–766, must be received on or before November 7, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION'' of this document. 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 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
Cynthia Giles-Parker, (PM 22).	Rm. 247, CM #2, 703–305–7740; e-mail: giles-parker cynthia@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224; e-mail: miller joanne@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–766] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number (PF-766) and appropriate petition number. Electronic comments on 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: September 29, 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. AgrEvo USA Company (AgrEvo)

PP 7F4910 and 7E4911

EPA has received pesticide petitions (PP 7F4910 and 7E4911) from AgrEvo USA Company (AgrEvo), Wilmington, DE 19808 proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.473(c) and part 186 by establishing tolerances for residues of glufosinate-ammonium in or on raw agricultural commodities derived from transgenic sugar beets and canola that are tolerant to the herbicide, glufosinate-ammonium: sugar beet roots at 0.7 ppm, sugar beet tops (leaves) at 1.3 ppm, canola seed at 0.4 ppm and the processed feeds: canola meal at 2.0 ppm and sugar beet molasses at 5.0 ppm. AgrEvo has also proposed to amend 40 CFR 180.473(a)(1) and part 185 by establishing a tolerance for residues of the herbicide, glufosinate-ammonium: butanoic acid, 2-amino-4-(hydroxymethylphosphinyl)-, monoammonium salt and its metabolite, 3-methylphosphinico-propionic acid expressed as glufosinate free acid

equivalents in or on the following raw agricultural commodity: potatoes at 0.4 ppm and the processed foods: potato flakes at 1.3 ppm and processed potatoes (including potato chips) at 1.0 ppm. The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by gas chromatography. 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.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of glufosinate-ammonium in plants is adequately understood for the purposes of these tolerances. The crop residue profile following selective use of glufosinate-ammonium on transgenic crops is different than that found in conventional crops. The only crop residue found after non-selective use is the metabolite, 3-methylphosphinico-propionic acid, which is found in only trace amounts. The principal residue identified in the metabolism studies after selective use of glufosinate-ammonium on transgenic crops is the acetylated derivative of the parent material, 2-acetamido-4-methylphosphinico-butanoic acid, with lesser amounts of glufosinate and 3-methylphosphinico-propionic acid.

2. *Analytical method*. There is a practical analytical method utilizing gas chromatography for detecting and measuring levels of glufosinate-ammonium and its metabolites in or on food with a general limit of quantification of 0.05 ppm. This method allows monitoring of food with residues at or above the levels proposed in these tolerances. This method has been validated by an independent laboratory and the petitioner has been advised that the EPA concluded its own successful method try out.

3. *Magnitude of residues*. Field residue trials with glufosinate-ammonium tolerant sugar beets and canola have been conducted in 1995 and 1996 and 1993 and 1994 respectively at several different use rates and timing intervals to represent the use patterns which would most likely result in the highest residue. In these trials, the primary residue in all samples was the combination of glufosinate and 2-acetamido-4-methylphosphinico-butanoic acid which was typically found at higher levels than 3-methylphosphinico-propionic acid. In sugar beets, the mean glufosinate-ammonium derived residues in treated roots did not exceed 0.70 ppm in trials conducted at 13 different sites representing the 6 major sugar beet producing regions in the U.S. The mean glufosinate-ammonium derived residues in treated tops (leaves) in these trials did not exceed 1.29 ppm when sampled at 60 days or more after treatment.

In canola, 11 out of 40 samples produced detectable residue levels above the limit of detection in harvested seed following treatment with glufosinate-ammonium at 14 trial locations. The highest level of residue found in these trials was 0.295 ppm and the total mean glufosinate derived residues in all samples containing detectable residues was 0.136 ppm.

For both sugar beet and canola, the tolerances levels have been proposed assuming the following: (1) a seasonal maximum rate of 1.1 pounds of active ingredient per acre for sugar beets and 0.9 pound of active ingredient per acre for canola and (2) a pre-harvest interval of 60 days for sugar beets and 65 days for canola.

Total residues of glufosinate-ammonium and its metabolite in potatoes desiccated with glufosinate-ammonium were determined in more than 40 trials conducted over approximately 13 locations during the period from 1987 to 1994. Within the pre-harvest interval of 7 to 56 days, all residue values (with one exception) did not exceed 0.4 ppm. A pre-harvest interval of 9 days is specified on the product label for potato desiccation and the seasonal maximum use rate is 0.4 pound of active ingredient per acre.

4. *Residue in processed commodities*. Studies have been conducted to determine the level of glufosinate derived residues found in or on the processed commodities from glufosinate tolerant sugar beet roots, canola seed and potatoes. The studies utilized treatments at significantly exaggerated rates to provide the necessary test sensitivity. In the sugar beet processing study, a concentration factor of 6.3x was determined for sugar beet molasses whereas there was no concentration of residues in either refined sugar or dried pulp.

In the canola processing study, a concentration factor of approximately 4 times was observed for the meal when the levels of terminal residues were compared between the seed and the toasted meal. There was no concentration of residues in the canola oil.

In the potato processing study, glufosinate residues appear to concentrate 2.3 fold in chips and 3.1 fold in flakes. Glufosinate residues do not appear to concentrate in the peel.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral LD_{50} values for glufosinate-ammonium technical ranged from 1,510 to 2,000 mg/kg in rats and from 200 to 464 mg/kg in mice and dogs. The acute dermal LD_{50} was 2,000 mg/kg in rabbits and ≥ 4,000 mg/kg in rats. The 4–hour rat inhalation LC_{50} was 1.26 mg/L in males and 2.6 mg/L in females. Glufosinate-ammonium was not irritating to rabbit skin but was slightly irritating to the eyes. Glufosinate-ammonium did not cause skin sensitization in guinea pigs. Glufosinate-ammonium should be classified as Tox Category II for oral toxicity, Tox Category III for inhalation and dermal toxicity and Tox Category IV for skin irritation and eye irritation.

