DEPARTMENT OF ENERGY

Federal Energy Regulatory Commission

[Docket No. CP00–381–000]

Williams Gas Pipelines Central, Inc.; Notice of Request Under Blanket Authorization

June 15, 2000.

Take notice that on June 7, 2000, Williams Gas Pipelines Central, Inc. (Williams), P.O. Box 20008, Owensboro, Kentucky 42304, filed in Docket No. CP00–381–000 a request pursuant to sections 157.205 and 157.211 of the Commission's Regulations (18 CFR 157.205 and 157.211) under the Natural Gas Act (NGA) for authorization to construct and operate delivery point facilities for service to Quivira Realty, Inc. (Quivira), in Johnson County, Kansas, under Quivira's blanket certificate issued in Docket No. CP82–479–000, pursuant to section 7 of the NGA, all as more fully set forth in the application which is on file with the Commission and open to public inspection. This filing may be viewed on the web at http://www.ferc.fed..us/online/htm (call 202–208–2222 for assistance).

Williams requests authorization to construct and operate delivery point facilities to serve Quivira, which requires the gas for residential air conditioning use. It is stated that Williams will use the facilities to transport up to 12 Dt equivalent of natural gas per day on a firm basis pursuant to section 284.223 of the Commission's regulations. Williams estimates the cost of the facilities at $9,100 and states that it would be reimbursed for the cost by Quivira. It is asserted that Williams has sufficient capacity to render the proposed service without detriment or disadvantage to its other existing customers and that Williams' tariff does not prohibit the addition of delivery point facilities. It is further asserted that the proposal will have no significant impact on Williams' peak day and annual deliveries.

Any questions regarding the application may be directed to David N. Roberts, Manager of Certificates and Tariffs, at (270) 688–6712, Williams Gas Pipelines Central, Inc., P.O. Box 20008, Owensboro, Kentucky 42304.

Any person or the Commission's staff may, within 45 days after issuance of the instant notice by the Commission, file pursuant to Rule 214 of the Commission's Procedural Rules (18 CFR 385.214) a motion to intervene or notice of intervention and pursuant to section 157.205 of the Regulations under the NGA (18 CFR 157.205) a protest to the request. If no protest is filed within the time allowed therefor, the proposed activity shall be deemed to be authorized effective the day after the time allowed for filing a protest. If a protest is filed and not withdrawn within 30 days after the time allowed for filing a protest, the instant request shall be treated as an application for authorization pursuant to Section 7 of the NGA.

Linwood A. Watson, Jr.,

Acting Secretary.

[FR Doc. 00–15570 Filed 6–20–00; 8:45 am]

BILLING CODE 6717–01–M

ENVIRONMENTAL PROTECTION AGENCY

[PF–945; FRL–6558–9]

Notice of Filing a Pesticide Petition To Establish a Tolerance for Certain Pesticide Chemicals in or on Food

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 docket control number PF–945, must be received on or before July 21, 2000.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the ''SUPPLEMENTARY INFORMATION.'' To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–945 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Shaja R. Brothers, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–3194; e-mail address: brothers.shaja@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under ''FOR FURTHER INFORMATION CONTACT.''

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this action under docket control number PF–945. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m.,

Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–945 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically.* You may submit your comments electronically by e-mail to: ''*opp-docket@epa.gov*,'' or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–945. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI 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. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under ''FOR FURTHER INFORMATION CONTACT.''

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

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 support granting of the petitions. Additional data may be needed before EPA rules on the petitions.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: June 2, 2000.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

The 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 petitioner and represents the view of the petitioner. The petition summaries announce 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.

I. Interregional Research Project Number 4

9E6026

EPA has received pesticide petition 9E6026 from the Interregional Research Project Number 4 (IR–4), New Jersey Agricultural Experiment Station, Rutgers University, New Brunswick, New Jersey 08903, proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of paraquat in or on the raw agricultural commodity (RAC) endive at 0.05 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition. This notice includes a summary of the petition prepared by Zeneca Ag Products, the registrant, 1800 Concord Pike. P.O. Box 15458, Wilmington, DE 19850–5458.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residues in plants is adequately understood based on studies depicting the metabolism of paraquat in carrots and lettuce following pre-emergence treatments and in potatoes and soybeans following desiccant treatment. The residue of concern in plants is the parent chemical, paraquat.

2. *Analytical method*. An adequate analytical method (spectrometric method) has been accepted and published in the Pesticide Analytical Manual (PAM Vol. II) for the enforcement of tolerances in plant commodities.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicity studies conducted with the 45.6% paraquat dichloride technical concentrate give the following results: oral lethal dose $(LD)_{50}$ in the rat of 344 milligrams/kilograms (mg/kg) males and 283 mg/kg females Category II; dermal LD_{50} in the rat of >2,000 mg/kg for

males and females (Category III); the primary eye irritation study showed corneal involvement with clearing within 17-days (Category II); and dermal irritation of slight erythema and edema at 72 hours (Category IV). Paraquat is not a dermal sensitizer. Acute inhalation studies conducted pursuant to EPA guideline with aerosolized sprays result in lethal concentration $(LC)_{50}$ of 0.6 to 1.4 µg paraquat cation/L (Category I). However, since paraquat dichloride has no measurable vapor pressure; and hydraulic spray droplets are too large to be respirable, inhalation exposure is not a concern in practice.

2. *Genotoxicity*. Paraquat dichloride was not mutagenic in the Ames test using *Salmonella typhinurium* strains TA1535, TA1538, TA98, and TA100; the chromosomal aberrations in the bone marrow test system; or in the dominant lethal mutagenicity study with CD–1 mice. Additionally, paraquat dichloride was negative for unscheduled DNA synthesis in rat hepatocyctes *in vitro* and *in vivo*. Paraquat was weakly positive in the mouse lymphoma cell assay only in the presence of metabolic activation. Paraquat dichloride was weakly positive in mammalian cells (lymphocytes) and positive in the sister chromatid exchange (SCE) assay in chinese hamster lung fibroblasts. Paraquat is non-mutagenic.

