regarding the applicability of this action to a partcular entity, consult the person listed in the ''FOR FURTHER INFORMATION CONTACT'' section. The Congressional Review Act, 5 U.S.C. 801 et seq., as added by the Small Business Regulatory Enforcement Fairness Act of 1996, does not apply because this action is not a rule, for purposes of 5 U.S.C. 804(3).

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

1. *Electronically*. You may obtain copies of this document and certain other available support documents from the EPA Internet Home Page at http://www.epa.gov/. You may access this document by selecting ''Laws and Regulations'' on EPA's Home Page and then looking 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/. To access information about the risk assessment for methyl parathion, go to the Home Page for the Office of Pesticide Programs or go directly to: http:www/epa.gov/oppsrrd1/op/methyl—parathion.htm.

2. *In person.* The Agency has established an official record for this action under docket control number 66272A. 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.

II. Correction

FR Doc.99-27800, published in the **Federal Register** of October 27, 1999, at page 57877, is corrected by removing from the first column of page 57881, ''Unit III.B. Notification of Possession of Canceled Products,'' and the following text:

No later than November 1, 1999, and pursuant to section 6(g) of FIFRA, any producer or exporter, registrant, applicant for a registration, applicant or holder of an experimental use permit, commercial applicator, or any person who distributes or sells any pesticide, who after the publication of this Notice possesses any stocks of the pesticide products identified on Table 2 of this notice, shall notify EPA and appropriate State and local officials of: (1) Such possession; (2) the quantity of canceled methyl parathion pesticide product possessed; and (3) the place at which the canceled methyl parathion pesticide product is stored.

List of Subjects

Enviornmental protection, Pesticides and pest.

Dated: November 24, 1999.

Jack E. Housenger,

Acting Director, Special Review and Reregistration Division.

[FR Doc. 99–31296 Filed 12-2-99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–900; FRL–6392–6]

Notice of Filing Pesticide Petitions 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–900, must be received on or before January 3, 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'' section. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–900 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Shaja Brothers, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 308–3194; and 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	Examples of potentially affected entities
Industry	111	Crop production
	112	Animal production
	311	Food manufacturing
	32532	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 in the ''FOR FURTHER INFORMATION CONTACT'' section.

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–900. 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–900 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, 401 M St., SW., 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–900. 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 in the ''FOR FURTHER INFORMATION CONTACT'' section.

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 supports 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: November 29, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

1. Interregional Research Project Number 4

1E4019, 7E4857, and 9E6009

EPA has received pesticide petitions (1E4019, 7E4857, and 9E6009) from the Interregional Research Project Number 4 (IR-4) New Jersey Agricultural Experiment Station, Rutgers University, New Brunswick, New Jersey 08903 proposing, under 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 tolerances for residues of the herbicide paraquat (1,1-dimethyl-4,4'-bypyridinium) derived from the application of the dichloride salt (calculated as the cation) in or on the raw agricultural commodities (RAC) globe artichoke, dry peas, and persimmon at 0.05, 0.3, and 0.05 parts per million (ppm), respectively. EPA has determined that the petitions contain 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 petitions. Additional data may be needed before EPA rules on the petitions. This notice includes a summary of the petitions prepared by Zeneca Ag Products, the registrant, 1800 Concord Pike, P.O. Box 15458, Wilmington, Delaware 19850-5458.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residue 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.

3. *Magnitude of residues*. Magnitude of residue data were collected from three sites in the major globe artichoke producing region of the United States. No residues exceed the proposed tolerance of 0.05 ppm, when globe artichokes are treated with 3.0 to 3.6 lb active ingredient/acre (ai/acre) of paraquat applied as three applications directed between the rows at approximately 7–day intervals and the last application 1–day prior to harvest. Residue data have been obtained from Washington and Idaho which represent 91% of the dry pea production in the United States. Mature dry peas were treated once with paraquat at either 0.5 or 1.0 lb ai/acre of paraquat 7 days prior to harvest. The highest residue recovered in the dry pea was 0.25 ppm. The other treated samples all had residues of ≤ 0.2 ppm. IR-4 is requesting the establishment of a tolerance for persimmon based on the 0.05 ppm tolerance established on guava. Applications of paraquat in persimmon would be the same as those in the Gramoxone Extra label for use on guava, utilizing a directed, postemergence application.

