Agency has considered the nature of the chemical and its pattern of use, application methods and rates, and level and extent of potential exposure. Based on these reviews, the Agency was able to make basic health and safety determinations which show that use of Cry1Ac and Cry2Ab stacked proteins when used in accordance with widespread and commonly recognized practice, will not generally cause unreasonable adverse effects to the environment.

III. Approved Applications

EPA issued a notice, published in the **Federal Register** of March 21, 2001 (66 FR 15867) (FRL–6770–6), which announced that Monsanto Company, 700 Chesterfield Parkway North, St. Louis, MO 63198, had submitted an application to register the pesticide product, Bollgard II Cotton (EPA File Symbol 524–LEE) containing the active *Bacillus thuringiensis* Cry2Ab protein and the genetic material necessary for its production (Vector GHBK11L) in cotton. Monsanto transformed a Bollgard cotton variety with vector GHBK11L using particle bombardment to add the Cry2Ab gene for full commercial registration on cotton. This product was not previously registered.

The application was approved on June 14, 2002, as Bollgard II, Plant-incorporated protectant containing Cry1Ac and Cry2Ab stacked proteins (EPA Registration Number 524–522) for a 20,000 acre seed increase.

List of Subjects

Environmental protection, Chemicals, Pesticides and pests.

Dated: August 7, 2002.

Janet L. Andersen,

Director, Biopesticides and Pollution Prevention Division, Office of Pesticide Programs.

[FR Doc. 02–20873 Filed 8–20–02; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2002–0208; FRL–7195–2]

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 ID number OPP–2002–0208, must be received on or before September 20, 2002.

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 ID number OPP–2002–0208 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Joanne Miller, 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@epamail.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 ID number OPP–2002–0208. 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 ID number OPP–2002–0208 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 ID number OPP–2002–0208. 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 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 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.

Dated: August 12, 2002.

Debra Edwards,

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.

Bayer Corporation

PP 0F6094

EPA has received a pesticide petition (0F6094) from Bayer Corporation, 8400 Hawthorn Road, Kansas City MO, 64120–0013, 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 tolerances for residues of propoxycarbazone-sodium, methyl 2-[[[[(4,5-dihydro-4-methyl-5-oxo-3-propoxy-1H-1,2,4-triazol-1-yl)carbonyl]amino]sulfonyl]benzoate, sodium salt and its metabolite, methyl 2-[[[[(4,5-dihydro-4-methyl-5-oxo-3-(2'-hydroxy-propoxy)-1H-1,2,4-triazol-1-yl)carbonyl]amino]sulfonyl]benzoate in or on the raw agricultural commodities (RACs) wheat forage, wheat hay, wheat straw, wheat grain, meat, and meat byproducts, (cattle, sheep, goats, horses, hogs), and milk at 1.5, 0.15, 0.05, 0.01, 0.05, and 0.002 parts per million (ppm); respectively. EPA has determined that the petition contain data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of MKH–6561 (propoxycarbazone-sodium) in wheat was rapid, as only minor amounts of MKH–6561 were found in some of the wheat matrices. The primary metabolic pathway in wheat appeared to be hydroxylation of the propoxy side chain of MKH–6561 to give Pr-2-OH MKH–6561 (methyl 2-[[[(4,5-dihydro-4-methyl-5-oxo-3-(2'-hydroxy-propoxy)-1H-1,2,4-triazol-1-yl)carbonyl]amino]sulfonyl]benzoate). Hydrolysis of Pr-2-OH MKH–6561 then gave Pr-2-OH NMT and, probably the sulfonamide methyl ester which was not observed in any wheat matrices. Hydrolysis of the sulfonamide methyl ester gave sulfonamide acid, which was in equilibrium with saccharin. A minor metabolic pathway was demethylation of MKH–6561 to yield *N*-desmethyl MKH–6561.