3. *Reproductive and developmental toxicity*. A 3-generation reproduction study in rats fed diets containing 0, 25, 75, and 150 ppm (0, 1.25, 3.75, or 7.5 mg of paraquat cation/kg/day, respectively) showed no effect on body weight gain, food consumption and utilization, fertility and length of gestation of the F0, F1, and F2 parents at any dose. The no observed adverse effect level (NOAEL) and lowest observed adverse effect level (LOAEL) for systemic toxicity are 25 ppm (1.25 mg/kg/day) and 75 ppm (3.75 mg/kg/day), respectively, expressed as paraquat cation, based on high mortality due to lung damage. The NOAEL for reproductive toxicity is ≥150 ppm 7.5 mg/kg/day; highest dose tested (HDT) expressed as paraquat cation, as there were no reproductive effects observed.

Two developmental toxicity studies were conducted in rats given gavage doses of 0, 1, 5, or 10 mg/kg/day and 0, 1, 3, or 8 mg/kg/day, respectively, expressed as paraquat cation. In the first study, the NOAEL for maternal toxicity was 1 mg/kg/day based on clinical signs of toxicity and decreased body weight gain at 5 mg/kg/day (the LOAEL). The NOAEL for developmental toxicity was set at 5 mg/kg/day based on delayed ossification of the forelimb and hindlimb digits. In the second study, the maternal and developmental NOAEL is 8 mg/kg/day HDT as there were no effects observed at any dose level. Based on both studies, the overall NOAEL for maternal and developmental toxicity is at least 3 mg/kg/day.

Two developmental toxicity studies were conducted in mice given gavage doses of 0, 1, 5, or 10 mg/kg/day and 0, 7.5, 15, or 25 mg/kg/day paraquat ion, respectively. In the first study the NOAEL and LOAEL for maternal toxicity are 5 mg/kg/day and 10 mg/kg/day, respectively, based on reductions in body weight gain and death (range-finding study). The NOAEL and LOAEL for developmental toxicity are 5 mg/kg/day and 10 mg/kg/day, respectively based on an increased number of litters and fetuses with partial ossification of the 4th sternebra at 10 mg/kg/day HDT. Both the maternal and developmental NOAELs are at 15 mg/kg/day in the second study. The maternal LOAEL of 25 mg paraquat cation/kg/day is based on death, decreases in body weight and body weight gain, and other clinical signs. The developmental LOAEL of 25 mg/kg/day is based on decreases in mean fetal weights, retarded ossification and other skeletal effects. According to the registrant, Paraquat dichloride is not a developmental toxin and the developmental/maternal NOAEL should be based on the second study and is 15 mg/kg/day.

4. *Subchronic toxicity*. A 90-day feeding study was conducted in dogs fed doses of 0, 7, 20, 60, or 120 ppm with a NOAEL of 20 ppm based on lung effects such as alveolitis and alveolar collapse seen at the LOAEL of 60 ppm. In a 21-day inhalation toxicity study, rats were exposed to respirable aerosols of paraquat at doses of 0, 0.01, 0.1, 0.5, or 1.0 µg/L with a NOAEL of 0.01 µg/L and a LOAEL of 0.10 µg/L based on histopathological changes to the epithelium of the larynx and nasal discharge.

5. *Chronic toxicity*. A 12-month feeding study was conducted in dogs fed dose levels of 0, 15, 30, or 50 ppm, expressed as paraquat cation. These levels corresponded to 0, 0.45, 0.93, or 1.51 mg of paraquat cation/kg/day, respectively, in male dogs or 0, 0.48, 1.00, or 1.58 mg of paraquat cation/kg/day, respectively for female dogs. There was a dose-related increase in the severity and extent of chronic pneumonitis in the mid-dose and high-dose male and female dogs. This effect was also noted in the low-dose male group, but was minimal when compared with the male controls. The systemic NOAEL is 15 ppm (0.45 mg/kg/day for males and 0.48 mg/kg/day for females, expressed as parquet cation). The systemic LOAEL is 30 ppm (0.93 mg/kg/day for males and 1.00 mg/kg/day for females, expressed as paraquat cation).

In a 2-year chronic feeding/carcinogenicity study, rats were fed doses of paraquat dichloride at 0, 25, 75, or 150 ppm which correspond to 0, 1.25, 3.75, or 7.5 mg of paraquat cation/kg/day. Paraquat enhanced the development of ocular lesions in all of the treated groups. The predominant lesions detected opthalmoscopically were lenticular opacities and cataracts. At test week 103, dose-related statistically significant ($P<0.001$) increases in the incidence of ocular lesions were observed only in the mid-dose and high-dose male and female groups. Based on these findings, the NOAEL (approximate) and the LOAEL for systemic toxicity, for both sexes, are 25 ppm (1.25 mg/kg/day) and 75 ppm (3.75 mg/kg/day), respectively.

In another 2-year chronic feeding/carcinogenicity study, rats were dosed at 0, 6, 30, 100, or 300 ppm, expressed as paraquat dichloride (nominal concentrations), equivalent to 0, 0.25, 1.26, 4.15, or 12.25 mg/kg/day, respectively (males) and 0, 0.30, 1.5, 5.12, or 15.29 mg/kg/day respectively (females), expressed as paraquat dichloride. The incidence of ocular changes were low and not caused by paraquat in this study. The systemic NOAEL is 100 ppm of paraquat dichloride (4.15 and 5.12 mg/kg/day, for males and females, respectively); or 3.0 mg/kg/day (males) and 3.7 mg/kg/day (females), expressed as paraquat cation. The systemic LOAEL is 300 ppm of paraquat dichloride (12.25 and 15.29 mg/kg/day, for males and females, respectively); or 9.0 mg/kg/day (males) and 11.2 mg/kg/day (females), expressed as paraquat cation.