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 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. *Genotoxicty*. 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 (UDS) 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 F_0, F_1, and F_2 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, the developmental/maternal NOAEL should be based on the second study and is 15 mg/kg/day. Paraquat dichloride is not a developmental toxin.

4. *Subchronic toxicity*. A 90–day feeding study 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. A 21–day inhalation toxicity study in 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*. In a 12–month feeding study 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 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, and 0.05 ppm in or on globe artichokes, dry peas, and persimmons 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—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, cotton, and persimmons and current percent crop treated information to estimate the ARC for the general population and 22 subgroups. The tolerance in globe artichoke resulted in a 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 1-6, the most highly exposed subgroup, the resulting ARC is 0.001077 mg/kg/day (23.9% of the cPAD).

ii. *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.

iii. *Drinking water* . The Registration Eligibility Document (RED) for paraquat has stated the following:

Paraquat is not expected to be a contaminant of ground water. 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 Ground Water Database* (1991). These detections are not considered to be representative of normal paraquat use. Therefore, paraquat is not expected to be a ground water 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 (50-1,000x), 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 cummulative 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, and persimmons 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 1-6 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 globe artichokes, dry peas, and persimmons.

2. Interregional Research Project Number 4

PP 9E6042

EPA has received a pesticide petition (9E6042) from the Interregional Research Project Number 4 (IR-4), Center for Minor Crop Pest Management, at the Technology Centre of New Jersey, 681 U.S. Highway #1, South, North Brunswick, NJ 08902-3390 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerances for residues of fenpropathrin, alpha-cyano-3-phenoxybenzyl 2,2,3,3-tetramethylcyclopropanecarboxylate, in or on the food commodities cucurbit vegetables (Crop Group 9) commodities at 0.5 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition. This notice includes a summary of the petition prepared by Valent USA Corporation, the registrant, P.O. Box 8025, Walnut Creek, CA 94596-8025.

A. Residue Chemistry

1. *Plant metabolism*. The plant metabolism of fenpropathrin has been studied in five different crops: cotton, apple, tomato, cabbage, and bean. Fenpropathrin, a cyanohydrin ester, has been labeled with radiocarbon in three positions -- cyclopropyl ring, aryl rings, and nitrile. The permutations of plant species and radiocarbon label position yield a total of 17 separate, reviewed studies. Each of the studies involved foliar treatment of the plants under either greenhouse or field conditions and, while the actual treatment conditions and times to harvest and analyses varied from study to study, the results of the many studies are consistent. The total toxic residue is best defined as parent, fenpropathrin.

Fenpropathrin remains associated with the site of application and only traces are found in seeds (e.g., bean or cotton) or in other parts of the plant not directly exposed to the application. Much of the parent residue can be removed from the plant material with a mild hexane/acetone or hexane rinse, demonstrating that the residue is located on or near the outside surface of the plant material. The primary metabolic pathway for fenpropathrin in plants is similar to that in mammals. There are no qualitatively unique plant metabolites; the primary aglycones are identical in both plants and animals.

2. *Analytical method*. Adequate analytical methodology is available to detect and quantify fenpropathrin (and its metabolites) at residue levels in numerous matrices. The methods use solvent extraction and partition and/or column chromatography clean-up steps, followed by separation and quantitation using capillary column gas-liquid chromatography with flame ionization detection. The extraction efficiency has been validated using radiocarbon samples from the plant and animal metabolism studies. The enforcement methods have been validated at independent laboratories and by EPA. The limit of quantitation (LOQ) for fenpropathrin is 0.01 ppm.