2. *Analytical method*—i. *Plant*. The proposed tolerance expression is MKH–6561 and Pr-2-OH MKH–6561. An analytical method was developed to measure these two analytes in plant matrices. The method was validated in wheat tissues. MKH–6561 and Pr-2-OH MKH–6561 were extracted from the wheat tissues with 0.05 M NH_4OH using accelerated solvent extraction. Trifluoroacetic acid (0.5 milliliter (mL)) and an isotopically labeled internal standard were added to the extract. The whole extract was loaded onto a C–18 solid phase extraction (SPE) cartridge. The C–18 SPE cartridge was washed with aqueous trifluoroacetic acid (0.1%) and aqueous acetic acid (0.1%). A three to one mixture of acetonitrile and aqueous acetic acid (0.1%) was used to elute the analytes from the C–18 SPE cartridge. Water and acetic acid were added to the sample which was analyzed by LC/MS/MS.

ii. *Animal*. The proposed tolerance expression is MKH–6561. An analytical method was developed to measure this analyte in animal tissues and milk. The method was validated in animal tissues and milk. MKH–6561 was extracted from the tissues with 0.05 M NH_4OH

using accelerated solvent extraction. Trifluoroacetic acid (0.5 mL) and an isotopically labeled internal standard were added to the extract which was then centrifuged at 2,000 rpm for 10 minutes. Approximately half of the sample was loaded onto a C–18 SPE cartridge. The C–18 SPE cartridge was washed with aqueous trifluoroacetic acid (0.1%) and aqueous acetic acid (0.1%). A three to one mixture of acetonitrile and aqueous acetic acid (0.1%) was used to elute the analytes from the C–18 SPE cartridge. Water and acetic acid were added to the sample which was analyzed by LC/MS/MS. Milk samples were analyzed by amending an aliquot of milk with trifluororacetic acid (0.5 mL) and isotopically labeled internal standard. The sample was purified by C–18 SPE as described above. The resultant sample was analyzed by LC/MS/MS.

3. *Magnitude of residues*. Twenty-one field trials were conducted in 19 locations to evaluate the quantity of MKH–6561 and Pr-2-OH MKH–6561 in wheat forage, hay, straw, and grain following treatment with MKH–6561, 70 water dispersible granule (WG) at an application rate of 45 grams of active ingredient per hectare in the spring or at 30 grams active ingredient per hectare in the fall and 30 grams active ingredient per hectare in the spring. The highest average field trial (HAFT) residue in wheat forage, hay, straw, and grain were 1.21, 0.12, 0.03, and <0.01 ppm respectively.

B. Toxicological Profile

1. *Acute toxicity*. i. MKH–6561 is of very low acute toxicity to fasted rats following a single oral administration. The acute oral LD_{50} is >5,000 milligrams/kilogram/body weight (mg/kg/bwt) for males and females.

ii. MKH–6561 is not toxic to rats following a single dermal application. The acute dermal LD_{50} is >5,000 mg/kg/bwt for males and females.

iii. An acute inhalation study with rats showed low toxicity with a 4–hour dust aerosol LC_{50} 5,030 mg/m^3 air for males and females.

iv. An eye irritation study in rabbits showed minimal irritation completely reversible within 48 hours.

v. A dermal irritation study in rabbits showed slight irritation completely reversible within 48 hours.

vi. MKH–6561 has no skin sensitizing potential under the conditions of the maximization test in guinea pigs.

2. *Genotoxicity*. The genotoxic action of MKH–6561 was studied in bacteria and mammalian cells with the aid of various *in vitro* test systems (*Salmonella* microsome test, hypoxanthine guanine phophoribosy transferase (HGPRT) test with chinese hamster V79 cells, cytogenetic study with chinese hamster V79 cells, and unscheduled DNA synthesis (UDS) test) and in one *in vivo* test (micronucleus test). None of the tests revealed any evidence of a mutagenic or genotoxic potential of MKH–6561. The compound did not induce point mutation, DNA damage or chromosome aberration (CA).

