These results indicate that dietary exposure to pyraclostrobin (registered and all proposed crop uses), from potential residues in food and water, will not exceed EPA's level of concern (100% of aPAD or cPAD). Overall, we can conclude with reasonable certainty that no harm will occur from either acute or chronic dietary exposure to pyraclostrobin residues.

2. *Non-dietary exposure*. Pyraclostrobin is currently registered for use on golf course turf. The Agency has evaluated the existing toxicological database for pyraclostrobin and has assessed the appropriate toxicological endpoints and the dose levels of concern for this use. Dermal absorption data indicate that absorption is 14%.

D. Cumulative Effects

Section 408(b)(2)(D)(v) of the FFDCA 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.'' Pyraclostrobin is a foliar fungicide which belongs to the new class of strobilurin chemistry. It is a synthetic analog of strobilurin A, a naturally occurring antifungal metabolite of the mushroom Strobillurus tenacellus. The active ingredient acts in the fungal cell through inhibition of electron transport in the mitochondrial respiratory chain at the position of the cytochrome-bc1 complex. The protective effect is due to the resultant death of the fungal cells by disorganization of the fungal membrane system. Pyraclostrobin also acts curatively to prevent the increase and spread of fungal infections by inhibiting mycelial growth and sporulation on the leaf surface. BAS 500 F inhibits spore germination, germ tube growth, and penetration into the host tissues.

EPA is currently developing methodology to perform cumulative risk assessments. At this time, there are no available data to determine whether BAS 500 F has a common mechanism of toxicity with other substances or to show 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, pyraclostrobin does not appear to produce a toxic metabolite that is also produced by other pesticides.

E. Safety Determination

1. *U.S. population*. Adding the proposed uses to those crops that are already on the pyraclostrobin label resulted in aggregate exposure of adults in the U.S. population to pyraclostrobin that utilized at most 67% of the aPAD and 40% of the cPAD. Therefore, no harm to the overall U.S. population would result from the use of pyraclostrobin on the proposed and existing crop uses.

2. *Infants and children*. All subpopulations based on age were considered. The highest potential exposure was predicted for the subgroup children (1–6 years old). Using the FQPA Safety Factor of 3X when appropriate, the addition of the proposed crops to those on the label would use less than 1% of the aPAD and 89% of the cPAD for children (1–6 years old). BASF therefore concludes that there is reasonable certainty that no harm will result to infants or children from aggregate exposure to pyraclostrobin residues on the proposed and existing crop uses.

F. International Tolerances

Maximum Residue Levels (MRLs) have been established for pyraclostrobin in Canada but no MRLs have been established by the Codex Alimentarius Commission.

[FR Doc. 04–19627 Filed 8–26–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0271; FRL–7676–7]

Iodine-potassium iodide; 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 identification (ID) number OPP–2004–0271, must be received on or before September 27, 2004.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Mary Waller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–9354; e-mail address: *waller.mary@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

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 this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any 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 Copies of this Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket identification (ID) number OPP–2004–0271. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access*. You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*.

An electronic version of the public docket is available through EPA's

electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and to Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically*. If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket/*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2004–0271. The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail*. Comments may be sent by e-mail to *opp-docket@epa.gov*, Attention: Docket ID number OPP–2004–0271. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

iii. *Disk or CD ROM*. You may submit comments on a disk or CD ROM that you mail to the mailing address identified in Unit I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By mail*. Send your comments to: Public Information and Records Integrity Branch (PIRIB) (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID number OPP–2004–0271.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA, Attention: Docket ID number OPP–2004–0271. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

D. How Should I Submit CBI to the Agency?

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is 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

docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed 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 ID 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 FFDCA 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: August 20, 2004.

Betty Shackleford,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by the petitioner and represents the view of the petitioner. 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.

Ajay North America L.L.C.