2. *Genotoxicty*. No evidence of genotoxicity was noted in an extensive battery of *in vitro* and *in vivo* studies. The petitioner has been advised by the EPA that negative studies determined acceptable included *Salmonella, E. Coli* and mouse lymphoma gene mutation assays, a mouse micronucleus assay, and an *in vitro* UDS assay.

3. *Reproductive and developmental toxicity*. Three developmental toxicity studies were conducted with rats, at dose levels ranging from 0.5 to 250 mg/kg/day. The No Observed Effect Levels (NOEL's) for maternal and developmental effects were determined to be 10 mg/kg/day for maternal toxicity and 50 mg/kg/day for developmental toxicity, based on the findings of hyperactivity and vaginal bleeding in dams at 50 mg/kg/day and increased incidence of arrested renal and ureter development in fetuses at 250 mg/kg/day.

A developmental toxicity study was conducted in rabbits at dose levels of 0, 2, 6.3 and 20 mg/kg/day. The maternal NOEL for this study was determined to be 6.3 mg/kg/day, based on increases in abortion and premature delivery, and decreases in food consumption and weight gain at 20 mg/kg/day. No evidence of developmental toxicity was noted at any dose level; thus the developmental NOEL was determined to be 20 mg/kg/day.

A 2-generation rat reproduction study was conducted at dietary concentrations of 0, 40, 120 and 360 ppm. The parental NOEL was determined to be 40 ppm (4 mg/kg/day) based on increased kidney weights at 120 ppm. The NOEL for reproductive effects was determined to be 120 ppm (12 mg/kg/day) based on reduced numbers of pups at 360 ppm.

4. *Subchronic toxicity*. A 90–day feeding study was conducted in Fisher

344 rats at dietary concentrations of 0, 8, 64, 500 and 4,000 ppm. Although slight evidence of toxicity was observed, there were no treatment-related histopathological findings at any dose level. The NOEL for this study was determined to be 8 ppm, based on increased kidney weights at 64 ppm.

A 90–day feeding study was conducted in NMRI mice at dietary concentrations of 0, 80, 320 and 1,280 ppm. There were no treatment-related pathological findings at any dose level but increases in absolute and relative liver weights, serum AST, and serum potassium levels were noted at 320 and/or 1,280 ppm. Based on these findings, the NOEL for this study was determined to be 80 ppm (16.6 mg/kg/day).

A 90–day feeding study was conducted in beagle dogs at dietary concentrations of 0, 4, 8, 16, 64 and 256 ppm. There were no treatment-related histopathological findings at any dose level. However, because of reduced weight gain and decreased thyroid weights at 64 and/or 256 ppm, the NOEL was determined to be 16 ppm (0.53 mg/kg/day).

5. *Chronic toxicity*. A 12–month feeding study was conducted in beagle dogs at dose levels of 0, 2, 5 and 8.5 mg/kg/day. The NOEL was 5 mg/kg/day based on clinical signs of toxicity, reduced weight gain and mortality at 8.5 mg/kg/day.

A 2–year mouse oncogenicity study was conducted in NMRI mice at dietary concentrations of 0, 20, 80 and 160 (males) or 320 (females) ppm. The NOEL was determined to be 80 ppm (10.8 and 16.2 mg/kg/day for males and females, respectively) based on increased blood glucose, decreased glutathione levels and increased mortality in the high-dose males and/or females. No evidence of oncogenicity was noted at any dose level.

A combined chronic toxicity/oncogenicity study was conducted in Wistar rats for up to 130 weeks at dietary concentrations of 0, 40, 140 and 500 ppm. A dose-related increase in mortality was noted in females at 140 and 500 ppm, while increased absolute and relative kidney weights were noted in 140 and 500 ppm males. Thus, the NOEL for this study was determined to be 40 ppm (2.1 mg/kg/day). No treatment-related oncogenic response was noted. However, the high-dose level in this study did not satisfy the EPA criteria for a Maximum Tolerated Dose and thus a data gap currently exists for a rat carcinogenicity study. All glufosinate-ammonium tolerances previously established by the EPA are time-limited because of this gap. A new rat oncogenicity study is currently being conducted and is due to the EPA by July 1, 1998.

6. *Animal metabolism*. Numerous studies have been conducted to evaluate the absorption, distribution, metabolism and/or excretion of glufosinate-ammonium in rats. These studies indicate that glufosinate-ammonium is poorly absorbed (5–10%) after oral administration and is rapidly eliminated, primarily as parent compound. Small amounts of the metabolites 3-methylphosphinico-propionic acid and 2-acetamido-4-methylphosphinico-butanoic acid were found in the excreta, although the latter is believed to be a result of a revisable acetylation and decetylation process by intestinal bacteria.

7. *Metabolite toxicology* . The primary residue resulting from the use of glufosinate-ammonium in genetically transformed sugar beets and canola that are tolerant to the herbicide, glufosinate-ammonium, consists of the metabolites, 2-acetamido-4-methylphosphinico-butanoic acid and 3-methylphosphinico-propionic acid. Only the latter metabolite is formed in conventional crops. A considerable number of toxicity studies have been conducted with these metabolites, including developmental toxicity studies in rats and rabbits with both metabolites and a 2-generation rat reproduction study with 2-acetamido-4-methylphosphinico-butanoic acid. Neither metabolite presents an acute toxicity hazard and both were determined to be non-genotoxic in an extensive battery of *in vitro* and *in vivo* genotoxicity studies. Neither metabolite demonstrated significant developmental toxicity to either rats or rabbits. Subchronic studies in rats, mice and dogs were conducted with both metabolites with no clear evidence for any specific target organ toxicity and with NOEL's or No Observed Adverse Effects Levels (NOAEL's) substantially higher than those seen with glufosinate-ammonium. Thus, these studies indicate that both metabolites are less toxic than the parent compound and do not pose any reproductive or developmental concerns.

C. Endocrine Effects

No special studies investigating potential estrogenic or endocrine effects of glufosinate-ammonium 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. These studies are generally considered to be sufficient to detect any endocrine effects but no such effects were noted in any of the studies with either glufosinate-ammonium or its metabolites.