3. *Reproductive and developmental toxicity*. i. In a 2–generation reproduction study, Wistar rats were administered MKH–6561 at levels of 0, 1,000, 4,000, or 16,000 ppm in the diet. The no observe adverse effect level (NOAEL) for reproductive parameters was established at 16,000 ppm (1,231 mg/kg bwt/day in males and 1,605 mg/kg bwt/day in females), the highest dose tested (HDT). The parental NOAEL was 1,000 ppm (80 mg/kg bwt/day in F_1 males and 93 mg/kg bwt/day in F_0 females).

ii. A developmental toxicity study was conducted with Wistar rats via oral gavage of MKH–6561 at levels of 0, 100, 300, and 1,000 mg/kg bwt/day on days 6 through 19 of gestation. There were no signs of maternal toxicity, embryotoxicity, fetotoxicity, or teratogenicity at the level of 1,000 mg/kg bwt/day. Therefore, the maternal and developmental NOAELs for rats were established at 1,000 mg/kg bwt/day, the limit dose for this study type. No teratogenic potential of MKH–6561 was evident in rats.

iii. Himalayan rabbits were administered MKH–6561 at levels of 0, 20, 100, 500, or 1,000 mg/kg bwt/day by oral gavage on days 6 through 28 post coitum in a test for developmental toxicity. A maternal NOAEL of 100 mg/kg bwt/day was established based on cold ears, alopecia, swelling of vulva, decreased feed, and water intake, body weight loss, gastrointestinal tract (GI) effects, liver effects, and thyroid hormone level effects. The gestation rate NOAEL of 100 mg/kg bwt/day was based on one abortion (assessed as secondary due to maternal toxicity) at 500 mg/kg bwt/day. The NOAEL for fetal parameters of 500 mg/kg bwt/day was based on placental effects, increased post-implantation loss, decreased number of fetuses, decreased fetal weight, retarded fetal skeletal ossification, and possible increase in lobulation of liver in fetuses at 1,000 mg/kg bwt/day. No teratogenic potential of MKH–6561 was evident in rabbits.

4. *Subchronic toxicity*. i. A 28–day dermal toxicity study in Wistar rats established a local and systemic NOAEL of 1,000 mg/kg bwt/day (the dermal limit dose) for males and females.

ii. A 14–week feeding study was conducted with Wistar rats at dietary dose levels of 0, 250, 1,000, 4,000, or 20,000 ppm. The NOAEL was determined to be 4,000 ppm (286.4 mg/kg bwt/day in males and 350.6 mg/kg bwt/day in females) based upon increased water consumption (reversible during the 4–week recovery period) and an irritative effect of the forestomach epithelium (reversible during the 4–week recovery period) in males and females dosed at 20,000 ppm as well as reduced glucose and triglyceride levels in females only dosed at 20,000 ppm.

iii. A 91–day feeding study was conducted with B6C3F_1 mice at dietary dose levels of 0, 625, 2,500, or 10,000 ppm. The NOAELs determined for males and females were 625 ppm (205 mg/kg bwt/day) and 2,500 ppm (1,159 mg/kg bwt/day), respectively, based on decreased body weights in 2,500 ppm males and 10,000 ppm females.

iv. A 2–month range-finding feeding study in Beagle dogs, at levels of 0, 1,000, 5,000, 10,000, and 40,000 ppm in the diet established a NOAEL of 10,000 ppm (322.2 mg/kg bwt/day in males and 285.6 mg/kg bwt/day in females) based on elevated hepatic biotransformation enzymes at 40,000 ppm.

5. *Chronic toxicity*. i. A 2–year chronic/oncogenicity study was conducted with male and female Fischer 344 rats at dietary levels of 0, 50, 500, or 1,000 mg/kg bwt/day for approximately the first 7 months of the study (dose adjustment). From approximately 7 months to study termination, the doses were 0, 1,000, 10,000, and 20,000 ppm in the diet. A chronic toxicity NOAEL of 1,000 ppm (43 mg/kg bwt/day in males and 49 mg/kg bwt/day in females) was determined based on increased urine pH and decreased body weight gain at 1–year (but not 2 years) at 10,000 ppm and 20,000 ppm. No carcinogenic potential was indicated.

ii. B6C3F_1 mice were administered MKH–6561 via the diet at levels of 0, 280, 1,400, and 7,000 ppm in a 2–year chronic feeding/carcinogenicity study. The chronic toxicity NOAEL was established at 1,400 ppm (369.0 mg/kg/day in males and 626.9 mg/kg bwt/day in females) based on retarded body weight development. No carcinogenic potential was indicated.

