reduction in offspring growth. The maternal NOAEL is similar and the reproductive NOAEL is significantly higher (above the limit dose of 1,000 mg/kg/day) than the NOAEL from the one–year feeding study in dogs used to establish the RfD.

iv. *Reference dose*. Since developmental and reproductive toxicity occurs at levels above the levels shown to exhibit parental toxicity and since these levels are significantly higher than those used to calculate the Reference Dose, K–I Chemical believes the Reference Dose of 0.20 mg/kg/day (20 mg/kg/day and an Uncertainty Factor of 100) is an appropriate measure of safety for infants and children.

Dietary exposure of the most highly exposed subgroup in the population, non-nursing infants (< 1 year old) is 0.025758 mg/kg bw/day. This accounts for 12.9 percent of the RfD. There are no residential uses of prohexadione calcium and contamination of drinking water is extremely unlikely. In addition, there were no significant findings in relevant toxicity studies (i.e., subchronic and chronic toxicity, teratology and multi-generation reproductive studies) which would suggest that prohexadione calcium produces endocrine related effects. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, K–I Chemical concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the residues of prohexadione calcium, including all anticipated dietary exposure and all other non-occupational exposures.

F. International Tolerances

A maximum residue level (MRL) has not been established for prohexadione calcium in peanuts, apples, pears or grass grown for seed by the Codex Alimentarius Commission.

[FR Doc. 01–7520 Filed 3–27–01; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–1011; FRL–6774–5]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical 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–1011, must be received on or before April 27, 2001.

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–1011 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Leonard Cole, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–5412; e-mail address: cole.leonard@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–1011. 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–1011 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–1011. 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 a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains 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 petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

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

Dated: March 19, 2001.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by the petitioner and represents the view of the petitioner. EPA is publishing the petition summary verbatim without editing it 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.

McLaughlin Gormley King Company

PP 0F6168

EPA has received a pesticide petition (PP 0F6168) from McLaughlin Gormley King Company, 8810 Tenth Avenue North, Minneapolis, MN 55427 proposing pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of esfenvalerate in or on the raw agricultural commodities unshelled peanut kernels, 0.20 parts per million (ppm); unshelled cocoa beans, 1.00 ppm; shelled almonds, 50 ppm; and shelled walnuts, 15 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.

A. Residue Chemistry

1. *Plant and animal metabolism*. The metabolism and chemical nature of residues of fenvalerate in plants and animals is adequately understood. The fate of fenvalerate has been extensively studied using radioactive tracers in plant and animal metabolism/nature of the residue studies previously submitted to the Agency. These studies have demonstrated that the parent compound is the only residue of toxicological significance. EPA has concluded that the qualitative nature of the residue is the same for both fenvalerate and esfenvalerate.

2. *Analytical method*. There is a practical analytical method utilizing electron-capture gas chromatography (GC) with nitrogen phosphorous detection available for enforcement with a limit of detection (LOD) that allows monitoring food with residues at or above tolerance levels. The LOD for this updated method is the same as that of the current pesticide analytical manual (PAM) II, which is 0.01 ppm.

3. *Magnitude of residues*. Fenvalerate is a racemic mixture of four isomers (S,S; R,S; S,R; and R,R). Technical Asana® (esfenvalerate) is enriched in the insecticidally active S,S-isomer (84%). Tolerance expressions are proposed for esfenvalerate based on the sum of all isomers. The following tolerances are proposed: unshelled peanut kernels, 0.20 ppm; unshelled cocoa beans, 1.00 ppm; shelled almonds, 50 ppm; and shelled walnuts, 15 ppm; resulting from post-harvest treatment. Magnitude of residue studies support the proposed tolerance.

B. Toxicological Profile

1. *Acute toxicity*. A battery of acute toxicity studies places technical esfenvalerate in toxicity category II for acute oral toxicity (rat LD_{50} 87.2 milligrams/kilograms (mg/kg)), category III for acute dermal (rabbit LD_{50} >2,000 mg/kg) and primary eye irritation (mild irritation in rabbits), and category IV for primary skin irritation (minimal skin irritation in rabbits that reversed within 72 hours after treatment). Acute inhalation on technical grade active ingredient was waived due to negligible vapor pressure. A dermal sensitization test on esfenvalerate in guinea pigs showed no sensitization.

