concern for dietary exposure to cryolite is skeletal fluorosis.

EPA estimated that total dietary fluoride exposure, including food and drinking water, is 0.085 mg/kg/day. Of this total exposure, the dietary (food) contribution is about 0.020 mg/kg/day for the U.S. population and 0.028 mg/kg/day for the highest exposed subgroup (nursing females 13 years old and over). The proposed mint tolerances will contribute no more than 0.000001 mg/kg/day to total dietary exposure. Thus, the proposed tolerance would have essentially no effect on total fluoride exposure. The total exposure to fluoride from all sources is well below the MCL of 4.0 mg/L (0.114 mg/kg/day).

2. *Infants and children*. EPA has previously concluded on December 5, 1997 (62 FR 64294), that based on current data requirements, the data base relative to prenatal and postnatal toxicity is complete. This data taken together suggest minimal concern for developmental or reproductive toxicity and do not indicate any increased prenatal or postnatal sensitivity. Therefore, EPA concluded that reliable data support the weight-of-evidence risk assessment approach for the assessment of risks to infants and children associated with the use of cryolite and that an additional safety factor is not needed.

F. International Tolerances

No Codex, European or other international tolerances are in effect for cryolite; thus potential dietary exposure to fluoride from the agricultural use of cryolite on crops would not include imported foodstuffs.

[FR Doc. 01–28861 Filed 11–20–01; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–1042; FRL–6799–1]

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–1042, must be received on or before Decmber 21, 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–1042 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Rita Kumar, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–8391; e-mail address: kumar.rita@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,'' ''Regulations and Proposed Rules,'' 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–1042. 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–1042 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–1042. 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 Comestic 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: October 24, 2001.

Peter Caulkins,

Acting 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.

ISK Biosciences Corporation

6F4662, 6F4701, 6F4755, 6E4773, 5E4474

EPA has received pesticide petitions (6F4662, 6F4701, 6F4755, 6E4773, 5E4474) from ISK Biosciences Corporation, 7470 Auburn Road, Suite A, Concord, OH, 44077, proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the nematicide, fosthiazate, ((RS)-S-sec-butyl O-ethyl 2-oxo-1,3-thiazolidin-3-ylphosphonothioate) and its metabolite ASC-67131 (BESoP, (RS)-S-sec-butyl O-ethyl N-2(methylsulfonyl)ethyl phosphoramidothioate)] in or on the raw agricultural commodities tomatoes and peanuts at 0.02 parts per million (ppm), potatoes at 0.03 ppm, and import tolerances on bananas and green coffee beans at 0.05 parts per million (ppm). 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 supports granting of the petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

1. *Plant metabolism*. Metabolism studies were conducted on tomatoes, potatoes, and peaches. Fosthiazate is extensively metabolized in plants by a combination of hydrolytic and oxidative processes that convert the parent to small polar fragments. Residues in mature edible plant parts consist of polar S-butyl group degradates and radioactivity that is reincorporated into plant natural products from thiazolidinone ring carbon atoms. Analyses of leaf and stem tissues established the identity of intermediate metabolites in the pathway.

2. *Analytical method*. A gas chromatographic analytical method using a flame photoionization detector in the phosphorus mode has been validated for enforcement purposes. The limit of detection for both fosthiazate and its metabolite ASC-67131 is 0.01 ppm for all crops (tomatoes, potatoes, peanuts, bananas, green coffee beans and roasted coffee.) The limit of detection for both parent and metabolite in instant coffee is 0.05 ppm.

3. *Magnitude of residues*—i. *Tomatoes*. The application rate of 4–6 lbs of the active ingredient per acre (a.i./acre) was applied and tomatoes harvested from 80–147 days after application in 6 of 15 test sites. No detectable residues of either parent or the metabolite ASC-67131 were found (LOD = 0.01 ppm). The proposed maximum label rate is 4.5 lb a.i./acre.

ii. *Potatoes*. The application rate of 4–6 lb of the a.i. was applied and potatoes harvested from 82–179 days after application in 11 test sites. The maximum fosthiazate residue found was 0.02 ppm at 4 lb ai/acre and 0.07 ppm at 6 lb ai/acre, while no detectable residues of the metabolite ASC-67131 were found. The proposed maximum label rate is 4.5 lb a.i./acre.

iii. *Peanuts*. The application rate of 4-6 lb of the a.i./acre was applied and peanuts harvested from 99–175 days after application in 16 test sites. No detectable residues of either parent or the metabolite ASC-67131 were found (LOD = 0.01 ppm) in either peanut nutmeat or hay. The proposed maximum label rate is 4.0 lb a.i./acre.