D. Aggregate Exposure

Glufosinate-ammonium is a non-selective, post-emergent herbicide with both food and non-food uses. As such, aggregate non-occupational exposure would include exposures resulting from consumption of potential residues in food and water, as well as from residue exposure resulting from non-crop use around trees, shrubs, lawns, walks, driveways, etc. Thus, the possible human exposure from food, drinking water and residential uses has been assessed below.

1. *Food*. For purposes of assessing the potential dietary exposure from food under the proposed tolerances, the petitioner has been advised that the EPA has estimated exposure based on the Theoretical Maximum Residue Contribution (TMRC) derived from the initially established tolerances for glufosinate-ammonium on apples, grapes, tree nuts, bananas, milk and the fat, meat and meat-by-products of cattle, goats, hogs, horses and sheep as well as the subsequently established tolerances for glufosinate-ammonium on field corn, soybeans, aspirated grain fractions, and the eggs, fat, meat and meat-by-products of poultry. The TMRC is obtained by using a model which multiplies the tolerance level residue for each commodity by consumption data which estimate the amount of each commodity and products derived from the commodity that are eaten by the U.S. population and various population subgroups. In conducting this exposure assessment, the EPA has made very conservative assumptions—100% of all commodities will contain glufosinate-ammonium residues and those residues would be at the level of the tolerance—which result in a large overestimate of human exposure. Thus, in making a safety determination for these tolerances, the Agency took into account this very conservative exposure assessment. In 62 FR 5333 (February 5, 1997), the Agency concluded that the original tolerances for apples, nuts, grapes and the secondary tolerances in animal commodities utilize 2.07% of the Reference Dose (RfD) and that the subsequent tolerances for the corn and soybean commodities will utilize 3.7% of the RfD.

2. *Drinking water*. There is presently no EPA Lifetime Health Advisory level or Maximum Contaminant Level established for residues of glufosinate-ammonium in water. The petitioner has

been advised by the EPA that all environmental fate data requirements for glufosinate-ammonium have been satisfied. The potential for glufosinate-ammonium to leach into groundwater has been assessed in a total of nine terrestrial field dissipation studies conducted in several states and in varying soil types. The degradation of glufosinate-ammonium in these studies was rapid, with half-lives ranging from a low of 6 to a high of 23 days. Despite the relatively high water solubility of glufosinate-ammonium, this compound did not appear to leach under typical test conditions. This is a result of the combination of its rapid degradation and its tendency to bind to certain soil elements such as clay or organic matter. Based on these studies and the expected conditions of use, the potential for finding significant glufosinate-ammonium residues in water is minimal and the contribution of any such residues to the total dietary intake of glufosinate-ammonium will be negligible.

3. *Non-dietary exposure*. As a non-selective, post-emergent herbicide, homeowner use of glufosinate-ammonium will consist primarily of spot spraying of weeds around trees, shrubs, walks, driveways, flower beds, etc. There will be minimal opportunity for post-application exposure since contact with the treated weeds will rarely occur. Thus, any exposures to glufosinate-ammonium resulting from homeowner use will result from dermal exposure during the application and will be limited to adults, not to infants or children. These exposures are not expected to pose any acute toxicity concerns. Furthermore, based on the US EPA National Home and Garden Pesticide Use Survey (RTI/5100/17-01F, March 1992), the average homeowner is expected to use non-selective herbicides only about 4 times a year. Thus, these exposures would not normally be factored into a chronic exposure assessment.

E. Cumulative Effects

The potential for cumulative effects of glufosinate-ammonium and other substances that have a common mechanism of toxicity must also be considered. The precise mechanism of action for the toxic effects of glufosinate-ammonium in animals is not known but is believed to result, at least in part, from interference with the neurotransmitter function of glutamate, to which it is a close structural analog. No other registered active ingredients are known to have a similar mechanism of action. Thus, no cumulative effects with other substances are anticipated. Furthermore, the residues in or on transgenic crops will consist primarily of the metabolites of glufosinate-ammonium, not glufosinate-ammonium itself. These metabolites are less toxic than glufosinate-ammonium and, because they are not structural analogs of glutamate, they should not cause the same effects. Thus, consideration of a common mechanism of toxicity is not appropriate at this time and only the potential risks of glufosinate-ammonium need to be considered in its aggregate exposure assessment.

F. Safety Determinations

1. *U.S. population*. Based on a complete and reliable toxicity database, the EPA has adopted an RfD value of 0.02 mg/kg/day using the NOEL of 2.1 mg/kg/day from the chronic rat toxicity study and a 100-fold safety factor. Using the Dietary Risk Evaluation System (DRES) with raw agricultural commodity residue values set at the established and proposed tolerance levels and with reasonable maximum market share estimates applied (''realistic'' case assessment), AgrEvo has calculated that aggregate dietary exposure to glufosinate-ammonium from the previously established tolerances and the proposed tolerances on sugar beets, canola and potatoes will utilize 2.1% of the RfD for the U.S. population (48 states). There is generally 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. Therefore, there is a reasonable certainty that no harm will result from aggregate exposure to glufosinate-ammonium residues to the U.S. population in general.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of glufosinate-ammonium, one should consider data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during pre-natal development. Reproduction studies provide information relating to reproductive and other effects on adults and offspring from pre-natal and post-natal exposure to the pesticide.

Three developmental toxicity studies in rats (including pre- and post-natal phases), a developmental toxicity study in rabbits, and a 2-generation rat reproduction study have been conducted with glufosinate-ammonium. No evidence of developmental toxicity was noted in rabbits, even at the maternally toxic dose level of 20 mg/kg/day. No developmental or reproductive effects were noted in rats except at parentally toxic dose levels. The NOEL's for maternal and developmental toxicity in the rat developmental toxicity studies were determined to be 10 mg/kg/day and 50 mg/kg/day, respectively, based on findings of hyperactivity and vaginal bleeding in dams at 50 mg/kg/day and increased incidence of arrested renal and ureter development in fetuses at 250 mg/kg/day. The parental and reproductive NOEL's in the 2-generation rat reproduction study were determined to be 40 ppm (4 mg/kg/day) and 120 ppm (12 mg/kg/day), respectively, based on increased parental kidney weights at 120 ppm and decreased numbers of pups at 360 ppm. In all cases, the reproductive and developmental NOEL's were greater than or equal to the parental NOEL's, thus indicating that glufosinate-ammonium does not pose any increased risk to infants or children.