A chronic feeding/carcinogenicity study was conducted in rats fed dose levels of 0, 25, 75, or 150 ppm, expressed as paraquat cation (nominal concentrations). These doses corresponded to 0, 1.25, 3.75, or 7.5 mg paraquat cation/kg/day, respectively. There was uncertain evidence of carcinogenicity (squamous cell carcinomas in the head region; ears, nasal cavity, oral cavity and skin) in males at 7.5 mg/kg/day HDT with a systemic NOAEL of 1.25 mg/kg/day. Upon submission of additional data to EPA, the incidence of pulmonary adenomas and carcinomas was well within historical ranges and it was determined that paraquat was not carcinogenic in the lungs and head region of the rat.

In another chronic feeding/carcinogenicity study, rats were fed dose levels of 0, 6, 30, 100, or 300 ppm,

expressed as paraquat dichloride. There were no carcinogenic findings in this study at the HDT. In a 2-year chronic feeding/concinogenicity study, SPF Swiss derived mice were fed paraquat dichloride at dose levels of 0, 12.5, 37.5, or 100/125 ppm, expressed as paraquat cation. These rates correspond to 0, 1.87, 5.62, and 15 mg/kg/day as cation. Because no toxic signs appeared after 35 weeks of dosing, the 100 ppm level was increased to 125 ppm at week 36. There were no carcinogenic effects observed in this study. The systemic NOAEL for both sexes is 12.5 ppm (1.87 mg/kg/day) and the systemic LOAEL is 37.5 ppm (5.6 mg/kg/day), each expressed as paraquat cation based on renal tubular degeneration in males and weight loss and decreased food intake in females.

Paraquat is classified Category E for carcinogenicity (no evidence of carcinogenicity in animal studies).

6. *Animal metabolism*. The qualitative nature of the residue in animals is adequately understood based on the combined studies conducted with ruminants (goats and cows), swine, and poultry. The residue of concern in eggs, milk, and poultry, and livestock tissues is the parent, paraquat.

C. Aggregate Exposure

In examining aggregate exposure, FQPA directs EPA to take into account available information concerning exposures from the pesticide residue in food and all other exposures for which there is reliable information. These other sources of exposure include drinking water, and non-occupational exposures, e.g., to pesticides used in and around the home. For estimating acute and chronic risks the Agency considers aggregate exposures from the diet and from drinking water. Exposures from uses in and around the home that may be short term, intermediate, or other durations may also be aggregated as appropriate for specific chemicals.

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure under the proposed tolerance, Zeneca has estimated aggregate exposure based on the tolerance levels of 0.05 ppm, 0.3 ppm, 0.05 ppm, and 0.05 ppm in or on globe artichokes, dry peas, persimmons, endive and from all other established tolerances. Percent crop treated was also incorporated into the assessment to derive an upper bound anticipated residue contribution (ARC). The registrant has concluded that there are no acute endpoints of concern for paraquat, and an acute aggregate assessment is not required. The chronic population adjusted dose (cPAD) for chronic dietary assessments is 0.0045 mg/kg/day, based on a NOAEL of 0.45 mg/kg/day from a 1-year dog study and the addition of a standard uncertainty factor of 100.

i. *Food.*— a. *Chronic dietary assessment*. A chronic dietary exposure analysis was performed using current and reassessed tolerance level residues, contributions from the proposed tolerance for use on globe artichoke, dry peas, persimmons, endive, and current percent crop treated information to estimate the ARC for the general population and 22 subgroups. The tolerance in globe artichoke resulted in an ARC of 0.0000001 mg/kg/day (0.002% of the cPAD) for the general population. The resulting ARC for the general U.S. population from all established uses is 0.000367 mg/kg/day (8.2% of the cPAD). For children ages 1 to 6, the most highly exposed subgroup, the resulting ARC is 0.001077 mg/kg/day (23.9% of the cPAD).

b. *Acute dietary assessment*. The registrant has determined that current data on paraquat shows no acute dietary endpoint of concern. Therefore, an acute dietary risk assessment was not conducted for paraquat.

ii. *Drinking water*. The registration eligibility document (RED) for paraquat has stated the following:

Paraquat is not expected to be a contaminant of groundwater. Paraquat dichloride binds strongly to soil clay particles and it did not leach from the surface in terrestrial field dissipation studies. There were, however, detections of paraquat in drinking water wells from two states cited in the pesticides in groundwater data base (1991). These detections are not considered to be representative of normal paraquat use. Therefore, paraquat is not expected to be a groundwater contaminant or concern based on normal use patterns.

Due to its persistent nature, paraquat could potentially be found in surface water systems associated with soil particles carried by erosion, however, paraquat is immobile in most soils, and at very high application rates (50–1000X), there was no desorption of paraquat from soils. Based on paraquat's normal use patterns and unique environmental fate characteristics, exposures to paraquat in drinking water are not expected to be obtained from surface water sources. Therefore, the only exposures considered in aggregate risk assessment for paraquat is chronic dietary.

2. *Non-dietary exposure*. Paraquat dichloride has no residential or other non-occupational uses that might result in non-occupational, non-dietary exposure for the general population. Paraquat products are restricted use, for use by certified applicators only, which means the general public cannot buy or use paraquat products.

D. Cumulative Effects

In assessing the potential risk from cumulative effects of paraquat and other chemical substances, the Agency has considered structural similarities that exist between paraquat and other bipyridylium compounds such as diquat dibromide. Examination of the toxicology data bases of paraquat and diquat dibromide, indicates that the two compounds have clearly different target organs. Based on available data, the registrant does not believe that the toxic effects produced by paraquat would be cumulative with those of diquat dibromide.