2. *Genotoxicty*. Esfenvalerate did not induce micronuclei in bone marrow of

mice given up to 150 mg/kg intraperitoneally. Esfenvalerate did not induce unscheduled DNA synthesis (UDS) in HeLa cells. Other genetic toxicology studies submitted on racemic fenvalerate indicate that the mixture containing equal parts of the four stereoisomers is not mutagenic in bacteria. The racemic mixture was also negative in a mouse host mediated assay and in a mouse dominant lethal assay.

3. *Reproductive and developmental toxicity*. Esfenvalerate was administered to pregnant female rats by gavage in a pilot developmental study at doses of 0, 1, 2, 3, 4, 5, and 20 mg/kg/day and a main study at 0, 2.5, 5, 10, and 20 mg/kg/day. Maternal clinical signs (abnormal gait and mobility) were observed at 2.5 mg/kg/day and above. A maternal no observed adverse effect level (NOAEL) of 2 mg/kg/day was established on the pilot study. The developmental NOAEL was >20 mg/kg/day.

Esfenvalerate was administered by gavage to pregnant female rabbits in a pilot developmental study at doses of 0, 2, 3, 4, 4.5, 5, and 20 mg/kg/day and a main study at doses of 0, 3, 10, and 20 mg/kg/day. Maternal clinical signs (excessive grooming) were observed at 3 mg/kg/day and above. A maternal NOAEL of 2 mg/kg/day was established on the pilot study. The developmental NOAEL was >20 mg/kg/day.

A 2–generation feeding study with esfenvalerate was conducted in the rat at dietary levels of 0, 75, 100, and 300 ppm. Skin lesions and minimal (non biologically significant) parental body weight effects occurred at 75 ppm. The NOAEL for reproductive toxicity was 75 ppm (4.2–7.5 mg/kg/day) based on decreased pup weights at 100 ppm.

4. *Subchronic toxicity.* Two 90–day feeding studies with esfenvalerate were conducted in rats--one at 50, 150, 300, and 500 ppm esfenvalerate, and a second at 0, 75, 100, 125, and 300 ppm to provide additional dose levels. The NOAEL was 125 ppm (6.3 mg/kg/day) based on clinical signs (jerky leg movements) observed at 150 ppm (7.5 mg/kg/day) and above. A three-month subchronic study in dogs was satisfied by 1 year oral study in dogs, in which the NOAEL was 200 ppm (5 mg/kg/day).

5. *Chronic toxicity*. The NOAEL was 200 ppm (5 mg/kg/day). An effect level for dietary administration of esfenvalerate to dogs of 300 ppm had been established earlier in a 3–week pilot study used to select dose levels for the chronic dog study.

One chronic study with esfenvalerate and three chronic studies with fenvalerate have been conducted in mice.

In an 18–month study, mice were fed 0, 35, 150, or 350 ppm esfenvalerate. Mice fed 350 ppm were sacrificed within the first 2 months of the study after excessive self-trauma related to skin stimulation and data collected were not used in the evaluation of the oncogenic potential of esfenvalerate. The NOAEL was 35 ppm (4.29 and 5.75 mg/kg/day for males and females, respectively) based on lower body weight and body weight gain at 150 ppm. Esfenvalerate did not produce carcinogenicity.

In a 2–year feeding study, mice were administered 0, 10, 50, 250, or 1,250 ppm fenvalerate in the diet. The NOAEL was 10 ppm (1.5 mg/kg/day) based on granulomatous changes (related to fenvalerate only, not esfenvalerate) at 50 ppm (7.5 mg/kg/day). Fenvalerate did not produce carcinogenicity.

In an 18–month feeding study, mice were fed 0, 100, 300, 1,000, or 3,000 ppm fenvalerate in the diet. The NOAEL is 100 ppm (15.0 mg/kg/day) based on fenvalerate-related microgranulomatous changes at 300 ppm (45 mg/kg/day). No compound-related oncogenicity occurred.

Mice were fed 0, 10, 30, 100, or 300 ppm fenvalerate for 20 months. The NOAEL was 30 ppm (3.5 mg/kg/day) based on red blood cell effects and granulomatous changes at 100 ppm (15 mg/kg/day). Fenvalerate was not carcinogenic at any concentration.

In a 2–year study, rats were fed 1, 5, 25, or 250 ppm fenvalerate. A 1,000 ppm group was added in a supplemental study to establish an effect level. The NOAEL was 250 ppm (12.5 mg/kg/day). At 1,000 ppm (50 mg/kg/day), hind limb weakness, lower body weight, and higher organ-to-body weight ratios were observed. Fenvalerate was not carcinogenic at any concentration. (A conclusion that fenvalerate is associated with the production of spindle cell sarcomas at 1,000 ppm was retracted by EPA).