iv. *Bananas*. The maximum application rate of 2 grams of the active

ingredient per mat was applied and bananas harvested from 0–126 days after application in 6 of 15 test sites. The maximum fosthiazate residue found was 0.03 ppm, while no detectable residues of the metabolite ASC-67131 were found.

v. *Coffee*. The maximum application rate of 2 grams of the active ingredient per plant was applied 60 days prior to harvest in eight test sites. The maximum fosthiazate residue found was 0.02 ppm, while no detectable residues of the metabolite ASC-67131 were found. After a single application of fosthiazate at 10 grams per plant 31 days before harvest, roasted coffee beans and instant coffee were analyzed. No residues were found above the limit of detection of the method in the processed fractions.

B. Toxicological Profile

1. *Acute toxicity*. Results of the acute toxicity testing are described below.

Oral toxicity to rats (suspended in corn oil)	LD_{50} is 73 mg/kg bwt (males) 57 mg/kg bwt (females)
Oral toxicity to rats (solution in water)	LD_{50} is 49 mg/kg bwt (males) 28 mg/kg bwt (females)
Dermal toxicity to rats	LD_{50} is 2,372 mg/kg bwt (males) 853 mg/kg bwt (females)
Inhalation toxicity to rats (4-hour) exposure	LC_{50} is 0.83 milligrams per Liter (mg/L) (males) 0.56 mg/L (females)
Dermal irritation, rabbits	No irritation
Eye irritation, rabbits no wash	Irritation reversible in 1 week, mortality
Eye irritation, rabbits with wash	Irritation, reversible in 1 week, no mortality
Skin sensitization Magnusson-Klingman	Positive
Acute delayed neurotoxicity: hen	Negative
Acute cholinesterase no observed adverse effect level (NOAEL): rat	No inhibition of cholinesterase activity at 4 mg/kg bwt in erythrocyte or brain
Acute neurotoxicity: rat	NOEL for functional observation battery, motor activity and neuropathology:10 mg/kg bwt

2. *Genotoxicty*. A battery of tests has been conducted to assess the genotoxic potential of technical fosthiazate. Assays conducted included *in vitro* gene mutation tests in bacteria and mammalian cell systems, a chromosomal aberration test in mammalian cells, a DNA repair test in bacteria and an *in vivo* micronucleus test in mice. Technical fosthiazate did not elicit a genotoxic response in any of the studies conducted.

3. *Reproductive and developmental toxicity*. In a developmental study with rats at 0, 3, 5, and 10 mg/kg bwt/day the NOAEL for maternal toxicity based on the maternal body weight reduction was 5 mg/kg bwt/day. The NOAEL for developmental effects was 10 mg/kg bw/day. Technical fosthiazate did not cause developmental toxicity in rats.

A developmental study with rabbits was conducted at dosages 0, 0.5, 1.0, 1.5 or 2.0 mg/kg bwt/day. The NOAEL for maternal effects in this study was 2.0 mg/kg bwt/day. This dosage was considered very close to a maternally toxic dose because, in the preliminary study, maternal lethality was observed at 5 mg/kg bwt/day and there were isolated incidences of maternal animals in extremis at 2.0 and 2.5 mg/kg bwt/day. The NOAEL for developmental effects was considered 2.0 mg/kg bwt/day. Technical fosthiazate was not teratogenic in rabbits.

In a two generation reproduction study, technical fosthiazate was administered via the diet at concentrations of 0, 3.2, 10.7, 32.2 or 107.2 ppm to CD rats. The group receiving 107.2 ppm was terminated at weaning of the F0 generation due to poor survival of offspring. In the first generation, there was a statistically significant increase in the length of gestation at 107.2 ppm. The difference was not significant at any other dosage or in the second generation. Viability indices and body weight gain of F1 offspring were reduced at 32.2 ppm and higher. These effects were particularly marked at 107.2 ppm. No effects on pup viability were observed in the second generation. The dietary concentration of 10.7 ppm which was equivalent to 0.86 mg/kg bwt/day was the NOAEL for the effects on pup survivability and pup body weight observed in this study.

4. *Subchronic toxicity*. In a 13–week feeding study in rats with a recovery phase, Sprague-Dawley rats received technical fosthiazate via the diet at concentrations of 0, 1.07, 10.7, 53.6 or 429 ppm for 13 weeks. The NOAEL for the study was 10.7 ppm technical fosthiazate in the diet based on inhibition of brain cholinesterase activity and adrenal effects. The effects were reversible.