E. Safety Determination

1. *U.S. population*. Based on the paraquat RED, the only exposure route of concern for paraquat is chronic dietary. Using the conservation assumptions presented earlier, EPA has established a cPAD of 0.0045 mg/kg/day. This was based on the NOAEL for the 1-year dog study of 0.45 mg/kg/day and employed a 100-fold uncertainty factor. Results of this aggregate exposure assessment, which includes EPA's reassessment of tolerances for existing crops and the tolerance for use on globe artichokes, dry peas, persimmons, and endive utilize 8.2% of the cPAD. Generally, exposures below 100% of the cPAD are of no concern because it represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, the registrant has concluded that there is reasonable certainty that no harm will result from aggregate exposures to paraquat residues.

2. *Infants and children*. Zeneca has determined that the established tolerances for paraquat, with amendments and changes as specified in this notice, meet the safety standards under the FQPA amendments to section 408(b)(2)(C) for infants and children. The safety determination for infants and children considers the factors noted above for the general population, but also takes into account the possibility of increased dietary exposure due to specific consumption patterns of infants and children, as well as the possibility of increased susceptibility to the toxic effects of paraquat residues in this population subgroup.

In determining whether or not infants and children are particularly susceptible to toxic effects from paraquat residues, Zeneca considered the completeness of the data base for developmental and

reproductive effects, the nature and severity of the effects observed, and other information.

Based on the current data requirements, paraquat has a complete data base for developmental and reproductive toxicity. In the developmental studies, effects were seen (delayed ossification in the forelimb and hindlimb digits) in the fetuses only at the same or higher dose levels than effects in the mother. In the reproduction study, no effects on reproductive performance were seen. Also because the NOAELs from the developmental and reproduction studies were equal to or greater than the NOAEL used for establishing the cPAD, the registrant concluded that it is unlikely that there is additional risk concern for immature or developing organisms. Finally, there is no epidemiological information suggesting special sensitivity of infants and children to paraquat. Therefore, the registrant found that an additional safety factor for infants and children is not warranted for paraquat.

Zeneca estimates that paraquat residues in the diet of non-nursing infants (less than 1 year) account for 17.6% of the cPAD and 23.9% of the cPAD for children aged 1 to 6 years. Further, residues in drinking water are not expected. Therefore, Zeneca has determined that there is reasonable certainty that dietary exposure to paraquat will not cause harm to infants and children.

F. International Tolerances

There is no approved CODEX maximum residue level (MRL) established for residues of paraquat on endive.

II. Interregional Research Project Number 4

0E6090

EPA has received a pesticide petition 0E6090 from the Interregional Research Project Number 4 (IR–4); Rutgers University, New Brunswick, NJ, 08903-0231 proposing, pursuant to section 408(d) of the Federal Food , Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of Fosetyl-Al in or on the raw agricultural commodity (RAC) cranberries at 0.5 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition. This notice includes a summary of the petition prepared by Aventis CropScience the registrant, P.O. Box 12014, 2 T.W. Alexander Drive, Research Triangle Park, NC, 27709.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of fosetyl-Al in plants is adequately understood. Adequate data on the nature of the residues in plants, including identification of major metabolites and degradates of fosetyl-Al, are available. Radiolabeled studies on the uptake, translocation and metabolism in plants show that the chemical proceeds through hydrolytic cleavage of the ethyl ester. The major residues are fosetyl-Al, phosphorus acid, and ethanol. The tolerances are established for the parent only, that is fosetyl-Al.

2. *Analytical method*. Adequate methods are available for enforcement purposes. There are two analytical methods acceptable for determining residues of fosetyl-Al in plants: a gas chromatography method is available for enforcement of tolerance in pineapple and is listed as method I in pesticide analytical manual (PAM), Vol. II; a gas chromatography/phosphorus specific flame photometric detector (FPD-P) method (Aventis CropScience method no. 163) for citrus has undergone a successful method tryout on oranges and has been sent to the Food and Drug Adminstration (FDA) for inclusion in PAM as method II.

3. *Magnitude of residues*. Field trials were conducted in EPA regions 1 (MA), 2 (NJ), 5 (WI), and 12 (OR). All field trial sites consisted of 1 untreated control plot and 1 treated plot. Each treated plot received four foliar spray applications of fosetyl-Al at a rate of 4.0 lb active ingredient per acre (ai/A) # 5% each, for a total of approximately 16 lb ai/A. The first application was made at approximately 93 days prior to harvest and subsequent applications were made at approximately 30-day intervals. Samples were collected at 3 or 4 days after the last application in all trials. Fosetyl-Al residues in treated samples ranged from <0.05 ppm to 0.35 ppm. Data from this study support the proposed tolerance of 0.5 ppm.

B. Toxicological Profile

1. *Acute toxicity*. A complete battery of acute toxicity studies for fosetyl-Al technical has been conducted. The lethal dose LD_{50} from the acute oral rat is 5.4 g/kg and the LD_{50} from an acute dermal rabbit study is >2 g/kg. The LC_{50} for a rat inhalation study is >1.73 mg/L. The acute oral rat and primary dermal irritation studies indicate category IV toxicity. A guinea pig dermal sensitization study shows fosetyl-Al is not a skin sensitizer. The primary eye irritation study in rabbits shows fosetyl-Al to be an eye irritant with Category I toxicity.

2. *Genotoxicity*. Fosetyl-Al is neither mutagenic nor genotoxic. The genetic toxicity potential of fosetyl-Al was assessed in several assays. Eight mutagenicity tests performed with fosetyl-Al were negative. The tests included two Ames assays with *S. typhimurium*, two phase induction assays using *E. coli*, two micronucleus studies in mice, one DNA repair assay using *E. coli* and one mutation assay in *Saccharomyces cereviseae*.