PP 3E6572

EPA has received a pesticide petition (3E6572) from Ajay North America L.L.C., 1400 Industry Road, Powder Springs, Georgia 30127 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish an exemption from the requirement of a tolerance for iodine-potassium iodide in or on the raw agricultural commodities bananas, grapes, and melons. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The primary residue of iodine-potassium iodide (AJ1629) is the inorganic halide, iodide (I). In the presence of organic matrices such as food items, the iodine in the iodine-potassium iodide complex is very rapidly reduced to iodide (I), with a reaction rate on the order of seconds. Due to the natural occurrence in all fruits and vegetables of anti-oxidants, which are the likely agents in this reduction, there is little likelihood of iodine remaining intact in any crop matrix.

2. *Analytical method*. A residue method was developed which converts all iodine species present to the iodide ion, which is then quantitated (This methodology measures total iodine in the crop, similar to the method used in the Food and Drug Administration (FDA) Total Diet Study). Since iodine is naturally present in most foods, there is a natural background level of iodine that varies by crop and location. (Natural levels of iodine in the environment are higher in coastal areas due to the proximity to oceanic sources of the element).

3. *Magnitude of residues*. The results of the Chilean and Costa Rican non-GLP residue studies demonstrate that the average residues found in the soil-treated grapes and melons and foliar applied banana crops are virtually identical to the residues found in the control samples. In the Chilean grape trials AJ1629 34EC was applied to the soil at a variety of rates. The average residue for treated Thompson grapes was 0.39 parts per million (ppm) compared to 0.40 ppm for untreated Thompson grapes. The average residue for treated Red Globe grapes was 0.21 ppm, compared to 0.22 ppm for untreated Red Globe grapes. AJ1629 EC was also used in a foliar application to bananas in Costa Rica. Iodine residues in the whole treated bananas ranged from non-detectable to 0.11 ppm, in comparison to the untreated bananas with a range of residues from non-detectable to 0.13 ppm. Residues were below the limit of quantitation (0.05 ppm) in both treated and control banana pulp samples. In Costa Rican melon samples, iodide was not detected in either control or AJ1629 treated samples.

B. Toxicological Profile

Iodine is an essential element necessary to maintain human health and normal function of the thyroid gland. The effects of both iodine deficiency and excessive iodine intake in humans are well known and have been documented in a robust body of scientific literature. The levels of iodine intake considered optimal for various populations are based on Recommended Daily Allowances (RDAs) established by various Agencies. The proposed uses of iodine for disease and nematode control on grapes and melons, and for disease control on banana plants would not result in significant changes in iodine intake for any populations or subpopulations. Thus, the proposed use will not have any impact on individuals or subpopulations that are currently at risk for either deficiencies or excesses in iodine intake.

There is universal agreement on the dangers of iodine deficiency. Iodine deficiency is well known to result in a range of disorders including hypothyroidism, goiter, reproductive impairment and developmental abnormalities. While it is clear that excess iodine intake can cause adverse health effects, there are differences of opinion in the scientific community

with regard to the dangers of iodine excess. Health professionals consider the risk of iodine deficiency to be a more serious concern than the risk of excess dietary iodine.

A number of organizations, including Agency for Toxic Substances and Disease Registry (ATSDR), International Programme on Chemical Safety (IPCS), National Academy of Sciences, and the Joint Food and Agriculture Organization/World Health Organization (FAO/WHO) Expert Committee on Food Additives, have reviewed the literature regarding iodine and detailed its effects on human health. The toxicology information provided below has been assembled primarily from these sources.

Iodine has been approved by FDA for use in drugs and has been deemed GRAS as a food additive. There are also a number of antimicrobial uses already approved by the EPA for iodine and iodophors including sanitization of food handling equipment. A cleared review dated February 26, 1993 (Case No.3080, Toxicology Branch, Phase 4 Review, Pat McLaughlin) indicated that iodine and potassium iodide are well known in the literature, and while iodine is very irritating, it is not very toxic internally. EPA accepted the interchangeability of iodine and potassium iodide (EPA Cleared review dated July 5, 1996, Iodine Greybeard submission No. S496959, Toxicology Branch, Sanjivani Diwan). Potassium iodide is not toxic and has been deemed GRAS by the FDA as a food additive.