3. *Magnitude of residues*. The field residue data to support the proposed fenpropathrin tolerance on the cucurbit vegetables crop grouping includes data on melons (cantaloupe) from 10 sites, cucumbers from 8 sites and summer squash from 7 sites providing data from 25 sites across the United States. Exaggerated rate and residue decline studies were included. In the samples that fit the proposed use pattern the average residue is 0.078 ppm with a maximum value of 0.31 ppm. Samples with measured residue values below the 0.01 ppm LOQ were assumed, for the purposes of calculation, to contain residue values of 0.005 ppm (1/2 the LOQ).

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicity studies with technical fenpropathrin: Oral lethal dose $(LD)_{50}$ in the rat is 54.0 mg/kg for males and 48.5 mg/kg for females - Toxicity Category I; dermal LD_{50} is 1,600 mg/kg for males and 870 mg/kg for females - Category II; acute inhalation (impossible to generate sufficient test article vapor or aerosol to elicit toxicity) - Category IV; primary eye irritation (no corneal involvement, mild iris and conjunctival irritation) - Category III; and primary dermal irritation (no irritation) - Category IV. Fenpropathrin is not a sensitizer.

2. *Genotoxicty*. An Ames Assay was negative for *Salmonella* TA98, TA100, TA1535, TA1537, and TA1538; and *E. coli* WP2uvrA (trp-) with or without metabolic activation. Sister Chromosome Exchange in Chinese hamster ovary (CHO) cells there were no increases in sister chromatid exchanges seen. Cytogenetics *in vitro* - negative for chromosome aberrations in CHO cells exposed *in vitro* to toxic doses (≥ 30 µg/ml) without activation; and to limit of solubility (1,000 µg/ml) with activation. *In Vitro* Assay in Mammalian Cells - equivocal results - of no concern. DNA Damage/Repair in Bacillus subtilis - not mutagenic or showing evidence of DNA damage at ≥ 5,000 µg/paper disk.

3. *Reproductive and developmental toxicity*. A 3–generation reproduction study was performed with rats dosed with fenpropathrin at concentrations of 0, 40, 120, or 360 ppm (0, 3.0, 8.9, or 26.9 mg/kg/day in males; 0, 3.4, 10.1, or 32.0 mg/kg/day in females, respectively). The parentals (male/female) systemic NOAEL is 40 ppm (3.0/3.4 mg/kg/day). The systemic LOAEL is 120 ppm (8.9/10.1 mg/kg/day) based on body tremors with spasmodic muscle twitches, increased sensitivity and maternal lethality. The reproductive NOAEL is 120 ppm (8.9/10.1 mg/kg/day), and the reproductive LOAEL is 360 ppm (26.9/32.0 mg/kg/day) based on decrease mean F_{1B} pup weight, increased F_{2B} loss. The pups (male/female) developmental NOAEL is 40 ppm (3.0/3.4 mg/kg/day), and the developmental LOAEL is 120 ppm (8.9/10.1 mg/kg/day) based on body tremors, increased mortality.

In a developmental toxicity study in rats, pregnant female rats were dosed by gavage on gestation days 6 through 15 at 0 (corn oil control) 0.4, 1.5, 2.0, 3.0, 6.0, or 10.0 mg/kg/day. The maternal NOAEL is 6 mg/kg/day and the LOAEL is 10 mg/kg/day based on death, moribundity, ataxia, sensitivity to external stimuli, spastic jumping, tremors, prostration, convulsions, hunched posture, squinted eyes, chromodacryorrhea, and lacrimation. The developmental NOAEL is > 10 mg/kg/day.

In a developmental toxicity study in rabbits, pregnant female New Zealand rabbits were dosed by gavage on gestation days 7 through 19 at 0, 4, 12, or 36 mg/kg/day. Maternal NOAEL is 4 mg/kg/day and the maternal LOAEL is 12 mg/kg/day based on grooming, anorexia, flicking of the forepaws. The developmental NOAEL is > 36 mg/kg/day highest dose tested (HDT).

4. *Subchronic toxicity*. In a subchronic oral toxicity study, rats were dosed at concentrations of 0, 3, 30, 100, 300, or 600 ppm in the diet. The LOAEL is 600 ppm (30 mg/kg/day) based on body weight reduction (female), body tremors, and increased brain (female) and kidney (male) weights. The NOAEL is 300 ppm (15 mg/kg/day).