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 toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. Further, the NOEL at 2.1 mg/kg/day from the chronic rat study with glufosinate-ammonium, which was used to calculate the RfD (discussed above), is already lower than the NOEL's from the reproductive and developmental studies with glufosinate-ammonium by a factor of at least 6-fold. Therefore, an additional safety factor is not warranted and an RfD of 0.02 mg/kg/day is appropriate for assessing aggregate risk to infants and children.

Using the DRES analysis with raw agricultural commodity residue values set at the established and proposed tolerance levels and with reasonable maximum market share estimates applied (''realistic'' case assessment), AgrEvo has calculated that aggregate dietary exposure to glufosinate-ammonium from the previously established tolerances and the proposed tolerances on sugar beets, canola and potatoes will utilize 5.5% of the RfD for non-nursing infants (1– year old), the most sensitive population sub-group and 5.3% of the RfD for children (1–6 year old), the second most sensitive population sub-group. Therefore, based on the completeness and reliability of the toxicity data and a comprehensive exposure assessment, it may be concluded that there is a reasonable certainty that no harm will result to

infants and children from aggregate exposure to glufosinate-ammonium residues.

G. International Tolerances

An analysis of the Codex Alimentarius Commission (Codex) tolerances has been conducted. While no international Codex tolerances for selective uses of tolerances for glufosinate-ammonium in the desiccation use pattern have been established for conventional canola (rapeseed) at 5 ppm, crude rapeseed oil at 0.05 ppm and potatoes at 0.5 ppm. These tolerances are established for the sum of glufosinate-ammonium and 3-methylphosphinico-propionic acid, calculated as glufosinate (free acid). The U.S. proposal for a 0.4 ppm tolerance for residues of glufosinate-ammonium in potatoes will be harmonized with the Canadian tolerance which has already been established at this level.

The Codex tolerances for glufosinate-ammonium in or on sugar beets have been established at 0.05 ppm in the beet and 0.1 ppm in the tops (leaves). AgrEvo intends to propose higher tolerances to the Codex commission for glufosinate-ammonium use on transgenic sugar beets in order to harmonize these tolerances with those proposed in the U.S. and elsewhere. (Joanne Miller)

2. K-I Chemical U.S.A., Inc.

PP 7F4821

EPA has received an amendment to pesticide petition (PP 7F4821) from K-I Chemical U.S.A., Inc. , White Plains, New York 10606, 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 herbicide, fluthiacet-methyl in or on the raw agricultural commodity popcorn grain at 0.02 ppm.

On April 14, l997, EPA announces receipt of a pesticide petition (PP 7F4821) from K-I Chemical U.S.A., Inc., 11 Martine Avenue, 9th Floor, White Plains, NY 10606, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the herbicide fluthiacet-methyl: Acetic acid, [[2-chloro-4-fluoro-5-[(tetrahydro-3-oxo-1*H*,3*H*-[1,3,4]thiadiazolo[3,4-*a*] pyridazin-1-ylidene)amino]phenyl]thio]-methylester in or on the raw agricultural commodities field corn grain and sweet corn grain (K + CWHR) at 0.02 ppm and corn forage and fodder at 0.05 ppm.

On September 4, 1997 K-I Chemical, U.S.A., Inc., amended PP 7F4821 to include a proposed tolerance for popcorn grain at 0.02 ppm. EPA has determined that the amended petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residues in corn is adequately understood following application of fluthiacet-methyl. Residue levels and the metabolic pathway are consistent with those in soybeans. Parent fluthiacet-methyl was the primary component of the residue seen in corn grain, forage, fodder and silage. Results of these studies have been submitted to the EPA.

2. *Analytical method*. K-I Chemical has submitted practical analytical methods (AG–603B and AG–624) for detecting and measuring the level of fluthiacet-methyl in or on corn and corn commodities and in animal tissues with a limit of detection that allows monitoring residues at or above the levels set for the proposed tolerance. The limit of quantitation of the crop method is 0.01 ppm in corn and corn commodities, 0.05 ppm in animal tissues and 0.01 ppm in milk. The crop method involves extraction, filtration, and solid phase clean up. Residue levels of fluthiacet-methyl are determined by gas chromatographic analysis utilizing a nitrogen phosphorus detector and a fused-silica column. The animal tissue method involves extraction, filtration, and partition. Determination of residue levels in animal tissues is by HPLC with UV detection via column switching using C1 and C18 columns. The analyte of interest in animal tissues and milk is the major animal metabolite CGA–300403. Residues of fluthiacet-methyl in corn are determined by gas chromatography.

3. *Magnitude of residues*. The residue of concern in corn is fluthiacet-methyl per se. Twenty-one field residue studies were conducted with corn grown in nineteen states. Fifteen of the studies were on field corn and six on sweet corn. No studies were conducted with popcorn, however K-I believes that the data on field and sweet corn support a tolerance in popcorn as well. Because the proposed use rate and pattern is the same for popcorn, it is reasonable to conclude that residues in popcorn grain will not exceed the proposed tolerance of 0.02 ppm. Residues in field and sweet corn forage after the day of application were less than the proposed tolerance of 0.05 ppm. Popcorn forage is not a fed commodity. Nonetheless, residues in popcorn forage or fodder are not expected to exceed the proposed tolerance of 0.05 ppm. The proposed tolerances of 0.02 ppm in field corn, sweet corn, and popcorn grain and 0.05 ppm in field corn and sweet corn forage and fodder are adequate to cover residues likely to occur when Action herbicide is applied to corn as directed.