In a 13–week study, beagle dogs received technical fosthiazate at dosages of 0, 0.054, 0.11, 0.54, or 5.4 mg/kg bwt/day daily. The NOAEL for adrenal effects and cholinesterase inhibition in the erythrocyte and brain, was 0.54 mg/kg bwt/day.

A 90–day dietary neurotoxicity study in rats was conducted at doses of 0, 0.07, 0.56 and 2.4 mg/kg bwt/day and of 0, 0.08, 0.57 and 2.5 mg/kg bwt/day to males and females, respectively. In spite of lower cholinesterase levels, no clinical signs of cholinesterase inhibition, no differences in the functional observational battery, in mean forelimb and hind limb grip strengths, mean foot-spread, mean motor activity values or neuropathology were observed in the animals administered 2.5 mg/kg bwt/day of technical fosthiazate via the diet. The NOAEL for inhibition of cholinesterase activity in the brain was 0.56 mg/kg bwt/day.

In a 21–day dermal toxicity study in rats, at dosages of 0, 0.5, 2.5, 25, or 250 mg/kg bwt/day by occluded dermal application for 21 days, the NOAEL in terms of cholinesterase inhibition in the brain was 2.5 mg/kg bwt/day.

5. *Chronic toxicity*. In a 2–year feeding study in rats, technical fosthiazate was administered to CD rats at dietary concentrations of 0, 1.07, 10.7, 53.6, or 214 ppm. Treatment did not change the incidence of any neoplasm. The NOAEL for the study which was based on adrenal effects and cholinesterase inhibition in the brain, was 10.7 ppm in the diet which was equivalent to 0.41 mg/kg bwt/day.

In the mouse oncogenicity study, technical fosthiazate was administered for a period of 102 weeks to CD–1 mice at concentrations of 0, 10.7, 32.2, 107, or 322 ppm. There was no evidence of oncogenic potential. The NOAEL of technical fosthiazate in CD–1 mice was considered to be 32.2 ppm which was equivalent to 3.32 mg/kg bwt/day.

A 12–month oral chronic toxicity study was conducted in beagle dogs at dose levels of 0, 0.05, 0.1, 0.5 and 5.0 mg/kg bwt/day. No treatment-related change in brain cholinesterase activity was noted. The NOAEL which was based upon adrenal effects was 0.5 mg/kg bwt/day.

Comparison of the toxicology data from subchronic (90 days exposure) and chronic studies showed no major differences in effects or in effect levels. Therefore, a single reference dose (RfD) for subchronic and chronic exposure is proposed.

6. *RfD*. Fosthiazate is nonteratogenic, nononcogenic, and nonmutagenic and there is no evidence of bioaccumulation. Inhibition of cholinesterase activity is considered the primary treatment-related effect from fosthiazate. Although cholinesterase activity was measured in plasma, erythrocytes and brain in the

toxicity studies with fosthiazate, the values from the brain are considered the most relevant for assessing adverse effects. For cholinesterase activity, only data for inhibition of activity in the brain will therefore be included in the selection of a NOAEL for the RfD. In addition to inhibition of cholinesterase activity, fosthiazate treatment was associated with other effects.

The lowest NOAEL value was 0.41 mg/kg bwt/day for the inhibition of brain cholinesterase activity from the 2–year feeding study in rats. In that study, rats were fed a constant dietary concentration of fosthiazate. The NOAEL for inhibition of brain cholinesterase activity was 10.7 ppm in the diet for both males and females. Since relative food consumption is a little lower in males than females, the compound consumption was a little lower in males. Since females have been shown to be more sensitive to the effects of fosthiazate than males, and NOAEL were observed in the female group fed a diet containing 10.7 ppm which gave a dose of 0.54 mg/kg bwt/day, the conclusion could be made that 0.54 mg/kg bwt/day would be an appropriate NOAEL. The more conservative value of 0.41 mg/kg bwt/day will be proposed to add to the certainty of no adverse effects. The standard safety factor of 100 will be applied to the conservative NOAEL of 0.41 mg/kg bwt/day to give a proposed RfD of 0.0041 mg/kg bwt/day.

7. *Animal metabolism*. Fosthiazate is extensively metabolized in rats by a combination of hydrolytic and oxidative processes that rapidly convert the parent molecule to small fragments, including CO_2. The carbon atoms of the thiazolidinone ring appear to be reincorporated into tissues based on the levels found in carcasses at termination. Extensive conjugation via the glutathione pathway appears to occur.