5. *Chronic toxicity*. In a chronic feeding/carcinogenicity study, rats were dosed at 0, 50, 150, 450, or 600 ppm in the diet (0, 1.93, 5.71, 17.06, or 22.80 mg/kg/day in males, and 0, 2.43, 7.23, 19.45, or 23.98 mg/kg/day in females). There was no evidence of carcinogenicity at any dose up to and including 600 ppm. The systemic NOAEL (male) is 450 ppm (17.06 mg/kg/day). The systemic NOAEL (female) is 150 ppm (7.23 mg/kg/day), and the systemic LOAEL (male) is 600 ppm based on increased mortality, body tremors, increased pituitary, kidney, and adrenal weights. The systemic LOAEL (female) is 450 ppm (19.45 mg/kg/day) based on increased mortality and body tremors.

In a chronic feeding/carcinogenicity study, mice were fed diets containing 0, 40, 150, or 600 ppm (0, 3.9, 13.7, or 56.0 mg/kg/day in males, and 0, 4.2, 16.2, or 65.2 mg/kg/day in females). Mortality was highest during the final quarter of the study, but the incidence was similar in all dosed and control groups. No other indications of toxicity or carcinogenicity were seen. The systemic NOAEL is > 600 ppm (HDT; male/female, 56.0/65.2 mg/kg/day).

6. *Animal metabolism*. In a metabolism study in rats, animals were dosed with fenpropathrin radiolabelled in either the alcohol or acid portion of the molecule. Rats received 14 daily oral low-doses of 2.5 mg/kg/day of unlabelled fenpropathrin followed by a 15th dose of either the alcohol or acid radiolabelled fenpropathrin. Groups of rats received a single dose of either of the two radiolabelled test articles at 2.5 mg/kg or 25 mg/kg. The major biotransformations included oxidation at the methyl group of the acid moiety, hydroxylation at the 4'-position of the alcohol moiety, cleavage of the ester linkage, and conjugation with sulfuric acid or glucuronic acid. Four metabolites were found in the urine of rats dosed with alcohol labeled fenpropathrin. The major metabolites were the sulfate conjugate of 3-(4'-hydroxyphenoxy)benzoic acid and 3-phenoxybenzoic acid (22-44% and 3-9% of the administered dose, respectively). The major urinary metabolites of the acid-labeled fenpropathrin were TMPA-glucuronic acid and TMPA-CH_2OH (11-26% and 6-10% of the administered dose, respectively). None of the parent chemical was found in urine. The major elimination products in the feces included the parent chemical (13-34% of the administered dose) and four metabolites. The fecal metabolites (and the percentage of administered dose) included CH_2OH-fenpropathrin (9-20%), 4'-OH-fenpropathrin (4-11%), COOH-fenpropathrin (2-7%), and 4'-OH-CH_2OH-fenpropathrin (2-7%). There are no qualitatively unique plant metabolites. The primary aglycones are identical in both plants and animals; the only difference is in the nature of the conjugating moieties employed.

7. *Metabolite toxicology*. The metabolism and potential toxicity of the small amounts of terminal plant metabolites have been tested on mammals. Glucoside conjugates of 3-phenoxy-benzyl alcohol and 3-phenoxybenzoic acid, administered orally to rats, were absorbed as the corresponding aglycones following cleavage of the glycoside linkage in the gut. The free or reconjugated aglycones were rapidly and completely eliminated by normal metabolic pathways. The glucose conjugates of 3-phenoxybenzyl alcohol and 3-phenoxy-benzoic acid are less toxic to mice than the corresponding aglycones.

8. *Endocrine disruption*. No special studies to investigate the potential for estrogenic or other endocrine effects of fenpropathrin have been performed. However, as summarized above, a large and detailed toxicology data base exists for the compound in all required categories. These studies include evaluations of reproduction and reproductive toxicity and detailed pathology and histology of endocrine organs following repeated or long-term exposure. According to the registrant, these studies are considered capable of revealing endocrine effects and no such effects were observed.