This position is based on section 180.34(d) of the CFR which states that ''If the pesticide chemical is not absorbed into the living plant or animal when applied (is not systemic), it may be possible to make a reliable estimate of the residues to be expected on each commodity in a group of related commodities on the basis of less data than would be required for each commodity in the group, considered separately.'' And, section 180.34(e) states that ''Each of the following groups of crops lists raw agricultural commodities that are considered to be related for the purpose of paragraph (d) of this section; field corn, popcorn, sweet corn (each in grain form).''

Residues of fluthiacet-methyl in treated field and sweet corn grain and sweet corn ears were less than the method LOQ (<0.01 ppm). Because the proposed use rate and pattern is the same for popcorn, it is reasonable to conclude that residues in popcorn grain will not exceed the proposed tolerance of 0.02 ppm. Residues in field and sweet corn forage after the day of application were less than the proposed tolerance of 0.05 ppm. Popcorn forage is not a feed commodity. Nonetheless, residues in popcorn forage or fodder are not expected to exceed the proposed tolerance of 0.05 ppm. The proposed tolerances of 0.02 ppm in field corn, sweet corn, and popcorn grain and 0.05 ppm in field corn and sweet corn forage and fodder are adequate to cover residues likely to occur when Action herbicide is applied to corn as directed.

B. Toxicological Profile

1. *Acute toxicity*.

- A rat acute oral study with an LD_{50} > 5,000 mg/kg.
- A rabbit acute dermal study with an LD_{50} > 2,000 mg/kg.
- A rat inhalation study with an LC_{50} > 5.05 mg/liter.
- A primary eye irritation study in the rabbit showing moderate eye irritation.
- A primary dermal irritation study in the rabbit showing no skin irritation.
- A primary dermal sensitization study in the Guinea pig showing no sensitization.

•Acute neurotoxicity study in rats. Neurotoxic effects were not observed. The NOEL was 2,000 mg/kg.

2. *Genotoxicity*. In vitro gene mutation tests: Ames test - negative; Chinese hamster V79 test - negative; rat hepatocyte DNA repair test - negative; E. Coli letal DNA damage test - negative. In vitro chromosomal aberration tests: Chinese hamster ovary - positive at cytotoxic doses; Chinese hamster lung - positive at cytotoxic doses; human lymphocyes - positive at cytotoxic doses. In vivo chromosome aberration tests: Micronucleus assays in rat liver - negative; mouse bone marrow test - negative.

3. *Reproductive and developmental toxicity*. Reproductive and developmental toxicity. Teratology study in rats with a maternal and developmental NOEL equal to or greater than 1,000 mg/kg/day.

Teratology study in rabbits with a maternal NOEL greater than or equal to 1,000 mg/kg/day and a fetal NOEL of 300 mg/kg based on a slight delay in fetal maturation. 2-generation reproduction study in rats with a NOEL of 36 mg/kg/day, based on liver lesions in parental animals and slightly reduced body weight development in parental animals and pups. [The treatment had no effect on reproduction or fertility.]

4. *Subchronic toxicity*. 90–day subchronic neurotoxicity study in rats. The NOEL was 0.5 mg/kg/day based on reduced body weight gain. No clinical or morphological signs of neurotoxicity were detected at any dose level. 28–day dermal toxicity study in rats with a NOEL equal to or higher than the limit dose of 1,000 mg/kg.

6–week dietary toxicity study in dogs with a NOEL of 162 mg/kg/day in males and 50 mg/kg/day in females based on decreased body weight gain and modest hematological changes.

90–day subchronic dietary toxicity study in rats with a NOEL of 6.2 mg/kg/day based on liver changes and hematological effects.

5. *Chronic toxicity*. 24–month combined chronic toxicity/carcinogenicity study in rats with a NOEL of 2.1 mg/kg/day. Based on reduced body weight development and changes in bone marrow, liver, pancreas and uterus the MTD was exceeded at 130 mg/kg/day. A positive trend of adenomas of the pancreas in male rats treated at 130 mg/kg/day and above may be attributable to the increased survival of the rats treated at high doses. 18–month oncogenicity study in mice with a NOEL of 0.14 mg/kg/day. Based on liver changes, the MTD was reached at 1.2 mg/kg/day. The incidence of hepatocellular tumors was increased in males treated at 12 and 37 mg/kg/day.

C. Endocrine effects

Based on the results of short-term, chronic, and reproductive toxicity studies there is no indication that fluthiacet-methyl might interfere with the endocrine system. Considering further the low environmental concentrations and the lack of bioaccumulation, there is no risk of endocrine disruption in humans or wildlife.

Animal metabolism. The results from hen and goat metabolism studies, wherein fluthiacet-methyl was fed at exaggerated rates, showed that the transfer of fluthiacet-methyl residues from feed to tissues, milk and eggs is extremely low. No detectable residues of fluthiacet-methyl (or metabolite CGA–300403) would be expected in meat, milk, poultry, or eggs after feeding the maximum allowable amount of treated corn and soybeans. This conclusion is based on residue data from the corn and soybean metabolism and field residue chemistry studies coupled with the residue transfer from feed to tissues, milk and eggs obtained in the goat and hen metabolism studies.

D. Aggregate Exposure

Aggregate exposure includes exposure from dietary exposure from food and drinking water; and non-dietary exposure from non-dietary uses of pesticides products containing the active ingredient, fluthiacet-methyl.

1. *Dietary exposure*. Dietary exposure consists of exposures from food and drinking water.

2. *Food*. In this assessment, K-I Chemical has conservatively assumed that 100% of all soybeans and corn used for human consumption would contain residues of fluthiacet-methyl and all residues would be at the level of the proposed tolerances. The potential dietary exposure to fluthiacet-methyl was calculated on the basis of the proposed tolerance which is based on an LOQ of 0.01 ppm in soybeans and 0.02 ppm in corn (2 x LOQ). The anticipated residues in milk, meat and eggs resulting from feeding the maximum allowable amount of soybean and corn commodities to cattle and poultry were calculated, and the resulting quantities were well below the analytical method LOQ. Therefore, tolerances for milk, meat and eggs are not required. Assuming 100% crop treated values, the chronic dietary exposure of the general U.S. population to fluthiacet-methyl would correspond to 2.3% of the RfD.