iii. A 1–year feeding study in Beagle dogs was conducted at 0, 2,000, 10,000, and 25,000 ppm in the diet. The NOAEL in males was determined to be 10,000 ppm (258.0 mg/kg bwt/day) based upon increased absolute adrenal gland weight without an increase in relative adrenal gland weight and slight enlargement of

zona fasciculata microscopically, without a correlation to adrenal gland weight in males dosed at 25,000 ppm. The NOAEL in females was determined to be 2,000 ppm (55.7 mg/kg bwt/day) based upon decreased food consumption and decreased relative heart weight in females dosed at 10,000 and 25,000 ppm.

6. *Animal metabolism*. i. A single oral dose of 2 mg/kg/bwt [triazolinone-3-^{14}C]MKH–6561 was administered to rats. Between 22% and 24% of the administered dose was absorbed. Maximum plasma radiation levels were observed 0.33 hours after dosing. Within 48 hours of dosing, between CA 88% and 97% of the radioactivity was excreted via urine and feces. Approximately 80–88% of the excreted radioactivity was unchanged parent compound. The highest single metabolite concentration was CA 3% of the administered dose. The terminal elimination half-live for total radioactivity was CA 12–13 hours, so no bioaccumulation of MKH–6561 or its metabolites will occur.

ii. Single oral doses of 2 mg/kg/bwt and 200 mg/kg/bwt [phenyl-UL-^{14}C]MKH–6561 were administered to rats. Between CA 21–31% of the administered dose was absorbed. Maximum plasma radiation levels were observed after 0.33 hours (low dose) and 1–hour (high dose). Within 48 hours of dosing, CA 97–104% of the administered dose was eliminated via urine and feces. Approximately 75–86% of the administered dose was eliminated as unchanged parent compound. The maximum single metabolite concentration was 8.8% of the administered dose. At the end of the study, less than 0.25% of the administered dose was found in organs and tissues. In a separate bile fistulation experiment, the predominantly fecal elimination was confirmed to be due to incomplete absorption of radioactivity from the GI tract. The terminal elimination half-live for total radioactivity was CA 9–11 hours, so no bioaccumulation of MKH–6561 or its metabolites will occur.

iii. Laying hens were given a daily dose of protonated MKH–6561 [phenyl-UL-^{14}C] at 3.12 mg/kg/bwt for 3 consecutive days. The residue levels were 1.343 ppm in liver, 0.017 ppm in muscle, 0.014 ppm in fat, 0.006 ppm in the day–1 eggs, 0.009 ppm in the day–2 eggs, and 0.012 ppm in the day–3 eggs. The residue levels based on a theoretical 1x application rate, as determined from residue levels observed in the MKH–6561 wheat field trials would all be considerably less than 0.001 ppm. The major residue identified in tissues and eggs were MKH–6561, Pr-2-OH MKH–6561, MKH–6561 sulfonamide methyl ester, and saccharin. The major metabolic pathway of MKH–6561 [phenyl-UL-^{14}C] in poultry was hydrolysis of the parent compound producing *N*-methyl propyl triazolinone and sulfonamide methyl ester. The sulfonamide methyl ester was then converted to saccharin. A minor pathway involved hydoxylation at the 2–position of the triazolinone propoxy group. In the liver, the major metabolic pathway led to the formation of protein bound MKH–6561 residue through conjugation with the amino acid serine.

iv. Laying hens were given a daily dose of protonated MKH–6561 [triazolinone-3-^{14}C] at 2.91 mg/kg/bwt for 3 consecutive days. The residue levels were 0.184 ppm in liver, 0.044 ppm in muscle, 0.015 ppm in the fat, 0.011 ppm in the day–1 egg, 0.016 ppm in the day–2 egg, and 0.022 ppm in the day–3 egg. The residue levels in tissues and eggs based on a theoretical 1x application, as determined from the residue levels observed in the MKH–6561 wheat field trials, would all be considerably less that 0.001 ppm. The metabolism of MKH–6561 [triazolinone-3-^{14}C] appeared to involve both hydroxylation at the 2–position of the propoxy group and hydrolysis of the phenyl sulfonamide linkage.