8. *Metabolite toxicology*. Comparison of the metabolism of fosthiazate by plants and in animals indicates that a number of the identified metabolites are common to both plants and animals but metabolism in plants is more extensive than in animals. There are, however, no metabolites of toxicological concern in plants that do not appear in animal studies.

9. *Endocrine disruption*. Although subtle histological changes were observed in the ovary and adrenals which are organs with endocrine function, there were no treatment-related effects associated with fosthiazate treatment which are indicative of an effect on endocrine function. Since the histological changes in the adrenals and ovaries were observed only at dosages which also inhibited cholinesterase activity, it is considered possible that the changes were physiological adaptions secondary to inhibition of cholinesterase activity. There were no other effects observed in the subchronic or chronic studies, such as changes to the uterus or mammary tissue or changes in urine production which might indicate a change in physiology related to the ovarian or adrenal changes.

In the reproduction study with fosthiazate, fertility and gestation indices were unaffected by fosthiazate even when administered at dietary concentrations which would result in severe inhibition of cholinesterase activity. At those high dietary concentrations (107.2 ppm) the only treatment-related difference in reproductive effect was a statistically significant increase in length of gestation. It is considered that the effect on gestation could have been secondary to maternal toxicity. The difference was not significant at any other dosage in the first or second generation.

From the reproduction study, reproductive capacity was unaffected by treatment; ovarian effects were not observed; and adrenal effects were observed only in groups administered 107.2 ppm.

The conclusion can be drawn that no effects on the endocrine system would be expected below the threshold for cholinesterase inhibition. Therefore, a NOAEL set for cholinesterase inhibition should also cover any other effects.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. For purposes of assessing the potential dietary exposure, EPA initially estimates exposure using the tolerance (i.e., 0.02 ppm on tomatoes and peanuts, 0.03 ppm on potatoes, 0.05 ppm on bananas and whole green coffee beans) as a worst case scenario. The potential exposure is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of treated products consumed by various population subgroups. In chronic analyses, the average consumption for all individuals in a population subgroup is used, while in acute analyses only consumers of treated commodities are included. While both potato and peanut fractions are fed to animals, metabolism studies show that residues are incorporated into natural products, and thus there is no exposure to residues of toxicological concern through secondary residues in meat, milk and eggs.

ii. *Drinking water*. The potential for residues of fosthiazate to occur in tap water, non-tap water, and water in commercially prepared food was also evaluated. Four field dissipation studies were conducted with fosthiazate in California, Georgia, North Carolina and Washington (Doc. # 3931–95-EF-000). These studies clearly demonstrate that fosthiazate and its degradates do not leach under field conditions, and that the DT90 of the parent compound ranged from 48–92 days. It is therefore reasonable to conclude that the potential for fosthiazate to contaminate ground water is extremely low. As fosthiazate will be incorporated into the soil after application, no significant runoff or spray drift is expected. Therefore, contamination of surface water is highly unlikely. Additionally for bananas and coffee, the proposed tolerance of fosthiazate is for import commodities only. Due to these factors, residues of fosthiazate are not expected in drinking water.

2. *Non-dietary exposure*. Since there are no domestic uses (home/garden) for fosthiazate, there are no non-occupational exposures.

D. Cumulative Effects

Fosthiazate is an organophosphate, with the most sensitive indicator of toxicity being inhibition of cholinesterase after both short- and long-term administration. While the exact mechanism for this effect may or may not be identical to other organophosphates, in the case of the present petitions, this effect is considered to be insignificant. This is due primarily to the extremely low exposure to the U.S. population from the proposed uses of fosthiazate. The incremental increase in exposure to organophosphates from the addition of fosthiazate is extremely small. For example, the highest exposed population subgroups from chronic exposure to residues in/on tomatoes, potatoes and peanuts are children 1–6 yrs with an estimated chronic exposure of 0.000132 mg/kg bwt/day, which represents 3.2% of the RfD. The highest exposed population subgroup from chronic exposure to residues in/on bananas is non-nursing infants 1 year old with an estimated chronic exposure of 0.000045 mg/kg bwt/day, which represents 1.1% of the RfD. The highest exposed population subgroup from chronic exposure to residues in/on coffee are seniors (55+), females (20+) and males (20+) with 0.000002 mg/kg bwt/day, which represents an insignificant portion of the RfD. When all crops, are included in the assessment estimated chronic exposure for children 1–6 yrs increases to 0.000173 mg/kg bwt/day, which represents 4.2% of the

RfD. This is particularly relevant in that this assessment assumed tolerance level residues for all crops (0.02 ppm for tomatoes and peanuts, 0.03 ppm for potatoes and 0.05 ppm for bananas and coffee). Indeed, when anticipated residues are used estimated exposure is less than 2% of the RfD for all population groups.