EPA has classified esfenvalerate in Group E--evidence of noncarcinogenicity for humans.

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6. *Animal metabolism*. After oral dosing with fenvalerate, the majority of the administered radioactivity was eliminated in the initial 24 hours. The metabolic pathway involved cleavage of the ester linkage followed by hydroxylation, oxidation, and conjugation of the acid and alcohol moieties.

7. *Metabolite toxicology*. The parent molecule is the only moiety of toxicological significance appropriate for regulation in plant and animal commodities.

8. *Endocrine disruption*. Estrogenic effects have not been observed in any studies conducted on fenvalerate or esfenvalerate. In subchronic or chronic studies there were no lesions in reproductive systems of males or females. In the recent reproduction

study with esfenvalerate, full histopathological examination of the pituitary and the reproductive systems of males and females was conducted. There were no compound-related gross or histopathological effects. There were also no compound-related changes in any measures of reproductive performance including mating, fertility, or gestation indices or gestation length in either generation. There have been no effects on offspring in developmental toxicity studies. EPA is required to develop an endocrine disrupter screening program. EPA will decide whether further testing of esfenvalerate is required when this program is in place.

C. Aggregate Exposure

1. *Dietary exposure*. Tolerances have been established for the residues of fenvalerate/esfenvalerate, in or on a variety of agricultural commodities. In addition, pending tolerance petitions exist for use of esfenvalerate on sugar beets, sorghum, head lettuce, celery, pistachios, and a number of other minor use commodities. For purposes of assessing dietary exposure, chronic and acute dietary assessments have been conducted using all existing and pending tolerances for esfenvalerate. EPA reviewed (August 2, 1997) the existing toxicology data base for esfenvalerate and selected the following toxicological endpoints. For acute toxicity, EPA established a NOAEL of 2.0 mg/kg/day from rat and rabbit developmental studies based on maternal clinical signs at higher concentrations. A margin of exposure (MOE) of 100 was required. For chronic toxicity EPA established the reference dose (RfD) for esfenvalerate at 0.02 mg/kg/day. This RfD was also based on a NOAEL of 2.0 mg/kg/day in the rat developmental study with an uncertainty factor (UF) of 100. Esfenvalerate is classified as a Group E. There is no evidence of carcinogenicity in either rats or mice.

2. *Food*. A chronic dietary exposure assessment was conducted using Novigen's dietary exposure estimate model (DEEM). Anticipated residues and adjustment for percent crop treated were used in the chronic dietary risk assessment. The percentages of the RfD utilized by the most sensitive sub-population, children 1 to 6 years, was 4.6% based on a daily dietary exposure of 0.000911 mg/kg/day. Chronic exposure for the overall U.S. population was 1.9% of the RfD based on a dietary exposure of 0.000376 mg/kg/day. This assessment has been approved by EPA and included pending tolerances (including lettuce) and all food tolerances for incidental residues from use in food handling establishments. EPA 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. Esfenvalerate is classified as a Group E carcinogen- -no evidence of carcinogenicity in rats or mice. Therefore, a carcinogenicity risk analysis is not required.

Potential acute exposures from food commodities were estimated using a Tier 3 (Monte Carlo) analysis and appropriate processing factors for processed food and distribution analysis. This analysis used field trial data to estimate exposure and Federal and market survey information to derive the percent of crop treated. EPA considered these data reliable and used the upper end estimate of percent crop treated in order to not underestimate any significant subpopulation. Regional consumption information was taken into account. The MOEs for the most sensitive sub-population (children 1 to 6 years) were 202 and 103 at the 99th and 99.9th percentile of exposure, respectively, based on daily exposures of 0.009908 and 0.019445 mg/kg/day. The MOEs for the general population are 355 and 171 at the 99th and 99.9th percentile of exposure, respectively, based on daily exposure estimates of 0.005635 and 0.011717 mg/kg/day. EPA has stated there is no cause for concern if total acute exposure calculated for the 99.9th percentile yields a MOE of 100 or larger. This acute dietary exposure estimate is considered conservative and EPA considered the MOEs adequate in a final rule (62 FR 63019, November 26, 1997).

3. *Drinking water*. Esfenvalerate is immobile in soil and will not leach into ground water. Due to the insolubility and lipophilic nature of esfenvalerate, any residues in surface water will rapidly and tightly bind to soil particles and remain with sediment, therefore not contributing to potential dietary exposure from drinking water.