3. *Reproductive and developmental toxicity*. Fosetyl-Al is not a reproductive toxicant and shows no evidence of estrogenic or androgenic related effects.

i. In a 3-generation reproduction study, fosetyl-Al was administered to rats at dietary levels of 0, 6,000, 12,000, or 24,000 ppm. No adverse effects on reproductive performance or pup survival were observed in any dose group. The lowest observed adverse effect level (LOAEL) was established at 12,000 ppm based on effects on animal weights and urinary tract changes. The no observed adverse effect level (NOAEL) for all effects was 6,000 ppm.

ii. A developmental study in rats dosed via oral gavage at 500, 1,000 or, 4,000 mg/kg/day showed a developmental NOAEL of 1,000 mg/kg. At 4,000 mg/kg, there was maternal toxicity, as evidenced by effects on animal weights, maternal deaths, increased resorptions and delayed fetal ossification.

iii. A rabbit developmental study showed no toxic effects at oral doses up to 500 mg/kg. Effects of fosetyl-Al on fetal development were observed only in the rat at a dose producing severe maternal toxicity. In the absence of maternal toxicity, no adverse effects on fetal development were observed, i.e. at 1,000 mg/kg/day in rats or at 500 mg/kg/day in rabbits.

4. *Subchronic toxicity*. In subchronic studies, no significant toxicity was observed even at doses exceeding the limit of 1,000 mg/kg/day.

i. A 21-day dermal study in rabbits showed mild to moderate skin irritation and a NOAEL of 1.5 g/kg/day.

ii. A 90-day feeding study in rats showed a NOAEL of >5,000 ppm; the LOAEL was 25,000 ppm with extramedullary hematopoiesis in the spleen.

iii. A 90-day dog feeding study showed a NOAEL of 10,000 ppm and a

LOAEL at 50,000 ppm, at which the test animals had a lower serum potassium level than untreated animal.

5. *Chronic toxicity*. Chronic toxicity studies have been conducted in dogs and rats:

i. *Dog*. Fosetyl-Al was fed to dogs for 2-years at concentrations of 0, 10,000, 20,000, and 40,000 ppm. The NOAEL was 10,000 ppm, equivalent to 250 mg/kg/day. The LOAEL was 20,000 ppm based on a slight degenerative effect on the testes. These testicular changes, as well as a few scattered clinical changes, were seen in the high dose dogs. No effects were observed in the urinary tract.

ii. *Rat*. Fosetyl-Al was administered via admixture in the diet to CD rats at target levels of 0, 2,000, 8,000, and 30,000/40,000 ppm for approximately 2-years. Based on these levels, respective doses were 100, 400 and 2,000/1,500 mg/kg/day. After 2-weeks at 40,000 ppm, this dietary level was reduced to 30,000 ppm due to the occurrence of red coloration of the urine and a decrease in body weight gain. Although these findings were no longer apparent after week 2, analytical verification of dietary levels revealed that the highest dietary level ranged from approximately 38,000 to 61,000 ppm during the first 32-weeks of the study. No significant differences in body weight or food consumption were noted at 2,000 or 8,000 ppm. No biologically significant differences were observed in ophthalmoscopy, hematology, clinical chemistry, or urinalysis for treated and control animals. Calculi in the urinary bladder were observed for several male and female rats in the high dose group. Non-neoplastic findings consisted of epithelial hyperplasia and inflammation in the urinary bladders of males at 30,000/40,000 ppm. Increased incidences of hydronephrosis, inflammation, and epithelial hyperplasia in the kidney were also observed in males from the high dose group. Females from the same group exhibited increased incidences of epithelial hyperplasia in the urinary bladder and hydronephrosis in the kidney. The NOAEL in the chronic rat study was 8,000 ppm (400 mg/kg/day).

The lowest NOAEL for chronic effects of fosetyl-Al is 10,000 ppm (250 mg/kg/day) based on the dog study. This NOAEL is based on minor changes at 20,000 ppm. In the rat, calculi in the urinary bladder and related histopathological changes in the bladder and kidneys of males and females were observed at 30,000/40,000 ppm.

6. *Carcinogenicity*. Long-term feeding studies were conducted with technical grade fosetyl-Al in mice and rats and with monosodium phosphite, the primary urinary metabolite of fosetyl-Al, in rats. These studies, in addition to a mechanistic study in rats, are described below:

i. *Rat*. Fosetyl-Al was administered via admixture in the diet to CD rats at target levels of 0, 2,000, 8,000, and 30,000/40,000 ppm for approximately 2-years. After 2-weeks at 40,000 ppm, this dietary level was reduced to 30,000 ppm due to the occurrence of red coloration of the urine and a decrease in body weight gain. Although these findings were no longer apparent after Week 2, analytical verification of dietary levels revealed that the highest dietary level ranged from approximately 38,000 to 61,000 ppm during the first 32-weeks of the study. Calculi in the urinary bladder were observed for several male and female rats at 30,000/40,000 ppm. Microscopic examination revealed transitional cell carcinomas and papillomas in the urinary bladders of high dose males. In addition, a statistically significant increase in adrenal pheochromocytomas (benign and malignant combined) was observed in males at 8,000 and 30,000/40,000 ppm. The adrenal slides were independently reread by two consulting pathologists who found no significant dose-related increases in the incidence of pheochromocytomas or hyperplasia. The NOAEL for fosetyl-Al in the chronic rat study was 8,000 ppm. A subsequent mechanistic study in rats conducted with dietary levels of 8,000, 30,000 and 50,000 ppm demonstrated that the massive doses of 30,000 and 50,000 ppm fosetyl-Al alter calcium/phosphorous homeostasis resulting in severe acute renal injury, similar to that observed in the chronic rat study, and the formation of calculi in kidneys, ureters, and bladder. Under conditions of chronic exposure, these effects could lead to the formation of bladder tumors as seen in the chronic rat study. At 8,000 ppm, no evidence of renal injury was observed, a result consistent with the absence of bladder tumors. Thus, the bladder tumors induced by fosetyl-Al were the result of acute renal injury followed by a chronic toxic reaction rather than a true carcinogenic effect.