3. *Drinking water*. Although fluthiacet-methyl has a slight to medium leaching potential; the risk of the parent compound to leach to deeper soil layers is negligible under practical conditions in view of the fast degradation of the product. For example, the soil metabolism half-life was extremely short, ranging from 1.1 days under aerobic conditions to 1.6 days under an aerobic conditions. Even in the event of very heavy rainfalls immediately after application, which could lead to a certain downward movement of the parent compound, parent fluthiacet-methyl continues to be degraded during the transport into deeper soil zones. Considering the low application rate of fluthiacet-methyl, the strong soil binding characteristics of fluthiacet-methyl and its degradates, and the rapid degradation of fluthiacet-methyl in the soil, there is no risk of ground water contamination with fluthiacet-methyl or its metabolites. Thus, aggregate risk of exposure to fluthiacet-methyl does not include drinking water

4. *Non-dietary exposure*. Fluthiacet-methyl is not registered for any other use and is only proposed for use on agricultural crops. Thus, there is no potential for non-occupational exposure other than consumption of treated commodities containing fluthiacet-methyl residue.

E. Cumulative Effects

A cumulative exposure assessment is not appropriate at this time because there is no information available to indicate that effects of fluthiacet-methyl in mammals would be cumulative with those of another chemical compound.

F. Safety Determination

1. *U.S. population*. Using the very conservative exposure assumptions described above coupled with toxicity data for fluthiacet-methyl, K-I Chemical calculated that aggregate, chronic exposure to fluthiacet-methyl will utilize no more than 2.3% of the RfD for the U.S. population. Because the actual anticipated residues are well below tolerance levels and the percent crop treated with fluthiacet-methyl is expected to be less than 25% of planted corn or soybeans, a more realistic estimate is that dietary exposure will likely be at least 20 times less than the conservative estimate previously noted (the margins of exposure will be accordingly higher). Exposures below 100% of the RfD are generally not of concern 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.

Also the acute dietary risk to consumers will be far below any significant level; the lowest NOEL from a short term exposure scenario comes from the teratology study in rabbits with a NOEL of 300 mg/kg. This NOEL is 2,000-fold higher than the chronic NOEL which provides the basis for the RfD (see above). Acute dietary exposure estimates which are based on a combined food survey from 1989 to 1992 predict margins of exposure of at least one million for 99.9% of the general population and for women of child bearing age. Margins of exposure of 100 or more are generally considered satisfactory. Therefore, K-I Chemical concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fluthiacet-methyl residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of fluthiacet-methyl, K-I Chemical considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. A slight delay in fetal maturation was observed in a teratology study in rabbits at a daily dose of 1,000 mg/kg. In a 2-generation reproduction study fluthiacet-methyl did not affect the reproductive performance of the parental animals or the physiological development of the pups. The NOEL was 500 ppm for maternal animals and their offspring, which is 50,000 fold higher than the RfD.

3. *Reference dose*. Using the same conservative exposure assumptions as was used for the general population, the percent of the RfD that will be utilized by aggregate exposure to residues of fluthiacet-methyl is as follows: 1.5% for nursing infants less than 1 year old, 5.9% for non-nursing infants, and 5.2% for children 1–6 years old. K-I Chemical concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to residues of fluthiacet-methyl.

G. International Tolerances

No international tolerances have been established under CODEX for fluthiacet-methyl. (Joanne Miller)

3. Zeneca Ag Products

PP 7F4864

EPA has received a pesticide petition (PP 7F4864) from Zeneca Ag Products, 1800 Concord Pike, P.O. Box 15458, Wilmington, DE 19850-5458] proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.507 by establishing a tolerance for residues of azoxystrobin (methyl(E)-2-(2-(6-(2-cyanophenoxy)pyrimidin-4-yloxy)phenyl)-3- methoxyacrylate) and the Z-isomer of azoxystrobin (methyl(Z)-2-(2-(6-(2-cyanophenoxy) pyrimidin-4-yloxy)phenyl)-3-methoxyacrylate)] in or on the raw agricultural commodities almond hulls at 4.0 ppm, cucurbits (chayotes, Chinese waxgourds, citron melons, cucumbers, gherkins, edible gourds, Mordica spp., cantaloupes, casabas, crenshaw melons, golden pershaw melons, honeydew melons, honey balls, mango melons, Persian melons, summer squashes, winter squashes, and watermelons) at 0.3 ppm, peanut hay at 1.5 ppm, pistachios at 0.01 ppm, rice grain at 4.0 ppm, rice hulls at 20 ppm, rice straw at 11 ppm, tree nuts (almonds, beech nuts, Brazil nuts, butternuts, cashews, chestnuts, chinquapins, filberts, hickory nuts, macadamia nuts, pecans, and walnuts) at 0.01 ppm, wheat bran at 0.12 ppm, wheat grain at 0.04 ppm, wheat hay at 13.0 ppm, and wheat straw at 4.0 ppm. It is also proposed that 40 CFR 180.507 be amended by establishment of a tolerance for the residues of azoxystrobin (methyl (*E*)-2-[2-[6-(2-cyanophenoxy)pyrimidin-4-yloxy]phenyl]-3-methoxyacrylate) in or on the following animal products: eggs at 0.4 ppm, cattle kidney at 0.06 ppm, liver of cattle, goat, horse, and sheep at 0.3 ppm, hog liver at 0.2 ppm, poultry liver at 0.4 ppm, meat and fat of cattle, goat, horse, sheep, poultry and swine at 0.01 ppm, and milk at 0.006 ppm. The proposed analytical methods use gas chromatography with nitrogen-phosphorous detection (GC-NPD) or, in mobile phase, high performance liquid chromatography with ultraviolet detection (HPLC-UV). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of azoxystrobin as well as the nature of the residues is adequately understood for purposes of the tolerances. Plant metabolism has been evaluated in three diverse crops, grapes, wheat, and peanuts, which should serve to define the similar metabolism of azoxystrobin in a wide range of crops. Parent azoxystrobin is the major component found in crops. Azoxystrobin does not accumulate in crop seeds or fruits. Metabolism of azoxystrobin in plants is complex, with more than 15 metabolites identified. These metabolites are present at low levels, typically much less than 5% of the TRR.