C. Aggregate Exposure

1. *Dietary exposure*. The chronic population adjusted dose (cPAD) is established at 0.025 mg/kg/day. The acute population adjusted dose (aPAD)

is established at 6.0 mg/kg/day (systemic). Thus, both chronic and acute dietary exposure and risk analyses are necessary.

Chronic and acute dietary exposure analyses were performed for fenpropathrin using anticipated residues and accounting for proportion of the crop treated. The crops included in the analyses are the cottonseed, currants, peanuts, strawberries, soybeans and grapes, and the crop groupings head and stem brassica, fruiting vegetables, cucurbit vegetables, citrus fruits, and pome fruits; processed products from these crops; and the resulting secondary residues in meat, milk, and eggs. Currants and soybeans (and soybean products) were entered into the analyses using tolerance-level residues and 100% or 1% of the crop treated, respectively. The fruiting vegetables (Crop Group 9), was substituted for tomatoes in the dietary exposure and risk analyses. IR-4 is presently working on this use expansion, and a tolerance petition adding fruiting vegetables and using these same dietary exposure analyses will be forthcoming. The various proportion of crop treated values were derived from published marketing data for crops for which there are existing fenpropathrin uses, and extrapolated from the uses of other pyrethroid insecticides for pending crops. Proportion of crop treated was assumed to be equal for all crops in a crop grouping. A report of these exposure/risk analyses has been submitted to the Agency including a detailed description of the methodology and assumptions used.

i. *Food*. Chronic dietary exposure was at or below 2.7% of the cPAD with apples and grapes the commodities contributing the most to chronic exposure. The anticipated residue contribution (ARC) is estimated to be 0.000204 milligrams/kilograms/bodyweight/day (mg/kg/ bwt/day) and utilize 0.8% of the cPAD for the overall U.S. population. The ARC for childern 1-6 years old and childern 7-12 years old (subgroups most highly exposed) are estimated to be 0.000678 mg/kg bwt/day and 0.000325 mg/kg bwt/day and utilizes 2.7 and 1.3% of the cPAD, respectively. The ARC for females (13+/Nursing) 0.000248 mg/kg bwt/day and utilizes 1.0% of the cPAD. The ARC for all infants (< 1–year old) and non-nursing infants (<1–year old) is 0.000243 mg/kg bwt/day and 0.000284 mg/kg bwt/day respectivley and utilizes 1.0% of the cPAD. The ARC for nursing infants (< 1–year old) is 0.000103 and utilizes 0.4% of the cPAD. Generally speaking, the registrant has no cause for concern if total residue contribution for published and proposed tolerances is less than 100% of the cPAD.

Acute dietary exposure was calculated at the 99.9th percentile of exposure and margins of exposure (MOE) were calculated for the U.S. population and the subpopulations with the highest risk, as follows: U.S. population (MOE of 490), females (13+) (MOE 927), all infants (MOE 347), nursing infants (< 1) (MOE 384), non-nursing infants (MOE 328), childern 1–6 years old (MOE 238), and childern 7-12 years old (MOE 410). In all cases, margins of exposure exceed one-hundred.

ii. *Drinking water*. Since fenpropathrin is applied outdoors to growing agricultural crops, the potential exists for fenpropathrin or its metabolites to reach ground or surface water that may be used for drinking water. Because of the physical properties of fenpropathrin, the registrant has determined that it is unlikely that fenpropathrin or its metabolites can leach to potable ground water.

To further quantify potential exposure from drinking water, surface water concentrations for fenpropathrin were estimated using genetic expected environmental concentration (GENEEC) 1.2, and the most intense field use scenario. The average 56–day concentration predicted in the simulated pond water was 0.22 parts per billion (ppb). The residence time of fenpropathrin in surface water has been measured and is short. In pond studies, fenpropathrin half-life in the water column were less than 1.5 days, thus this 56–day modeled half-lifes probably considerably overestimates any real surface water concentration. Using standard assumptions about body weight (bwt) and water consumption, the chronic exposure from drinking water would be 6.3×10^{-6} and 2.2×10^{-5} mg/kg bwt/day for adults and children, respectively; less than 0.09% of the cPAD for children. Based on this worse case analysis, the contribution of water to the dietary risk is negligible.