A similar situation applies to acute exposure (and risk) from the proposed uses. For tomatoes, potatoes and peanuts, the highest exposed subgroup is all infants 1 year old, with an acute exposure of 0.000479 mg/kg bwt/day at the 95th percentile for consumers only. This results in a MOE of 8,300, which exceeds the traditional level considered to provide adequate protection by nearly two orders of magnitude. When residues on bananas and coffee beans are included in the assessment, children 1-6 yrs have an estimated acute exposure at the 95th percentile of 0.000588 mg/kg bwt/day, which results in an MOE of 6,800. Again, when anticipated residues, as calculated for acute exposure (i.e., the highest field trial residue), are used in the assessment for all the proposed crops, the highest exposure is only 0.000456 mg/kg bwt/day at the 95th percentile, with an MOE of 8,700 for all infants (consumers only). Indeed MOE's at the 99.9th percentile of exposure are far higher than generally is considered to be safe by the agency for all population subgroups.

E. Safety Determination

1. *U.S. population.* Using the conservative exposure assumptions and the proposed RfD and acute NOEL described above, dietary exposure was calculated.

As discussed above, even under the ''worst-case'' chronic exposure scenario, a very small portion of the RfD was used. When anticipated residues for tomatoes, potatoes and peanuts are used in the chronic dietary exposure assessment, the estimated exposure is 0.000068 mg/kg bwt/day, for the total U.S. population (or 1.7% of the RfD). When bananas and coffee beans are included in the assessment, the estimated exposure is 0.000083 mg/kg bwt/day for the total U.S. population (or 2.0% of the RfD).

The acute exposure estimates clearly indicate that exposures provide adequate MOEs at the 95th percentile of exposure. The U.S. population has an estimated 95th percentile exposure value of 0.000246 mg/kg bwt/day, equivalent to an MOE of 16,000 for tomatoes, potatoes and peanuts. When bananas and coffee are included in the assessment, the estimated 95th percentile exposure for the total U.S. population is 0.000279 mg/kg bwt/day, which results in an MOE of 14,000. These values are more than 2 orders of magnitude higher than a level considered to provide adequate protection. The exposure estimate for fosthiazate when highest field trial residue is used is 0.000187 mg/kg bwt/day, representing an MOE of 21,000, including all crops. Therefore, since there are no other avenues of exposure (see aggregate exposure section of this document) ISK Biosciences Corporation concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fosthiazate residues from use on tomatoes, potatoes, peanuts, bananas and coffee.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of fosthiazate, data from developmental toxicity studies and other appropriate studies are considered. ISK Biosciences Corporation calculates that children 1–6 (the highest exposed subgroup) have an estimated chronic dietary exposure of 0.000132 mg/kg bwt/day, which represents only 3.2% of the RfD using worst case assumptions. When bananas and coffee beans are included, these estimates are 0.000173 mg/kg bwt/day and 4.2% of the RfD for children 1–6. When anticipated residues are used in calculating chronic dietary exposure, only 1.1% of the RfD is consumed for this population subgroup and 1.3% of the RfD after bananas and coffee are included in the assessment. Acute exposure estimates similarly show no concern as all infants 1 year of age (the highest exposed subgroup) have MOEs of 8,300 even when using worst case assumptions. When bananas and coffee are included in the assessment, children 1–6 years (the highest exposed subgroup) have an MOE of 6,800. Therefore, since there are no other avenues of exposure other than dietary, there is reasonable certainty that no harm will result to infants and children from aggregate exposure to fosthiazate from use on tomatoes, potatoes, peanuts, bananas and coffee.

F. International Tolerances

There are no Codex maximum residue levels established for residues of fosthiazate.

[FR Doc. 01–28739 Filed 11–20–01; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–1044; FRL–6802–2]

Notice of Filing of Pesticide Petitions 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 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–1044, must be received on or before December 21, 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–1044 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: Driss Benmhend, Biopesticides and Pollution Prevention Division (7511C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–9525; e-mail address: Benmhend.driss@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