A screening evaluation of leaching potential of a typical pyrethroid was conducted using EPA's pesticide root zone model (PRZM). Based on this screening assessment, the potential concentrations of a pyrethroid in ground water at depths of 1 and 2 meters are essentially zero (much less than 0.001 parts per billion (ppb)).

Surface water concentrations for pyrethroids were estimated using PRZM^3 and exposure analysis modeling system (EXAMS) using standard EPA cotton runoff and Mississippi pond scenarios. The maximum concentration predicted in the simulated pond was 0.052 ppb. Concentrations in actual drinking water would be much lower than the levels predicted in the hypothetical, small, stagnant farm pond model since drinking water derived from surface water would be treated before consumption. Chronic drinking water exposure was estimated to be 0.000001 mg/kg/day for both the U.S. general population and for non-nursing infants. Less than 0.1% of the RfD was occupied by both population groups.

Using these values, the contribution of water to the acute dietary risk estimate was estimated for the U.S. population to be 0.000019 mg/kg/day at the 99th percentile and 0.000039 mg/kg/day at the 99.9th percentile resulting in MOEs of 105,874 and 51,757, respectively. For the most sensitive subpopulation, non-nursing infants less than 1 year old, the exposure is 0.000050 mg/kg/day and 0.000074 mg/kg/day at the 99th and 99.9th percentile, respectively, resulting in MOEs of 39,652, and 27,042, respectively. Therefore there is reasonable certainty of no harm from exposure to esfenvalerate from drinking water.

4. *Non-dietary exposure*. Esfenvalerate is registered for non-crop uses including spray treatments in and around commercial and residential areas, treatments for control of ectoparasites on pets, home care products including foggers, pressurized sprays, crack and crevice treatments, lawn and garden sprays, and pet and pet bedding sprays. For the non-agricultural products, the very low amounts of active ingredient they contain, combined with the low vapor pressure (1.5×10^{-9} mm mercury at 25 ^{o}C) and low dermal penetration, would result in minimal inhalation and dermal exposure.

To assess risks from (nonfood) short-term and intermediate-term exposure, EPA has recently selected a toxicological endpoint of 2.0 mg/kg/day, the NOAEL from the rat and rabbit developmental studies. For dermal penetration/absorption, EPA selected 25% dermal absorption based on the weight-of-evidence available for structurally-related pyrethroids. For inhalation exposure, EPA used the oral NOAEL of 2.0 mg/kg/day and assumed 100% absorption by inhalation. Individual non-dietary risk exposure analyses were conducted using a flea infestation scenario that included pet spray, carpet, and room treatment, and lawn care, respectively. The total potential short-term and intermediate-term aggregate non-dietary exposure including lawn, carpet, and pet uses are: 0.000023 mg/kg/day for adults, 0.00129

mg/kg/day for children 1 to 6 years, and 0.00138 mg/kg/day for infants less than one year old.

EPA concluded that the potential non-dietary exposure for esfenvalerate are associated with substantial margins of safety (62 FR 63019).

5. *Aggregate exposure—dietary and non dietary exposure*. EPA has concluded that aggregate chronic exposure to esfenvalerate from food and drinking water will utilize 2.0% of the RfD for the U.S. population based on a dietary exposure of 0.000378 mg/kg/day. The major identifiable subgroup with the highest aggregate exposure are children 1 to 6 years old. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health.

The acute aggregate risk assessment takes into account exposure from food and drinking water. The potential acute exposure from food and drinking water to the overall U.S. population provides an acute dietary exposure of 0.011756 mg/kg/day with an MOE of 170. This acute dietary exposure estimate is considered conservative, using anticipated residue values and percent crop treated data in conjunction with Monte Carlo analysis.

Short-term and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. The potential short-term and intermediate-term aggregate risk for the U.S. population is an exposure of approximately 0.0082 mg/kg/day with an MOE of approximately 244.

It is important to acknowledge that these MOEs are likely to significantly underestimate the actual MOEs due to a variety of conservative assumptions and biases inherent in the exposure assessment methods used for their derivation. Therefore, it can be concluded that the potential non-dietary and dietary aggregate exposures for esfenvalerate are associated with a substantial degree of safety. EPA has previously determined (62 FR 63019) that there was reasonable certainty that no harm will result from aggregate exposure to esfenvalerate residues. Head lettuce was included in that risk assessment.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' In a final rule on esfenvalerate (62 FR 63019) EPA concluded, ''available information'' in this context might 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. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on chemical-specific data, much of which may not be presently available.