A carcinogenicity study in rats was conducted with monosodium phosphite administered via dietary mixture at levels of 2,000, 8,000, and 32,000 ppm. No evidence of carcinogenicity was observed in this study.

ii. *Mouse*. A 2-year feeding/carcinogenicity study was conducted in mice fed diets containing fosetyl-Al at 0, 2,500, 10,000, or 20,000/30,000 ppm. The 20,000 ppm dose was increased to 30,000 ppm during week 19 of the study. The NOAEL for all effects was 20,000/30,000 ppm (3,000/4,500 mg/kg/day). There were no carcinogenic effects observed under the conditions of this study.

7. *Animal metabolism*. Rat metabolism studies showed that most of the radiolabel rapidly appeared in exhaled carbon dioxide. There was also some radiolabel excreted in the urine as phosphite, along with a smaller amount as the unchanged parent compound. It appears that fosetyl-Al is essentially completely absorbed after ingestion and extensively hydrolyzed to carbon dioxide which is exhaled. The phosphite is excreted in the urine without further oxidation to phosphate. Aluminum does not appear to be absorbed to a significant extent from the gastrointestinal trac.

8. *Metabolite toxicology*. There are no metabolites of toxicological concern. The tolerances are established for the parent only, that is fosetyl-Al.

9. *Endocrine disruption*. No evidence of estrogenic or androgenic effects were noted in any study with fosetyl-Al. No adverse effects on mating or fertility indices and gestation, live birth, or weaning indices were noted in a 3-generation rat reproduction study at doses well above EPA's limit of 1,000 mg/kg/day. Therefore, Aventis CropScience concludes that fosetyl-Al does not have any effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. EPA has established the chronic reference dose (RfD) for fosetyl-Al at 2.5 mg/kg/day. This RfD is based on a NOAEL of 250 mg/kg/day from a 2-year feeding study in dogs and the use of a 100 fold safety factor to account for inter-species and intra-species differences. No appropriate endpoint attributable to a single dose exposure was identified in oral toxicity studies. Therefore, an acute RfD was not established and there is no expectation of acute risk. Since no dermal or systemic toxicity was seen at the limit dose following repeated dermal applications in the 21-day toxicity study using rats, no endpoint value was calculated for short-and intermediate-term exposure and risk. The Agency has concluded that fosetyl-Al is unlikely to pose a carcinogenic hazard to humans. Therefore, a cancer exposure and risk assessment is not appropriate.

i. *Food*. For all currently registered uses of fosetyl-Al, chronic food exposure for various subgroups of the U.S. population was estimated by EPA through the use of the dietary exposure evaluation model (DEEM) software. The DEEM analysis evaluated the individual

food consumption as reported by respondents in the U. S. Department of Agricultural (USDA) 1989–1991 nationwide continuing surveys of food intake by individuals. As the risk estimate was low for even the most highly exposed subpopulation, no anticipated residues were used. In the surveys, 100% crop treated and tolerance level residues were assumed for all crops. The calculated potential exposure for the U.S. population is 0.077 mg/kg/day resulting in utilization of 3% of the chronic population adjusted dose (cPAD). Potential exposure for the most highly exposed group, children (1–6 years), is 0.157 mg/kg/day and corresponds to 6% of the chronic cPAD. Aventis CropScience anticipates that the incremental exposure resulting from the proposed use on cranberries will be minimal and that dietary exposure for the proposed tolerance in addition to all existing tolerances for fosetyl-Al will be well below the Agency's level of concern.

ii. *Drinking water*. There is no established maximum contaminant level (MCL) or health advisory level for fosetyl-Al. The potential for ground water and/or surface water contamination by fosetyl-Al and its degradates is expected to be very low, in most cases, due to the rapid degradation of the compound in soil to non-toxic degradates under both aerobic and anaerobic conditions. Under aerobic laboratory conditions, the half-life of fosetyl-Al is between 1 and 1.5 hours in loamy sand, silt loam and clay loam and 20 minutes in sandy loam soil. The degradation proceeds through the hydrolysis of the ethyl ester bond, resulting in the formation of phosphorous acid and ethanol. The ethanol is further degraded into carbon dioxide. Based on the short half-life of fosetyl-Al and the known fate of phosphates under anaerobic conditions, EPA determined that an anaerobic soil metabolism study was not necessary. An anaerobic aquatic soil metabolism study was conducted. When anaerobic conditions were established by flooding soil, the half-life was 40 hours with silty clay loam and 14 hours with sandy loam soil. Aventis CropScience expects that potential fosetyl-Al residues in drinking water are not a significant contribution to aggregate exposure.

2. *Non-dietary exposure*. Fosetyl-Al is currently registered for residential use on turf and ornamental plants. Chronic exposure is not expected for residential uses. There is also no expectation of acute risk. No appropriate endpoint attributable to a single dose exposure was identified in oral toxicity studies and consequently, an acute RfD cannot be calculated. No endpoint value is calculable for short-term and intermediate-term exposure and a risk analysis cannot be performed since no dermal or systemic toxicity was seen at the limit dose following repeated dermal applications in the 21-day toxicity study using rats. The Agency has previously concluded that fosetyl-Al is unlikely to pose a carcinogenic hazard to humans. Therefore, a cancer exposure and risk assessment is not appropriate. Thus, Aventis CropScience concludes that the ornamental and turf uses do not add significantly to the aggregate exposure for fosetyl-Al.