2. *Non-dietary exposure*. Fenpropathrin, as the product TAME 2.4 EC Spray, is a restricted use material and registered for professional non-food use both indoors and outdoors on ornamentals and non-bearing nursery fruit trees. Fenpropathrin has no animal health, homeowner, turf, termite, indoor pest control, or industrial uses. Quantitative information concerning human exposure from this ornamental use is not available, but exposure to the general public from this use of fenpropathrin is expected to be minimal. No endpoints of concern were identified for occupational or residential, dermal or inhalation exposures of any duration. Thus, no risk assessment is needed.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that the Agency must consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' Available information in this context include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way.

E. Safety Determination

1. *U.S. population—i. Chronic risk—adults*. Using the dietary exposure assessment procedures described above for fenpropathrin, calculated chronic dietary exposure resulting from residue exposure from existing and proposed uses of fenpropathrin is minimal. The estimated chronic dietary exposure from food for the overall U.S. population is less than 1% of the cPAD. Addition of the small but worse case potential chronic exposure from drinking water (calculated above, 6.3 x 10-6 mg/kg bwt/day) to the highest chronic exposure value from food increases the maximum occupancy of the cPAD only slightly from 0.99% to 1.02%. Generally, the Agency has no cause for concern if total residue contribution is less than 100% of the cPAD.

ii. *Acute Risk—adults*. The potential acute exposure from food to the U.S. population and various non-child/infant populations subgroups (shown above) provide MOE values greatly exceeding 100. Addition of the worse case, but very small ''background'' dietary exposure from water is not sufficient to change the MOE values significantly. The registrant concludes that there is a reasonable certainty that no harm will result to the overall U.S. population from aggregate, acute exposure to fenpropathrin residues.

2. *Infants and children—safety factor for infants and children*. In assessing the potential for additional sensitivity of

infants and children to residues of fenpropathrin, FFDCA section 408 provides that EPA shall apply an additional margin of safety, up to ten-fold, for added protection for infants and children in the case of threshold effects unless EPA determines that a different margin of safety will be safe for infants and children.

i. *Chronic risk—infants and children.* Using the dietary exposure assessment procedures described above, calculated chronic dietary exposure resulting from residue exposure from existing and proposed uses of fenpropathrin is minimal. The estimated chronic dietary exposure from food to infant and child subgroups ranges from 2.7% [children (1-6 years), 0.000678 mg/kg bwt/day] to 0.4% [nursing infants (< 1–year), 0.000103 mg/kg bwt/day] of the cPAD. Addition of the small but worse case potential chronic exposure from drinking water (calculated above, 2.2 x 10_{-5} mg/kg bwt/day) to the highest chronic exposure value from food increases the maximum occupancy of the cPAD only slightly from 2.7% to 2.8%. The registrant concludes that there is a reasonable certainty that no harm will result to infant and child subgroups of the U.S. population from aggregate, chronic exposure to fenpropathrin residues.

ii. *Acute risk—infants and children.* The potential acute exposure from food to the various child and infant population subgroups all provide MOE values exceeding 100. Addition of the worse-case, but very small ''background'' dietary exposure from water (2.2 x 10^{-5} mg/kg bwt/day) is not sufficient to change the MOE values significantly. The registrant concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate, acute exposure to fenpropathrin residues.

F. International Tolerances

There are no Codex, Canadian, or Mexican residue limits for residues of fenpropathrin in or on cucurbit vegetables (Crop Group 9).

[FR Doc. 99–31442 Filed 12–2–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[FRL–6500–3]

Regulatory Reinvention (XL) Pilot Projects

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of availability of Albuquerque Pretreatment Project XL Draft Final Project Agreement.