2. *Analytical method*. An adequate analytical method, gas chromatography with nitrogen-phosphorous detection (GC-NDP) or, in mobile phase, by high performance liquid chromatography with ultraviolet detection (HPLC-UV), is available for enforcement purposes with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances. The Analytical Chemistry Section of the EPA concluded that the method(s) are adequate for enforcement. Analytical methods are also available for analyzing meat, milk, poultry, and eggs and also underwent successful independent laboratory validations.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral toxicity study in rats of technical azoxystrobin resulted in an LD_{50} of >5,000 milligrams/kilogram (limit test) for both males and females. The acute dermal toxicity study in rats of technical azoxystrobin resulted in an LD_{50} of >2,000 milligrams/kilogram (limit dose). The acute inhalation study of technical azoxystrobin in rats resulted in an LC_{50} of 0.962 milligrams/liter in males and 0.698 milligrams/liter in females. In an acute oral neurotoxicity study in rats dosed once by gavage with 0, 200, 600, or 2,000 milligrams/ kilogram azoxystrobin, the systemic toxicity no observed effect level (NOEL) was 200 milligrams/kilogram and the systemic toxicity lowest observed effect level (LOEL) was 200 milligrams/kilogram, based on the occurrence of transient diarrhea in both sexes. There was no indication of neurotoxicity at the doses tested. This acute neurotoxicity study is considered supplementary (upgradable) but the data required are considered only to be confirmatory. Zeneca has submitted the required confirmatory data; these data have been scheduled for review by the Agency.

2. *Genotoxicty (mutagenicity)*. Azoxystrobin was negative for mutagenicity in the salmonella/ mammalian activation gene mutation assay, the mouse micronucleus test, and the unscheduled DNA synthesis in rat hepatocytes/mammalian cells (in vivo/ in vitro procedure study). In the forward mutation study using L5178 mouse lymphoma cells in culture, azoxystrobin tested positive for forward gene mutation at the TK locus. In the *in vitro* human lymphocytes cytogenetics assay of azoxystrobin, there was evidence of a concentration related induction of

chromosomal aberrations over background in the presence of moderate to severe cytotoxicity.

3. *Reproductive and developmental toxicity*. In a prenatal development study in rats gavaged with azoxystrobin at dose levels of 0, 25, 100, or 300 mg/kg/day during days 7 through 16 of gestation, lethality at the highest dose caused the discontinuation of dosing at that level. The developmental NOEL was greater than or equal to 100 mg/kg/day and the developmental lowest observed effect level (LOEL) was >100 mg/kg/day because no significant adverse developmental effects were observed. In this same study, the maternal NOEL was not established; the maternal LOEL was 25 mg/kg/day, based on increased salivation.

In a prenatal developmental study in rabbits gavaged with 0, 50, 150, or 500 mg/kg/day during days 8 through 20 of gestation, the developmental NOEL was 500 mg/kg/day and the developmental LOEL was >500 mg/kg/day because no treatment-related adverse effects on development were seen. The maternal NOEL was 150 mg/kg/day and the maternal LOEL was 500 mg/kg/day, based on decreased body weight gain.

In a 2-generation study, rats were fed 0, 60, 300, or 1,500 ppm of azoxystrobin. The reproductive NOEL was 32.2 mg/kg/day. The reproductive LOEL was 165.4 mg/kg/day. Reproductive toxicity was demonstrated as treatment-related reductions in adjusted pup body weights as observed in the F18 and F2. pups dosed at 1500 ppm (165.4 mg/kg/day).

4. *Subchronic toxicity*. In a 90-day rat feeding study the NOEL was 20.4 mg/kg/day for males and females. The LOEL was 211.0 mg/kg/day based on decreased weight gain in both sexes, clinical observations of distended abdomens and reduced body size, and clinical pathology findings attributable to reduced nutritional status.

In a subchronic toxicity study in which azoxystrobin was administered to dogs by capsule for 92 or 93 days, the NOEL for both males and females was 50 mg/kg/day. The LOEL was 250 mg/kg/day, based on treatment-related clinical observations and clinical chemistry alterations at this dose.

In a 21-day repeated-dose dermal rat study using azoxystrobin, the NOEL for both males and females was greater than or equal to 1,000 mg/kg/day (the highest dosing regimen); a LOEL was therefore not determined.

5. *Chronic toxicity and carcinogenicity*. In a 2–year feeding study in rats fed diets containing 0, 60, 300, and 750/1,500 ppm (males/females), the systemic toxicity NOEL was 18.2 mg/kg/day for males and 22.3 mg/kg/day for females. The systemic toxicity LOEL for males was 34 mg/kg/day, based on reduced body weights, food consumption, and food efficiency; and bile duct lesions. The systemic toxicity LOEL for females was 117.1 mg/kg/day, based on reduced body weights. There was no evidence of carcinogenic activity in this study.

In a 1–year feeding study in dogs to which azoxystrobin was fed by capsule at doses of 0, 3, 25, or 200 mg/kg/day, the NOEL for both males and females was 25 mg/kg/day and the LOEL was 200 mg/kg/day for both sexes, based on clinical observations, clinical chemistry changes, and liver weight increases that were observed in both sexes.

In a 2–year carcinogenicity feeding study in mice using dosing concentrations of 0, 50, 300, or 2,000 ppm, the systemic toxicity NOEL was 37.5 mg/kg/day for both males and females. The systemic toxicity LOEL was 272.4 mg/kg/day for both sexes, based on reduced body weights in both at this dose. There was no evidence of carcinogenicity at the dose levels tested.