D. Cumulative Effects

Effects associated with fosetyl-Al are unlikely to be cumulative with any other compound. The formation of calculi and bladder tumors in rats are the only significant toxicological effects observed with fosetyl-Al. These effects were observed in the rat only at a dose which, not only exceeds estimated human exposure by several orders of magnitude, but is in excess of EPA's dose limit for carcinogenicity studies. Therefore, an aggregate assessment based on common mechanisms of toxicity is not appropriate as exposure to humans will be well below the levels producing calculi and bladder tumors in rats. Further, considering the rapid elimination of fosetyl-Al in the rat metabolism study, any effects associated with fosetyl-Al are unlikely to be cumulative with any other compound. Based on these reasons, only the potential risks of fosetyl-Al are considered in the exposure assessment.

E. Safety Determination

1. *U.S. population*. Chronic risk estimates associated with exposure to fosetyl-Al in food and water are expected to be well below the Agency's level of concern. The DEEM chronic exposure analysis previously performed by the Agency for all currently registered food uses showed that the U.S. general population, 3% of the cPAD is occupied by dietary (food) exposure. For the most highly exposed subgroup, children 1–6 years old, 6% of the cPAD is occupied by dietary (food) exposure. The contribution of fosetyl-Al residues in surface and ground water to chronic aggregate exposure is expected to be minimal. The incremental exposure resulting from the proposed use on cranberries is also expected to be negligible. Therefore, Aventis CropScience concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fosetyl-Al residues.

2. *Infants and children*. No indication of increased susceptibility of rat or rabbit fetuses to *in utero* and/or postnatal exposure was noted in the developmental and reproductive toxicity studies. The Agency has previously determined that no additional safety factor to protect infants and children is necessary for this product.

Using the conservative assumptions described in the exposure section above (unit II.C.), aggregate exposure to fosetyl-Al from currently registered food uses will utilize up to 6% of the cPAD for infants and children. The incremental exposure to fosetyl-Al resulting from the proposed use on cranberries is expected to be minimal and even when considered in addition to the potential for exposure to residues in drinking water and from non-dietary, non-occupational exposure, the aggregate exposure to fosetyl-Al is not expected to exceed 100% of the cPAD. Aventis CropScience concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to fosetyl-Al residues.

F. International Tolerances

There are presently no Codex alimentarius commission maximum residue levels established for residues of fosetyl-Al.

III. Interregional Research Project Number 4

8E5012

EPA has received a pesticide petition 8E5012 from the Interregional Research Project Number 4 (IR–4), New Jersey Agricultural Experiment Station, P.O. Box 231 Rutgers University, New Brunswick, NJ 08903 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of cyprodinil in or on the raw agricultural commodities dry bulb onion, green onion, and strawberries at 0.6, 4.0, and 5.0 parts per million (ppm).

A. Residue Chemistry

1. *Plant metabolism*. Novartis believes the metabolism of cyprodinil has been well characterized in plants. The metabolism profile supports the use of an analytical enforcement method that accounts for only parent cyprodinil.

2. *Analytical method*. Analytical method AG–631A is a practical analytical method involving extraction, filtration, and solid phase cleanup of samples with analysis by high performance liquid chromotography

(HPLC) and ultra-violet ray (UV). The limits of quantitation (LOQ) for various commodities are as follows: fruit, grain, juice-0.02 ppm; forage, fodder, straw-0.05 ppm; and grapes-0.01 ppm.

3. *Magnitude of residues*. This petition is supported by field trials conducted on representative members of the bulb vegetable crop grouping and strawberries. All samples were analyzed by residue method AG–631A to determine the residues of cyprodinil. In bulb vegetables, the maximum residue found on representative commodities were 3.9 ppm and 2.7 ppm, for green onion and bulb onion, respectively. The maximum residue found in strawberries was 3.3 ppm. A tolerance of 5.0 ppm for strawberries has been proposed.

B. Toxicological Profile

Cyprodinil appears to pose relatively little human toxicity risk due to low use rate, low risk to groundwater, low dietary risk and low worker exposure. The risk from acute dietary exposure to cyprodinil is considered to be very low. The mammalian toxicity studies that have been conducted to support the tolerances of cyprodinil are listed below.

1. *Acute toxicity*. The following are results from the acute toxicity tests conducted on the technical material:

i. A rat acute oral study for cyprodinil with a LD_{50} of 2,796 milligrams/kilograms(mg/kg).

ii. A rat acute dermal study for cyprodinil with a $LD_{50} > 2,000$ mg/kg.

iii. A rat inhalation study for cyprodinil with a $LC_{50} > 1.2$ mg/liter air.

iv. A primary eye irritation study in rabbits showing cyprodinil as minimally irritating.

v. A primary dermal irritation study in rabbits showing cyprodinil as slightly irritating.

vi. A skin sensitization study in guinea pigs showing cyprodinil as a weak sensitizer.

2. *Genotoxicity*. The following are results from the genotoxicity test:

i. *In vitro gene mutation test*. Ames assay-negative; chinese hamster V79 cell test-negative; rat hepatocyte DNA repair test-negative.

ii. *In vitro chromosome test*. Chinese hamster ovary cell cytogenetic test-negative.

iii. *In vivo mutagenicity test*. Mouse bone marrow test-negative.