In an FDA Assessment entitled ''Potassium Iodide and Potassium and Calcium Iodates: Proposed Affirmation of GRAS Status as Direct Human Food Ingredients with Specific Limitations'' (42 FR 29925, June 10, 1977), the opinion of the Select Committee was that ingested potassium iodine and other iodides are readily absorbed and utilized to the extent required for nutritional needs, the excess being excreted primarily in the urine. There was no evidence in the studies on experimental animals and man available to the Committee that indicated acute or chronic toxic effects, including mutagenic, teratogenic, and carcinogenic effects, resulting from the consumption of potassium iodide by euthyroid individuals in amounts that are several orders of magnitude greater than those now being consumed in the daily diet.

In the 2002 National Academy of Sciences Report, the RDA of iodine for adult men and women is 0.15 mg/day (150 µg/day) and the Tolerable Upper Intake Level for adults is 1.1 mg/day. Intake varies considerably from day to day and the RDAs are based on average intakes. The RDA for children 1-8 years old is 0.09 milligram/day (mg/day) (90 µg/day). The Tolerable Upper Intake Level is 0.2 mg/day for children 1-3 years old, 0.3 mg/day for children 4-8 years old and 0.6 mg/day for children 9-13. The Tolerable Upper Intake Level is 0.9 mg/day for adolescents 14-18 years of age.

The Joint FAO/WHO Expert Committee on Food Additives assessed the human data on iodine for the purpose of establishing a maximum tolerated daily intake [661. Iodine (WHO Food Additives Series 24)]. The assessment concluded that iodine is an essential dietary element which is required for synthesis of thyroid hormones. The Committee concluded that the human response to excess iodine is variable and that the maximum tolerable level of iodine appears to be in the range from somewhat above recommended dietary allowances to one (1.0) mg/day. As indicated by Pennington (Pennington, J.A.T. (1990): A review of iodine toxicity reports. J. Am. Diet Assoc. 90: 1571-1581.), most people are very tolerant of excess iodine intake from food. Subpopulations with autoimmune thyroid disease and iodine deficiency respond adversely to intake of iodine, which would be considered safe for the general population.

The principal sources of iodine in the diet include, milk and dairy products, seafood, iodized salt and infant formula. The mean intake of iodine from food in the United States is 0.29 to 0.41 mg/day for females and males age 19 and above. For children 1-3 years old the mean intake is 0.3 mg/day, for children 4-8 years old the mean intake is 0.38 mg/day and for children 9-13 years old the mean intake is 0.38 to 0.49 mg/day. For adolescents 14-18 years of age, the mean intake is 0.33 to 0.53 mg/day. While this level of intake is considered adequate for the general population, the results of the National Health and Nutrition Examination Surveys I and III (NHANES I and III), conducted during the periods 1971–1974 and 1988–1994, respectively, show that the median iodine concentration in the population decreased more than 50% between the two surveys.

Residue trials of AJ1629 using maximum application rates on soil-treated grapes and melons and foliar applied bananas show that iodine residue levels in the food commodities are comparable for control and treated crops. Therefore, the proposed uses of iodine for disease and nematode control on grapes and melons, and for disease control on banana plants would not result in significant changes in iodine intake for any populations or subpopulations.

The toxicology data described below has been developed from human studies rather than animal studies. Therefore, an uncertainty factor is not needed to account for interspecies variability. The acute Minimal Risk Level (MRL) for iodine has been set at 0.01 milligram/kilogram/day (mg/kg/day) based on a NOAEL of 0.024 mg/kg/day for healthy adult humans. The chronic MRL is 0.01 mg/kg/day based on a no observed adverse effect level (NOAEL) of 0.01 mg/kg/day total iodide intake and a lowest observed effect level (NOAEL) of 0.029 mg/kg/day for subclinical hypothyroidism in healthy human children. An uncertainty factor is not needed to account for variability in sensitivity within species because the NOAEL is based on children, a sensitive subpopulation.

1. *Acute toxicity*. Acute oral: 315 mg/kg (Category II); acute dermal: >3,000 mg/kg Category III); acute inhalation: 0.363 mg/L (Category II); dermal irritation: corrosive (Category I); eye irritation: waived based on dermal irritation; dermal sensitization: not a sensitizer.

2. *Genotoxicty*. Iodine has been examined in a number of studies and it has not been found to be mutagenic in a variety of eukaryotic cell systems. The ATSDR draft report indicates that, ''potassium iodide, I2, and povidone iodine (0.1–10 mg/mL) did not show mutagenic effects in L5178Y mouse lymphoma cells or in transforming activity in Balb/c 3T3 cells grown in culture.'' Additionally, potassium iodide and I2 were negative for mutagenicity in drosophila melanogaster and I2 was negative in His+ revertant assay in saccharomyces cerevisiae. Iodide is a free-radical scavenger and has been shown to decrease hydrogen peroxide-induced reversion in strain TA104 of *Salmonella* typhimurium.

3. *Reproductive and developmental toxicity*—i. *Reproductive.* According to the ATSDR draft report on iodine toxicology, excessive iodine intake may result in hypothyroidism or hyperthyroidism. The effects of excess iodine on reproductive function are secondary to thyroid gland dysfunction.

To counteract the negative effects of iodine deficiency during pregnancy, the RDA for iodine in pregnant women is higher than that for the general population. In addition, the authors of the comparative iodine nutrition surveys, NHANES I (1971–1974) and III (1988–1994), voiced concern about the increased proportion of women of child-bearing age and pregnant women who

are in the iodine deficiency range. This trend is particularly important because iodine deficiency disorders include goiter, hypothyroidism, mental retardation, reproductive impairment, cretinism, decreased child survival and varying degrees of other growth and developmental abnormalities. The most damaging effect of iodine deficiency is on the developing brain. If severe enough to affect thyroid hormone synthesis during fetal and postnatal life, iodine deficiency will result in hypothyroidism and brain damage. Such iodine deficiency, therefore, can lead to irreversible intellectual deficits with great impact on populations. Correction of iodine deficiency dramatically decreases the prevalence of these disorders. In the U.S., it is an FDA requirement that infant formula must contain supplemental iodine.

ii. *Developmental.* While there are developmental effects related to excessive iodine consumption, ATSDR indicated in their draft toxicological profile for iodine, that ''iodine deficiency is far more likely to cause prenatal and postnatal hypothyroidism and be associated with neurologic injury leading to cretinism, a developmental effect.'' Other effects that might be caused by iodine deficiency during development include severe mental retardation, neurologic abnormalities, growth retardation, or abnormal pubertal development. Iodine deficiency during pregnancy, infancy, or early childhood also may cause endemic cretinism. The symptoms of cretinism are mental and physical retardation, deaf-mutism, and various neurological abnormalities. Hypothyroidism due to iodine deficiency may be cured with iodine administration, but the effects of cretinism are not reversible. The effects of iodine deficiency on development far outweigh the effects of excess iodine intake.

4. *Subchronic toxicity.* Dietary iodine deficiency stimulates TSH secretion which results in thyroid hypertrophy. The enlargement of the thyroid gland due to iodine deficiency is called endemic goiter. Iodine intakes consistently lower than 0.050 mg/day usually result in goiter. Women and adolescent girls seem especially at risk. Most goiterous individuals are clinically euthyroid. Large goiters may cause obstructive complications of the trachea, esophagus, and blood vessels of the neck. Goiters also are associated with an increased risk of other thyroid diseases and malignant growth.

Hypothyroidism is the primary effect of subchronic exposures to excess iodine. Below are summaries of several human studies conducted with iodine over 14 to 28 day periods. The ATSDR draft report has set the MRL for acute-duration oral exposure (1-14 days) at 0.01 mg/kg/day.

Several subchronic studies on humans were reviewed by ATSDR including one conducted on 18 healthy male and female adults over 14 days at daily oral doses of 1.5 mg I/day as sodium iodide with a background intake of 0.2 mg/day. The total iodide intake was approximately 1.7 mg I/day (approximately 0.024 mg/kg/day). There were significantly depressed serum concentrations of TT4, FT4 and TT3 and significantly elevated serum TSH concentrations compared with pretreatment levels. Hormone levels were within the normal range and, therefore, the subjects were not clinically hypothyroid.

Another study was conducted on ten healthy male adults over a 14–day period using oral doses of 0, 0.5, 1.5 and 4.5 mg I/day as sodium iodide. Including a background iodide intake of between 0.25–0.32 mg/day, total intake was 0.3, 0.8, 1.8 and 4.8 mg/day. These levels are approximately equivalent to 0.004, 0.011, 0.026 and 0.069 mg/kg/day. Small but significant, transient decreases in serum TT4 and FT4 concentrations and an increase in serum TSH concentrations were seen at the 1.8 and 4.8 mg/day dose level relative to the pretreatment values. The magnitude of the changes at the higher iodide dosages yielded hormone concentrations that were within the normal range and, thus, would not represent clinically significant thyroid suppression.

A 14 to 28 day study was conducted on 30 elderly adult females given daily doses of 0.5 mg I/day, with a background exposure of about 0.072–0.1 mg/day, for a total iodide intake of 0.6 mg/day or 0.0086 mg/kg/day. There were significantly decreased serum concentrations of FT4 but, on average, the magnitude of the changes did not produce clinically significant depression in thyroid hormone levels. Five subjects had serum TSH concentrations typically considered at the high end of the normal range.

5. *Chronic toxicity.* Severe and prolonged iodine deficiency results in deficient supply of thyroid hormones. This condition, which is referred to as hypothyroidism or myxedema, is characterized by reduced metabolic rate, cold intolerance, weight gain, puffy facial features, edema, a hoarse voice, and mental sluggishness.

Chronic exposure to an excess of iodine primarily results in conditions such as hypothyroidism, hyperthyroidism, and/or thyroid autoimmunity. A NOAEL of 0.01 mg/kg/day and a LOAEL of 0.029 mg/kg/day were established for subclinical hypothyroidism in healthy human children. This NOAEL was used to set the MRL of 0.01 mg/kg/day for humans. The NOAEL was not adjusted for sensitivity, because it was based upon a sensitive subpopulation, children. The LOAEL based upon slight thyroid enlargement is not indicative of functional impairment.

Studies have shown that the chronic MRL for children, adults and the elderly is the same.

6. *Animal metabolism.* The above proposed iodine potassium- iodide uses do not include any animal feed items; therefore, animal metabolism data are not necessary.

7. *Metabolite toxicology.* There are no metabolites of toxicological concern.

8. *Endocrine disruption.* The thyroid effects normally associated with iodine result from either a deficiency or from excessive iodine intake as noted in the toxicology discussion above. The principal direct effects of excessive iodine ingestion on the thyroid gland are hypothyroidism, hyperthyroidism and thyroiditis. Most iodine-induced hypothyroidism is transient. Epidemiological and clinical literature suggests that hyperthyroidism occurs most often in people who have a previous history of iodine deficiency, goiter or thyroid disease. Thyroiditis is an inflammation of the thyroid gland which is often secondary to thyroid gland autoimmunity. The proposed use of iodine will not add to overall intake or exposure, and therefore will not increase risk of endocrine disruption.

9. *Additional information.*. Iodine deficiency disorders include goiter, hypothyroidism, mental retardation, reproductive impairment, cretinism, decreased child survival and varying degrees of other growth and developmental abnormalities. The most damaging effect of iodine deficiency is on the developing brain. If severe enough to affect thyroid hormone synthesis during fetal and postnatal life, iodine deficiency will result in hypothyroidism and brain damage. Correction of iodine deficiency dramatically decreases the prevalence of these disorders. Thus, in the United States, infant formula must contain 0.005 to 0.075 mg (5 to 75 µg) of iodine per 100 kilocalories.

As indicated by Pennington (1990), most people are very tolerant of excess iodine intake from food. Subpopulations with autoimmune thyroid disease and iodine deficiency respond adversely to intake of iodine, which would be considered safe for the general population. The principal effects of

excess iodine intake for the general population are thyroiditis, goiter, hypothyroidism, hyperthyroidism, sensitivity reactions, thyroid papillary cancer, and acute responses in some individuals. There may be other unrecognized sources (i.e., in addition to food, water, and supplements) of iodine that increase the risk of adverse effects. Available evidence clearly corroborates that the adverse effects of iodine deficiency far outweigh the risks associated with the ingestion of excess iodine.

C. Aggregate Exposure

1. *Dietary exposure.* An exemption from the requirement of tolerance is proposed for iodine-potassium iodide on bananas, grapes, and melons. For purposes of assessing the potential dietary exposure to iodine, a review of the open literature has been conducted.

i. *Food.* Nearly every food (raw agricultural commodity or processed/prepared food item) contains measurable amounts of iodine. As discussed in the Residue Chemistry, Magnitude of the Residues section (A.3.), residue studies conducted to date for AJ1629 demonstrated that residues from AJ1629 in the soil-treated grapes and melons and foliar applied banana crops are virtually identical to the residues found in the control samples. Therefore, exposure to iodine through dietary intake is not expected to increase due to the use of AJ1629.

ii. *Drinking water.* An exposure assessment for drinking water is not necessary due to the proposed use pattern of iodine-potassium iodide.

2. *Non-dietary exposure.* Iodine is widely used in disinfectants, germicides, and related products. These products are readily available and have been widely used for many years. A non-dietary exposure assessment is not necessary due to the proposed use pattern of iodine-potassium iodide.

D. Cumulative Effects

To our knowledge there are currently no available data or other reliable information indicating that any toxic effects produced by iodine would be cumulative with those of other chemical compounds; thus only the potential risks of iodine have been considered in this assessment of its aggregate exposure.

E. Safety Determination

1. *U.S. population.* Iodine is a naturally occurring element, present in air, soil, water and food at levels that vary, depending on geographic location. It is ubiquitous and is found in all non-treated crops in varying amounts. Residue studies with crops from AJ1629 trials have shown that the average residues of iodine (as iodide) in treated crops are indistinguishable from residues in untreated crops. Since the dietary intake of iodine is not expected to increase because of the proposed uses of AJ1629, there is a reasonable certainty that no harm will result from its use.

2. *Infants and children.* As noted above, iodine is a naturally occurring element that infants and children will be exposed to through a variety of sources including water and food. In the U.S. iodine is a mandated nutrient in baby formula, required to be present at levels of 5 to 75 micrograms/100 kilocalories of formula. Residues from the use of AJ1629 are virtually indistinguishable from residues in untreated crops, therefore, exposure from pesticidal use will be very minimal.

F. International Tolerances

There are no known international tolerances for residues of iodine-potassium iodide in food or animal feed.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0270]; FRL–7675–2]

Fenhexamid; 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 identification (ID) number OPP–2004–0270, must be received on or before September 27, 2004.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Sidney Jackson, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–7610; e-mail address: *jackson.sidney@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532).

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. Other types of entities not listed in this unit could also be affected. If you have any 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 Copies of this Document and Other Related Information?

1. *Docket.* EPA has established an official public docket for this action under docket ID number OPP–2004–0270. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access.* You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*.

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/*