Although at present the Agency is not certain how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides for which common mechanism issues can be resolved. These pesticides include those that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed). Although esfenvalerate is similar to other members of the synthetic pyrethroid class of insecticides, EPA does not have, at this time, available data to determine whether esfenvalerate has a common method of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, esfenvalerate does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that esfenvalerate has a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*. Both the chronic and acute toxicological endpoints are derived from maternal NOAELs of 2.0 mg/kg/day in developmental studies in rats and rabbits. There were no fetal effects. In addition, no other studies conducted with fenvalerate or esfenvalerate indicate that immature animals are more sensitive than adults. Therefore, the safety factor used for protection of adults is fully appropriate for the protection of infants and children; no additional safety factor is necessary as described below. A chronic dietary exposure assessment using anticipated residues, monitoring information, and percent crop treated indicated the percentage of the RfD utilized by the general population to be 2.0%. There is generally no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health.

For acute exposure, a MOE greater than 100 is considered adequate. A Tier 3 acute dietary exposure assessment found the general population to have MOEs of 355 and 171 at the 99th and 99.9th percentile of exposure, respectively. These values were generated using actual field trial residues and market share data for percentage of crop treated. These results depict an accurate exposure pattern at an exaggerated daily dietary exposure rate.

Short-term and intermediate-term aggregate exposure risk from chronic dietary food and water plus indoor and outdoor residential exposure for the U.S. population is an exposure of approximately 0.0082 mg/kg/day with an MOE of approximately 244.

Therefore, there is a reasonable certainty that no harm will result from chronic dietary, acute dietary, non-dietary, or aggregate exposure to esfenvalerate residues.

2. *Infants and children*. FFDCA section 408 provides that EPA shall apply an additional ten-fold margin of safety for infants and children unless EPA determines that a different margin of safety will be safe for infants and children. EPA has stated that reliable data support using the standard MOE and UF (100 for combined interspecies

and intraspecies variability) and not the additional ten-fold MOE/UF when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor. In a final rule (62 FR 63019), EPA concluded that reliable data support use of the standard 100-fold UF for esfenvalerate, and that an additional UF is not needed to protect the safety of infants and children. This decision was based on no evidence of developmental toxicity at doses up to 20 mg/kg/day (ten times the maternal NOAEL) in prenatal developmental toxicity studies in both rats and rabbits; toxicity to offspring only at dietary levels which were also found to be toxic to parental animals in the 2–generation reproduction study; and no evidence of additional sensitivity to young rats or rabbits following prenatal or postnatal exposure to esfenvalerate.

A chronic dietary exposure assessment found the percentages of the RfD utilized by the most sensitive subpopulation to be 4.8% for children 1 to 6 years based on a dietary exposure of 0.000957 mg/kg/day. The percent RfD for children 7 to 12 years was 3.0%. The Agency has no cause for concern if RfDs are below 100%.

The most sensitive subpopulation, children 1 to 6 years, had acute dietary MOEs of 202 and 103 at the 99th and 99.9th percentile of exposure, respectively. Nursing infants had MOEs of 195 and 146 at the 99th and 99.9th percentile of exposure, respectively. Non-nursing infants had MOEs of 304 and 158 at the 99th and 99.9th percentile of exposure, respectively. The Agency has no cause for concern if total acute exposure calculated for the 99.9th percentile yields an MOE of 100 or larger. EPA has concluded that the potential short-term or intermediate-term aggregate exposure of esfenvalerate from chronic dietary food and water plus indoor and outdoor residential exposure to children (1 to 6 years old) is 0.0113 mg/kg/day with an MOE of 177. For infants (less than 1 year old) the exposure is 0.0098 mg/kg/day with an MOE of 204. Thus, there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to esfenvalerate residues (62 FR 63019).

F. International Tolerances

Codex maximum residue levels (MRLs) have been established for residues of fenvalerate on a number of crops that also have U.S. tolerances. There are some minimal differences between the section 408 tolerances and certain Codex MRL values. These differences could be caused by differences in methods to establish tolerances, calculate animal feed, dietary exposure, and as a result of different agricultural practices. Therefore, some harmonization of these maximum residue levels will be required.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–1007; FRL–6775–1]

Notice of Filing Pesticide Petitions to Establish Tolerances for a Certain Pesticide Chemical 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 a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket control number PF–1007, must be received on or before April 27, 2001.

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–1007 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Linda Hollis, Biopesticides and Pollution Prevention Division (7511C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–8263; e-mail address: hollis.linda@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–1007. 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,