SUMMARY: EPA is today requesting comments on a draft Project XL Final Project Agreement (FPA) for the City of Albuquerque. The FPA is a voluntary agreement developed collaboratively by Albuquerque, stakeholders, the State of New Mexico, and EPA. Project XL, announced in the **Federal Register** on May 23, 1995 (60 FR 27282), gives regulated sources the flexibility to develop alternative strategies that will replace or modify specific regulatory requirements on the condition that they produce greater environmental benefits.

If implemented, the draft FPA and a site specific rulemaking would allow Albuquerque to conduct pollution prevention outreach and implementation at up to 50 new businesses per year, and integrate stormwater pollution prevention aspects with its pretreatment program. Albuquerque would attempt to initially reduce loadings of 13 pollutants of concern, and optimize resources to achieve competitive institutional integration of pollution prevention and pretreatment program work. Albuquerque would start the project by conducting sewer sub-basin monitoring to determine where 13 pollutants predominate within the collection system. Through this approach, Albuquerque will focus its efforts to identify and address the most significant industrial, commercial, and residential areas, or conduct project outreach. Albuquerque also proposes to conduct workshops and case studies demonstrating implementation of best management practices (BMPs) for pretreatment dischargers, problem areas, and follow-up needs. One way Albuquerque will demonstrate greater environmental benefit is by monitoring pollutant loadings before and after its pollution prevention outreach and implementation efforts. One of Albuquerque's initial goals would be to try to reduce aluminum, cadmium, chromium, copper, cyanide, fluoride, lead, mercury, molybdenum, nickel, selenium, silver, and zinc by 10–25%. The site specific rulemaking setting forth the specific regulatory flexibility to be implemented will be developed with the assistance of stakeholders and will ensure that the project will fully comply with applicable federal requirements under the Clean Water Act.

DATES: The period for submission of comments ends on December 27, 1999.

ADDRESSES: All comments on the draft Final Project Agreement should be sent to: Adele Cardenas, 6EN–XP, U.S. EPA REGION 6, 1445 Ross Avenue, Suite # 1200, Dallas, TX 75202–2733, or Chad Carbone, U.S. EPA, 401 M Street, SW, Room 1027WT (1802), Washington, DC 20460. Comments may also be faxed to Ms. Cardenas at (214) 665–3177 or Mr. Carbone at (202) 401–2474. Comments will also be received via electronic mail sent to: cardenas.adele@epa.gov or carbone.chad@epa.gov.

FOR FURTHER INFORMATION CONTACT: To obtain a copy of the draft Final Project Agreement, contact: Adele Cardenas, 6EN–XP, U.S. EPA REGION 6, 1445 Ross Avenue, Suite # 1200, Dallas, TX 75202–2733, or Chad Carbone, U.S. EPA, 401 M Street, SW, Room 1027WT (1802), Washington, DC 20460. The documents are also available via the Internet at the following location: ''http://www.epa.gov/ProjectXL''. In addition, public files on the Project are located at EPA Region 6 in Dallas. Questions to EPA regarding the documents can be directed to Adele Cardenas at (214) 665–7210 or Chad Carbone at (202) 260–4296. Additional information on Project XL, including documents referenced in this notice, other EPA policy documents related to Project XL, application information, and descriptions of existing XL projects and proposals, is available via the Internet at ''http://www.epa.gov/ProjectXL''.

Dated: November 23, 1999.

Lisa Lund,

Deputy Associate Administrator, for Reinvention Programs, Office of Reinvention.

[FR Doc. 99–31353 Filed 12–2–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

Certain New Chemicals; Receipt and Status Information

[OPPTS–51937; FRL–6394–4]

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: Section 5 of the Toxic Substances Control Act (TSCA) requires any person who intends to manufacture (defined by statute to include import) a new chemical (i.e., a chemical not on the TSCA Inventory) to notify EPA and comply with the statutory provisions pertaining to the manufacture of new chemicals. Under sections 5(d)(2) and 5(d)(3) of TSC, EPA is required to publish a notice of receipt of a premanufacture notice (PMN) or an application for a test marketing exemption (TME), and to publish periodic status reports on the chemicals