According to the new proposed guidelines for Carcinogen Risk Assessment (April, 1996), the appropriate descriptor for human carcinogenic potential of azoxystrobin is ''Not Likely''. The appropriate subdescriptor is ''has been evaluated in at least two well conducted studies in two appropriate species without demonstrating carcinogenic effects''.

6. *Animal metabolism*. In the study of metabolism in the rat, azoxystrobin--unlabeled or with a pyrimidinyl, phenylacrylate, or cyanophenyl label--was administered to rats by gavage as a single or 14-day repeated doses. Less than 0.5% of the administered dose was detected in the tissues and carcass up to 7 days post-dosing and most of it was in excretion- related organs. There was no evidence of potential for bioaccumulation. The primary route of excretion was via the feces, though 9 to 18% was detected in the urine of the various dose groups. Absorbed azoxystrobin appeared to be extensively metabolized. A metabolic pathway was proposed showing hydrolysis and subsequent glucuronide conjugation as the major biotransformation process. This study was classified as supplementary but upgradable; the company has submitted data intended to upgrade the study and these data have been reviewed.

C. Dietary Exposure

1. *Food*. The primary route of human exposure to azoxystrobin is expected to be dietary ingestion of both raw and processed agricultural commodities from bananas, grapes, peaches, peanuts, tomatoes, tree nuts, pistachios, rice, cucurbits, and wheat. A chronic dietary exposure analysis (combined years 1989 - 1992 U.S. Department of Agriculture's Nationwide Food Consumption Survey using the Technical Assessment Systems, Inc. ''EXPOSURE 1'' software) was conducted using tolerance level residues and 100% crop treated information to estimate the TMRC for the general population and 22 subgroups.

2. *Drinking water*. There is no established Maximum Concentration Level for residues of azoxystrobin in drinking water. The potential exposures associated with azoxystrobin 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 proposed uses were granted.

3. *Non-dietary exposure*. The Agency evaluated the existing toxicological database for azoxystrobin and assessed appropriate toxicological endpoints and dose levels of concern that should be assessed for risk assessment purposes. Dermal absorption data indicate that absorption is less than or equal to 4%. No appropriate endpoints were identified for acute dietary or short term, intermediate term, and chronic term (noncancer) dermal and inhalation occupational or residential exposure. Therefore, risk assessments are not required for these exposure scenarios and there are no residential risk assessments to aggregate with the chronic dietary risk assessment.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' EPA does not have, at this time, available data to determine whether azoxystrobin 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, azoxystrobin does not appear to be structurally similar to any other pesticide chemical at this time. No metabolites of azoxystrobin that are of toxicological concern are known to the Agency. Azoxystrobin appears to the only pesticide member of its class of

chemistry and there are no reliable data to indicate that this chemical is structurally or toxicologically similar to existing chemical substances at this time. Therefore, it appears unlikely that azoxystrobin bears a common mechanism of activity with other substances. For the purposes of this tolerance action, it is not appropriate to assume that azoxystrobin has a common mechanism of toxicity with other substances.

E. Safety Determination

The chronic toxicity Reference Dose (RfD) for azoxystrobin is 0.18 mg/kg/day, based on the NOEL of 18.2 mg/kg/day from the rat chronic toxicity/carcinogenicity feeding study in which decreased body weight and bile duct lesions were observed in male rats at the LOEL of 34 mg/kg/day. This NOEL was divided by an Uncertainty Factor of 100, to allow for interspecies sensitivity and intraspecies variability.

1.As part of the hazard assessment process, the available toxicological database was reviewed to determine if there are toxicological endpoints of concern. For azoxystrobin, the Agency does not have a concern for acute dietary exposure since the available data do not indicate any evidence of significant toxicity from a 1–day or single event exposure by the oral route. Therefore, an acute dietary risk assessment is not required for azoxystrobin at this time.

2. *U.S. population*. The chronic dietary exposure analysis showed that exposure from the proposed new tolerances in or on tree nuts, pistachios, cucurbits, rice, and wheat for the general U.S. population would be 1.1% of the RfD. This analysis used a value of 0.05 ppm for banana pulp rather than the value of 0.5 that has been established for banana (whole fruit including peel) because adequate data were submitted to support use of the lower value in the dietary risk analyses.

3. *Infants and children*. The chronic dietary exposure analysis, using the same tolerances and commodities that were used for the same analysis for the general U.S. population showed that the exposure of Non-nursing Infants (the subgroup with the highest exposure) would be 4.1% of the RfD.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a margin of exposure analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. In either case, EPA generally defines the level of appreciable risk as exposure that is greater than 1/100th of the no observed effect level in the animal study appropriate to the particular risk assessment. This hundredfold uncertainty (safety) factor/margin of exposure (safety) is designed to account for combined inter- and intraspecies variability. EPA believes that reliable data support using the standard hundredfold margin/factor not the additional tenfold margin/factor when EPA has a complete database under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard margin/factor. The database for azoxystrobin is complete except that the acute and subchronic neurotoxicity studies require upgrading. The upgrade data are confirmatory only, have been submitted by the company, and await review by the Agency.

There was no evidence of increased susceptibility of infants or children to azoxystrobin. Therefore, no additional uncertainty factors are considered necessary at this time.

F. Endocrine Effects

EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect...''. The Agency is currently working with interested shareholders, including other government agencies, public interest groups, industry, and research scientists, to develop a screening and testing program and a priority setting scheme to implement this program. Congress has allowed three (3) years from the passage pf FQPA (August 3, 1999) to implement this program. When this program is implemented, EPA may require further testing of azoxystrobin and end-use product formulations for endocrine disrupter effects. There are currently no data or information suggesting that azoxystrobin has any endocrine effects.

G. International Tolerances

There are no Codex Maximum Residue Levels established for azoxystrobin. (Cynthia Giles-Parker)

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ENVIRONMENTAL PROTECTION AGENCY

[PF–765; FRL–5745–9]

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–765, must be received on or before November 7, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically 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: