Research Strategy clearly identifies the appropriate strategic directions for a core research program that will develop the methods, models, and measurements to strengthen the scientific foundation for assessing the potential consequences of global change on human health, ecosystems, and social well-being in the United States.

The emphasis of the Program's research and assessment strategy is on understanding the risks and opportunities presented by global change, the interdependent and interactive effects of multiple stresses, the human dimensions of global change (*i.e.*, human activities that catalyze, as well as those that respond to global change), and adaptation options.

After considering recommendations from extramural advisory groups, as well as from senior scientists from across EPA's Program and Regional Offices, ORD has identified, in the Research Strategy, the strategic directions for its Global Change Research Program. While the Research Strategy delineates the research areas comprising the framework for the Global Change Research Program, the details of the research areas, including the scientific approach at the individual project level, and the anticipated products, performance measures, and schedules, will be included in subsequent research plans and are not a part of this Research Strategy. ERG is undertaking the establishment of a peer review panel to review the Research Strategy.

Dated: January 17, 2001.

William H. Farland,

Acting Deputy Assistant Administrator for Science, Office of Research and Development.

[FR Doc. 01–2178 Filed 1–23–01; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–993; FRL–6758–9]

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–993, must be received on or before [*insert date 30 days after date of publication in the* **Federal Register**].

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

FOR FURTHER INFORMATION CONTACT: By mail: Joanne I. Miller, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–6224; e-mail address: miller.joanne@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:

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–993. 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–993 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–993. 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: January 12, 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 petitioners. 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.

Aventis CropScience

F6160

EPA has received a pesticide petition (F6160) from Aventis CropScience, P.O. Box 12014, 2 T.W. Alexander Drive, Research Triangle Park, NC 27709 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 Iodosulfuron-methyl-sodium, methyl 4-iodo-2-[3-(4-methoxy-6-methyl-1,3,5-triazin-2-yl)-ureidosulfonyl]benzoate, sodium salt in or on the raw agricultural commodityies corn grain at 0.05 parts per million (ppm), corn forage and stover at 0.1 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of iodosulfuron-methyl-sodium (methyl 4-iodo-2-[3-(4-methoxy-6- methyl-1,3,5-triazin-2-yl)-ureidosulfonyl]benzoate, sodium salt) in wheat, as representative of the cereals grain crop grouping has been investigated and is understood. The results of two metabolism studies in wheat show that the total radioactive residue levels in wheat commodities were very low. The principal compound was the parent, iodosulfuron-methyl-sodium. The metabolism in wheat proceeded via hydrolysis of iodosulfuron-methyl-sodium to three metabolites, AE F0031838 (2-amino-4-hydroxy-6-hydroxymethyl-1,3,5-triazine), AE F075736 (methyl-2-[3-(4-methyoxy-6-methyl-1,3,5-triazin-2-yl)ureidosulfonyl]benzoate), and AE F145741(methyl 2-[3-(4-hydroxy-6-methyl-1,3,5-triazin-2-yl)ureidosulfonyl]-4-iodo-benzoate) in harvested straw and at extremely low levels in grain. A fourth metabolite, AE F059411(2-amino-4-methoxy-6-methyl-1,3,5-triazine) was only detected in the straw, again at very low levels. All metabolites characterized in plants were also found in the animal metabolism studies.

2. *Analytical method*. Based on the results of the metabolism studies, the analytical targets selected were only the parent compound, iodosulfuron-methyl-sodium and AE F075736, based on its potential toxicological significance. Extractable residues of iodosulfuron-methyl-sodium and AE F075736 are extracted from the crop matrices corn grain, forage and stover by blending with acetonitrile. After blending, the extract is filtered, volume reduced, partitioned, evaporated to dryness, dissolved in dichloromethane and cleaned-up. The organic extract is rotary evaporated to dryness and analyzed by HPLC/UV. The limit of quantification (LOQ) is 0.025 ppm in corn grain and 0.05 ppm in corn forage and stover.

3. *Magnitude of residues*. Residue trials were carried out in a total of 21 U.S. residue field trials using a water dispersible granule (WG) formulation containing 20 percent w/w iodosulfuron-methyl-sodium. The preparation was applied in a split application of 5 g/ha followed by 2.5 g/ha. Pre-harvest intervals were between 37 to 53, 58 to 102 and 58 to 125 days for forage, grain and stover, respectively. Grain, stover and forage of field corn did not contain residues of iodosulfuron-methyl-sodium at or above the respective limits of quantification of 0.025, 0.05 and 0.05. Also no residues of the metabolite AE F075736 were found in corn grain, stover or forage at harvest above the respective limits of quantification of 0.025, 0.05 and 0.05 mg/kg. It is proposed, therefore, that AE

F075736 is not included in the tolerance expression. Tolerances of iodosulfuron-methyl-sodium are proposed at twice the limit of quantification of the analytical method, namely 0.05, 0.1 and 0.1 mg/kg in grain, stover and forage, respectively. In a corn processing study, no residues above 0.025 mg/kg were observed in corn grain following treatment of the crop at the nominal rate of 25 followed by 12.5 g/ha. This exaggerated rate is approximately eighteen times the maximum proposed label rate. Since no residues were observed in the raw agricultural commodity, neither analysis of the processed commodities nor tolerances are required. Although corn grain is fed to cattle and poultry and cattle may be grazed on forage or fed stover, tolerances in meat, milk or eggs are not necessary because none of these commodities contained iodosulfuron-methyl-sodium or its metabolite.

B. Toxicological Profile

1. *Acute toxicity*. Iodosulfuron-methyl-sodium is slightly toxic following acute oral exposure, no more than slightly toxic following acute dermal exposure and practically non-toxic following acute inhalation exposure. The acute rat oral LD_{50} of iodosulfuron-methyl-sodium was 2,678 mg/kg (combined males plus females). The acute rat dermal LD_{50} was greater than 2,000 mg/kg and the 4-hour rat inhalation LC_{50} was < 2.81 mg/l. Iodosulfuron-methyl-sodium was non-irritating to rabbit skin and caused corneal involvement or irritation clearing in 7 days or less. Based on these results, iodosulfuron-methyl-sodium would be classified as EPA Category IV for inhalation toxicity and dermal irritation and EPA Category III for eye irritation, dermal and oral toxicity. Technical iodosulfuron-methyl-sodium was not a sensitizer to skin.

2. *Genotoxicty*. Testing for possible genotoxic properties of the technical active substance of iodosulfuron-methyl-sodium in several *in vitro* and *in vivo* test systems on different endpoints gave consistently negative results. The *in vitro* testing battery was comprised of investigations for gene mutation in bacterial and mammalian cells, examination of chromosomal aberration in Chinese Hamster cells and testing for unscheduled DNA-synthesis (UDS) in primary rat hepatocytes. The test program was complemented by a mouse micronucleus assay as an indirect investigation on the end-point chromosomal aberration *in vivo*. As there was no evidence of genotoxicity, the overall weight of evidence indicates that iodosulfuron-methyl-sodium is not genotoxic.

3. *Reproductive and developmental toxicity*. A rat developmental toxicity (teratogenicity) study was conducted at dose levels of 0, 100, 315, and 1,000 mg/kg/day. No increased mortality was noted. High dose dams exhibited clinical signs of toxicity including increased salivation, some body weight effects and statistically significantly decreased food consumption. Treatment-related fetal effects were seen only at the high dose of 1,000 mg/kg body weight, expressed by slightly increased incidences of retarded skeletal ossification, blood in the abdominal cavity and distended kidney pelvis. The mid dose dams reduction in food consumption was marginal (7.9 mg/kg versus 8.1 mg/kg for controls). Therefore, the no observable adverse effect level (NOAEL) with respect to maternal and fetal toxicity was 315 mg/kg body weight.

A rabbit developmental (teratogenicity) toxicity study was conducted at dose levels of 0, 25, 100 and 400 mg/kg/day. No treatment related deaths or clinical signs were seen except reduced defecation at 100 and 400 mg/kg. At 400 mg/kg, reduced body weight gain were observed. Food consumption was decreased in all dose level groups. No compound related effects were noted during necropsy except one animal at 400 mg/kg which had white depression on the liver. Fetal weights, crown rump lengths, litter sizes, number of live fetuses and placental weights were not affected by administration of iodosulfuron-methyl-sodium. The NOEL was considered to be 25 mg/kg for maternal toxicity and 400 mg/kg for fetal toxicity. In a 2-generation rat reproduction study with iodosulfuron-methyl-sodium, dietary concentrations of 0, 50, 500, and 5,000 ppm were administered to Wistar male and female rats. Iodosulfuron- methyl-sodium did not cause adverse effects on reproduction, fertility, mating behavior in parents or malformations in the offspring at any dose level tested. Treatment-related changes in parental animals were limited to significant decreases in body weight gains for males and females. Depression of body weight gain was also seen during the gestation periods in females. Retarded body weight gain in pups at the high dose level of 5,000 ppm was seen during lactation. At 5,000 ppm, a slightly statistically significant increase in the number of supernumerary implantation sides was observed in F1 females only. Based on depression of body weight development in parental animals during all phases and on toxicity to the fetuses/offspring at 5,000 ppm, the NOEL for parental animals and offspring was determined to be 500 ppm (equivalent to daily test substance intakes of 25.6 to 116.8 mg/kg/ body weight depending on the phase of the study).

4. *Subchronic toxicity*. In a 90-day rat feeding study, iodosulfuron-methyl-sodium was administered at dietary concentrations of 0, 200, 1,000, 5,000 and 10,000 ppm to groups of 10 male and 10 female Sprague Dawley rats. Futher 10 males and 10 females fed either 0, 5,000 or 10,000 ppm were maintained on control diet for a further 4 weeks to examine reversibility of possible effects. Treatment related depression in body weight gains was seen in males and females at 10,000 ppm and at 5,000 ppm after 13 weeks. Depression of body weight gains was partly reversible during the 4-week recovery period. Overall food consumption was reduced in the 10,000 ppm males. No effects on food consumption were observed in the other dose level groups. Food consumption was comparable in all groups after the 4-week recovery period. Total red cell count and hemoglobin and hematocrit were slightly to marginally reduced in females at 10,000 ppm. No such changes could be seen in the respective recovery animals. Liver weight to body weight ratio was slightly increased in females at 1,0000 ppm compared to controls. This effect was no longer seen in recovery animals. Based on body weight effects at 10,000 and 5,000 ppm and the hepatocyte enlargement in males at 10,000 ppm, the NOEL was considered to be 10,000 ppm, equivalent to a daily intake of 71 mg/kg/day. In a 90-day feeding study in mice, iodosulfuron-methyl-sodium was administered at dietary concentrations of 0, 700, 2,100, and 7,000 ppm. There were no treatment related deaths or clinical signs. Terminal body weight was reduced and body weight gain in males at 7,000 ppm compared to controls. There were no treatment-related effects on food consumption or hematological evaluations. A treatment-related statistically significant increase in alkaline phosphatase was seen in males at 7,000 ppm. Treatment related effects on organ weights were observed in livers of males at 7,000 ppm and 2,100 ppm and in females at 7,000 ppm. Based on depression of body weight development in males and liver effects in both sexes at 7,000 ppm and liver effects in males at 2,100 ppm, the NOEL was considered to be 700 ppm, equivalent to daily intakes of 119 mg/kg body weight for males and 139 mg/kg body weight for females. In a 90-day dog

feeding study, iodosulfuron-methyl-sodium was administered to beagle dogs at dietary concentrations of 0, 200, 1,200 and 7,200 ppm. iodosulfuron-methyl-sodium at dietary concentrations of 7,200 ppm showed effects on the hemapoietic system, in particular on maturation of blood cells in the bone marrow for both sexes. Decreased body weight gain was also seen in the highest dose. At 7,200 ppm, increased absolute and relative liver weights for males and females were observed. Absolute kidney weights in males and relative kidney weights in males and females were increased. Absolute and relative spleen weights were increased in males at 7,200 ppm. All dogs, at 7,200 ppm, had a generalized hemapoietic hyperplasia. Extramedullary hemopoiesis was also detected in the spleen for males and females, in the liver for females and in the mediastinal lymph node for male dogs. At 1,200 ppm one of four females had generalized hemopoietic hyperplasia in the sternal medullary cavities with moderate extramedullary hemopoiesis in the spleen and a reduction in the mature granulocyte forms in the marrow smear. The dietary concentrations of 7,200 ppm clearly exceeded the maximum tolerated dose (MTD). Based on the findings in one female at 1,200 ppm, the NOEL was considered to be 200 ppm, equating to 8.1 mg/kg/day for males and 8.4 mg/kg/day for females.

5. *Chronic toxicity*. Testing was performed in Sprague-Dawley rats and CD-1 mice using dietary concentrations up to and including 7,000 ppm and 1,750 ppm respectively. In the combined chronic toxicity/carcinogenicity study an interim sacrifice in 10 animals per sex and group as an early check for possible effects was performed after 12 months. Doses of 331 mg/kg bw (males) or 452 mg/kg bw (females) in rats caused marked decreases of body weight gains and terminal body weights of high dose animals. Slight body weight effects were also seen in the mid dose of 29.7 mg/kg bw (m) or 39.1 mg/kg bw (famales). The NOAEL was equivalent to a dietary intake of 2.96 mg/kg bw (males) or 3.91 mg/kg bw (females). No body weight effects but hepatotoxicity was seen in mice in line with the results of the 90-day study. Liver effects in the form of pigment deposition were seen in most of the males and part of the females at the top dose of 1,750 ppm. With respect to the marked lipofuscin storage as observed in the 90-day study the high dose of the oncogenicity study had been selected at 1,750 ppm and pigment deposition was seen even at this lower dose due to the longer study duration. In addition hepatocyte enlargement and increased mononuclear cell infiltration was seen in both sexes at the high dose and also in males at the mid dose. There were no significant increases in neoplastic changes in rats or mice after administration of the mentioned doses for the animals natural lifespan. Based on the available chronic toxicity data, Aventis CropScience believes the Reference Dose (RfD) for iodosulfuron-methyl-sodium is 0.03 mg/kg/day based on the most sensitive species, rat. Iodosulfuron-methyl-sodium was not oncogenic in rats or mice and is not likely to be carcinogenic in humans. Aventis Crop Science believes, iodosulfuron-methyl-sodium should be classified as a '' Not Likely'' carcinogen based on the lack of carcinogenicity in rats and mice.

6. *Animal metabolism*. The absorption, distribution metabolism and excretion of, iodosulfuron-methyl-sodium is well understood mammals. Wistar rats were orally administered low doses of 10 mg/kg/ body weight and 500 mg/kg body weight. After specific toxic effects had become obvious in the dog, absorption, distribution, elimination and in particular metabolism were also examined in Beagle dogs using an oral low dose of 6 mg/kg bw which was close to the 90-day NOEL, as well as an oral high dose of 200 mg/kg bw. The influence of the label position was examined using two different labels (U-14C-phenyl and 2-14C-triazinyl-label), iodosulfuron-methyl-sodium was metabolized by hydrolysis of the methylester of the benzoic acid function to AE F145740 (4-iodo- 2-[3-(4-methoxy-6-methyl-1,3,5-triazin-2-yl)-ureidosulfonyl]benzoic acid and o-demethylation at the 1,3,5-traizine leading to AE F145741 after single and repeated dosing. Oxidative hydroxylation of the 6-methyl group of the 1,3,5-triazinyl moiety was also observed. Breakdown of the sulfonylurea bridge possibly due to amidases leads to AE F114368 (methyl 2-sulfamoylbenzoate) and AE 0031850 (2-aminosulfonyl-4-iodo-benzoic acid) which cyclised to AE F143133 (6-iodo-1,2-benzisothiazol-3(2H)-one-1,1-dioxide). The cleavage of the iodine-phenyl- bond resulting in AE F075736 and AE F161778 (methyl 2-[3-(4-hydroxy-6-methyl-1,3,5-triazin-2-yl)ureidosulfonyl]benzoate) was observed to be a minor metabolic reaction. Overall no significant difference in the metabolic profile between sexes, dose levels or following repeated dosing in the rat were found. Metabolites identified in the dog study were the same as those found in rats The metabolism of, iodosulfuron-methyl-sodium in ruminants is adequately understood. A dairy cow was dosed with the compound at a level equivalent to 14.23 ppm in the diet for 7 days. The compound appeared to be well absorbed and rapidly excreted mainly in the urine. Total residue levels were very low. The major metabolite identified in all tissues and milk was unchanged, iodosulfuron-methyl-sodium together with up to 7 minor metabolites. All of the metabolic products of iodosulfuron-methyl-sodium were also observed in the rat. The metabolism of iodosulfuron-methyl-sodium in poultry is also adequately understood. Laying hens were fed the compound at a level equivalent to 10 ppm in the diet for 14 days. Residue levels were low in all commodities. Unchanged iodosulfuron-methyl-sodium was the major metabolite identified in all of the tissues and yolks. Up to 6 minor metabolites of iodosulfuron-methyl-sodium were also detected in all tissues and excreta which were identical to those formed in the rat.

7. *Endocrine disruption*. No special studies have been conducted to investigate the potential of iodosulfuron- methyl-sodium to induce estrogenic or other endocrine effects. However, no evidence of estrogenic or other endocrine effects have been noted in any of the standard toxicology studies that have been conducted with this product and there is no reason to suspect that any such effects would be likely.

C. Aggregate Exposure

1. *Dietary exposure*. Iodosulfuron-methyl-sodium is proposed for use as an herbicide on corn. No non- agricultural uses are anticipated. The potential sources of exposure would consist of any potential residues in food and drinking water. As indicated in Unit B. there are no acute toxicity concerns and thus only chronic exposure has been evaluated.

i. *Food*. Chronic dietary analysis was conducted to estimate exposure to potential iodosulfuron- methyl-sodium residues in/on corn. A Tier One analysis was conducted using the DEEM software and the 1994-1996 CSFII food consumption data. It was assumed that residues were at tolerance levels of 0.05 ppm (twice the limit of quantification) in grain and that 100% of crop was treated. Additionally, based on the results from appropriate studies, it was assumed that there was no concentration into processed commodities and that contributions

from residues in meat, milk or eggs are not required. A chronic RfD of 0.03 mg/kg /day is derived from the most sensitive species, rat. Using these inputs the chronic dietary exposure estimate from residues of iodosulfuron-methyl-sodium for the U.S. population was 0.000079 mg/kg /day or 0.3% of its RfD. For the sub-population with the highest exposure, non-nursing infants, the chronic dietary exposure estimate from residues of iodosulfuron-methyl-sodium was 0.000201 mg/kg /day, or 0.7% of its RfD. These values are highly conservative, having been based on worst case assumptions of tolerance level residues and 100% of the crop treated.

ii. *Drinking water.* EPA's Standard Operating Procedure (SOP) for drinking water exposure and risk assessments was used to perform the drinking water assessment. This SOP uses a variety of tools to conduct drinking water assessment. These tools include water models such as SCI-GROW, GENEEC, PRZMS/EXAMS, and monitoring data. If monitoring data are not available then the models are used to predict potential residues in surface and ground water and the highest value is assumed to be the potential drinking water residue. In the case of iodosulfuron-methyl-sodium monitoring data do not exist therefore model calculations were used to estimate a water residue. The calculated drinking water levels of comparison (DWLOC) for chronic exposures for all adults and children greatly exceed the drinking water estimated concentrations (DWEC) from the models. The chronic DWLOC for adults is 1,047 ppb. The chronic DWLOC for children/toddlers is 298 ppb. The worst case chronic DWEC is 0.015 ppb based on a PRZM/EXAMS simulation of runoff into surface water in a standard EPA exposure assessment scenario for corn (MLRA 111, Ohio). The DWEC represents combined residues of iodosulfuron-methyl-sodium and its metabolite AE F075736, expressed as iodosulfuron-methyl-sodium equivalents.

2. *Non-dietary exposure.* Exposure to iodosulfuron-methyl-sodium for the mixer/loader/ground boom/aerial applicator was calculated using the Pesticide Handlers Exposure Database (PHED). It was assumed that the product would be applied to a maximum of 50 hectares per day (125 A/day) by ground boom applicator and 140 hectares per day (350 A/day) by aerial applicator at a maximum use rate of 2 grams active ingredient. Normal work attire consisting of long-sleeved shirt, long pants, and protective gloves was assumed in the PHED assessment. Margins of exposure (MOEs) for a 70 kg operator were calculated utilizing a dermal NOEL of 810 mg/kg body weight/day from the rat dermal toxicity study and an inhalation NOAEL of 8 mg/kg body weight/day based on a 90–day dog feeding study. There were no signs of developmental toxicity in the rabbit developmental toxicity study. The combined MOE (inhalation plus dermal) for iodosulfuron-methyl-sodium was 1,101,000 for a ground operator undertaking mixing, loading and spraying. For aerial application where the mixer/loader was assumed to be a different operator from the pilot combined MOEs were 629,000 for the mixer/loader and 10,131,000 for the pilot. The results indicate that large margins of safety exist for the proposed use of iodosulfuron-methyl-sodium. The timing of iodosulfuron-methyl-sodium application to corn is such that field reentry shortly after spraying is atypical. Therefore estimations of worker reentry exposure were not considered necessary.

D. Cumulative Effects

There is no available data at this time to determine whether iodosulfuron-methyl-sodium has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Therefore a cumulative assessment was not done for this chemical.

E. Safety Determination

1. *U.S. population.* Using the conservative assumptions described above, based on the completeness and reliability of the toxicity data, it is concluded that aggregate exposure, in this case food only, to the proposed uses of iodosulfuron-methyl-sodium will utilize at most 0.3% of the reference dose for the U.S. population. The actual exposure is likely to be much less as more realistic data and models are developed. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risk to human health. Drinking water levels of comparison based on the dietary exposure are much greater than highly conservative estimated levels, and would be expected to be well below the 100% level of the RfD, if they occur at all. Therefore, there is a reasonable certainty that no harm will occur to the U.S. Population from aggregate exposure (food and drinking water) to iodosulfuron-methyl-sodium.

2. *Infants and children.* No evidence of increased sensitivity to fetuses was noted in developmental toxicity studies in rats or rabbits. There has been no indication of reproductive effects or indication of increased sensitivity to the offspring in the 2-generation rat reproduction study. No additional safety factor to protect infants and children is necessary as there is no evidence of increased sensitivity in infants and children.

Using the conservative assumptions described in the exposure section above, the percent of the reference dose that will be used for exposure to residues of iodosulfuron-methyl-sodium in food for non-nursing infants (the most highly exposed sub group) is 0.7%. The children (1-6) exposure uses 0.6% of the reference dose. As in the adult situation, drinking water levels of comparison are much higher than the worst case drinking water estimated concentrations and are expected to use well below 100% of the reference dose, if they occur at all. Therefore, there is a reasonable certainty that no harm will occur to infants and children from aggregate exposure to residues of iodosulfuron-methyl-sodium.

F. International Tolerances

There are no Codex Alimentarius Commission maximum residue levels established for residues of iodosulfuron-methyl-sodium.

[FR Doc. 01–2182 Filed 1–23–01; 8:45 am]
BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–00690; FRL–6758–6]

Pesticide Guidelines; Request for Information to Update Plant Commodity Table

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA intends to update its guidance on the residue data requirements that support registration of pesticides under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and that support tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA) for use in the conduct of human health risk assessments. The Agency will update the Series 860—Residue Chemistry Test Guidelines by revising Table 1 in OPPTS 860.1000, describing raw agricultural commodities (RACs), processed foods, and livestock feedstuffs because of changes in commercial food/feed processing practices, livestock feeding practices, and consumer consumption patterns. The Agency seeks information from