3. *Reproductive and developmental toxicity*. Cyprodinil is not a reproductive or developmental hazard, as is demonstrated by the results of the following studies:

i. *Rat oral developmental*. An oral developmental study in the rat with a maternal no observed adverse effect level (NOAEL) of 200 mg/kg based on reductions in body weight gain and food consumption and a fetal NOAEL of 200 mg/kg based on decreased pup weight and delayed skeletal growth at 1,000 mg/kg.

ii. *Rabbit oral developmental study*. An oral developmental study in the rabbit with a maternal NOAEL of 150 mg/kg based on reduction in body weight gain and a fetal NOAEL of 400 mg/kg based on the absence of any fetal effects.

iii. *Rat 2-generation reproduction study*. A 2-generation reproduction study in the rat with a systemic NOAEL of 100 ppm and a fetal NOAEL of 1,000 ppm (100 mg/kg). A slight decrease in pup weight at birth and subsequent body weight gain during the lactation phase was observed only at the maternally toxic dose of 4,000 ppm without any effects on reproduction and fertility.

4. *Subchronic toxicity*. These tests are summarized below:

i. A 28-day dermal study in the rat with a NOAEL of 5 mg/kg based on clinical signs.

ii. A 90-day feeding study in the dog with a NOAEL of 1,500 ppm (37.5 mg/kg) based on reduced food intake and body weight.

iii. A 90-day feeding study in the mouse with a NOAEL of 500 ppm (75 mg/kg) based on liver histologic changes.

iv. A 90-day feeding study in the rat with a NOAEL of 50 ppm (5 mg/kg) based on hematologic and histologic findings.

5. *Chronic toxicity*. The reference dose (RfD) for cyprodinil is 0.0375 mg/kg/day. This value is based on the systemic NOAEL of 3.75 mg/kg/day in the rat chronic feeding study with a 100-fold safety factor to account for interspecies extrapolation and intraspecies variability.

i. A 12-month feeding study in the dog with a NOAEL of 2,500 ppm (62.5 mg/kg) based on liver histologic changes.

ii. An 18-month carcinogenicity feeding study in the mouse with a NOAEL of 2,000 ppm (300 mg/kg). The maximum tolerated dose (MTD) was 5,000 ppm based on reduction in body weight gain and no evidence of carcinogenicity was seen.

iii. A 24-month chronic feeding/carcinogenicity study in the rat with a NOAEL of 75 ppm (3.75 mg/kg) based on hematologic and histologic findings. The MTD was 2,000 ppm based on liver histopathology and no evidence of carcinogenicity was seen.

6. *Animal metabolism*. Ruminant metabolism shows extensive degradation following a pathway that is similar to plants. Extrapolating from goat studies, none of the metabolites, including parent compound, will be near the normal minimum range for detection by analytical methods (0.01 to 0.05 ppm). Therefore, parent residues will be proposed as an adequate marker for total residues of cyprodinil in animals. The analysis also demonstrates that livestock tolerances are not required in conjunction with this petition.

7. *Endocrine disruption*. Cyprodinil does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. Developmental toxicity studies in rats and rabbits and a reproduction study in rats gave no indication that cyprodinil might have any effects on endocrine function related to development and reproduction. The chronic studies also showed no evidence of a long-term effect related to the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. For the purposes of assessing the potential dietary exposure under the proposed tolerances, Novartis has estimated aggregate exposure from the previously established tolerances for the raw agricultural commodities: almond nutmeat at 0.02 (ppm), almond hulls at 0.05 ppm, grapes at 2.0 ppm, raisins at 3.0 ppm, pome fruit crops at 0.1 ppm, wet apple pomace at 0.15 ppm, and stone fruit crops at 2.0 ppm; and the requested tolerances of strawberries at 5.0 ppm, dry bulb onion at 0.6 ppm, and green onion at 4.0 ppm. The tier 1 chronic cyprodinil assessment displayed below used tolerance values listed in 40 CFR 180.532 for all commodities; 100% market share was assumed for all crops. Results of the cyprodinil assessment are displayed below as a percentage of the chronic RfD.

Population	Chronic RfD
U.S. Population	11.5%
All infants (< 1 year)	24.6%
Nursing infants (< 1 year).	10.7%
Non-nursing infants (< 1 year).	28.6%
Children (1–6 years)	31.1%
Children (7–12 years).	13.5%

ii. *Drinking water*. The potential for exposure to cyprodinil through drinking water (surface or ground water) is slight due to the minimal level of this chemical anticipated to reach these

bodies of water. This expectation is based on the rapid degradation of cyprodinil and the recommended low use rates that will further restrict the amount of chemical available for leaching or run-off.

2. *Non-dietary exposure.* Novartis believes that the potential for non-occupational exposure to the general public is unlikely except for potential residues in food crops discussed above. The proposed uses for cyprodinil are for agricultural crops and the product is not used residentially in or around the home.

D. Cumulative Effects

Consideration of a common mechanism of toxicity is not appropriate at this time since there is no information to indicate that toxic effects produced by cyprodinil would be cumulative with those of any other chemicals. Consequently, only the potential exposure to cyprodinil is considered in this risk assessment.

E. Safety Determination

1. *U.S. population.* For the U.S. population (48 contiguous states) chronic exposure was 11% of the RfD. EPA usually 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. Novartis concludes that there is a reasonable certainty that no harm will result from aggregate exposure to cyprodinil.

2. *Infants and children.* Maximum expected chronic exposure to cyprodinil in the diets of the most sensitive sub-populations, for non-nursing infants (<1–year old) and 31.1% of the RfD for childern (1–6 years old) was calculated to be 28.6% of the RfD.

F. International Tolerances

Codex maximum residue levels (MRLs) have not been established for residues.

[FR Doc. 00–15161 Filed 6–20–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–942; FRL–6557–3]

Notice of Filing a Pesticide Petition to Establish a Tolerance for Certain Pesticide Chemicals in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket control number PF–942, must be received on or before July 21, 2000.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the ''SUPPLEMENTARY INFORMATION.'' To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–942 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Richard J. Gebken, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–6701; e-mail address: gebken.richard@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under ''FOR FURTHER INFORMATION CONTACT.''

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this action under docket control number PF–942. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–942 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services