v. Goats were dosed with 1.0 mg/kg/bwt of MKH–6561 [phenyl-UL-^{14}C] for 3 consecutive days. Residue levels were 3.643 ppm in liver, 0.486 ppm in kidney, 0.009 ppm in muscle, 0.004 ppm in fat, 0.015 ppm in day–1 milk and, 0.022 ppm in day–2 milk. The metabolic pathway was based on hydrolysis of the sulfonamide to yield MKH–6561 sulfonamide methyl ester and saccharin. The saccharin was then conjugated to proteins which were found mainly in the liver and kidney.

vi. Goats were dosed with MKH–6561 [triazolinone-3-^{14}C] at a dose of 0.98 mg/kg/bwt for 3 consecutive days. Residue levels were 0.171 ppm in liver, 0.425 ppm in kidney, 0.040 ppm in muscle, 0.007 ppm in fat, 0.046 ppm in day–1 milk, and 0.057 ppm in day–2 milk. The metabolism of MKH–6561 involved the cleavage of the phenyl sulfonylurea side chain and the hydroxylation of the propyl side chain on the triazolinone ring system after the cleavage of the phenyl sulfonylurea side chain.

7. *Metabolite toxicology*. i. 4-OH-saccharin is of low acute toxicity to fasted rats following a single oral administration. The acute oral LD_{50} is >5,000 mg/kg/bwt for males and females. 4-OH-saccharin is considered non-mutagenic with and without S9 mix in the plate incorporation as well as in the preincubation modification of the *Salmonella* microsome test.

ii. MKH–8394 is of very low acute toxicity to fasted rats following a single oral administration. The acute oral LD_{50} is>5,000 mg/kg/bwt for males and females. MKH–8394 is considered non-mutagenic with and without S9 mix in the plate incorporation as well as in the preincubation modification of the *Salmonella* microsome test.

iii. KTS–9061 (Pr-2-OH MKH–6561) is not toxic to fasted rats following a single oral administration. The acute oral LD_{50} is>5,000 mg/kg/bwt for males and females. KTS–9061 is considered non-mutagenic with and without S9 mix in the plate incorporation as well as in the preincubation modification of the *Salmonella*/microsome test. KTS–9061 is considered non-clastogenic with and without S9 mix CA test *in vitro* using chinese hamster V79 cells. Wistar rats were administered KTS–9061 via the diet at levels of 0, 800, 4,000, and 10,000 ppm for approximately 4 weeks. The NOAEL was determined to be 10,000 ppm (905.3 mg/kg bwt/day in males and 880.0 mg/kg bwt/day in females), the HDT.

iv. KTS–9304 has low to moderate acute toxicity to fasted rats following a single oral administration. The acute oral LD_{50} was 263 mg/kg/bwt in males and 1,756 mg/kg/bwt in females. KTS–9304 is considered non-mutagenic with and without S9 mix in the plate incorporation as well as in the preincubation modification of the *Salmonella*/microsome test.

8. *Endocrine disruption*. There is no evidence to suggest that MKH–6561 has an effect on the endocrine system. Studies in this data base include evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following short-term and long-term exposure. These studies revealed no endocrine effects due to MKH–6561.

9. *Other studies*. i. An acute neurotoxicity screening study in Wistar rats established a NOAEL for males and females of 2,000 mg/kg/bwt (HDT).

ii. A 13–week neurotoxicity screening study in Wistar rats established a NOAEL of 20,000 ppm (1,321 mg/kg bwt/day in males and 1,651 mg/kg/day in females) (HDT). No neurotoxic potential was observed..

iii. A Plaque-Forming-Cell Assay to investigate immunotoxicological potential was performed on male Wistar rats after an approximate 4–week exposure of 0, 4,000, 10,000, or 20,000 ppm in the diet. The Plaque-Forming-Cell Assay NOAEL was 20,000 ppm (2,144 mg/kg bwt/day; HDT). The

overall study NOAEL was 10,000 ppm (986 mg/kg bwt/day) based upon increased water intake at 20,000 ppm.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food.* Estimates of chronic dietary exposure to residues of MKH–6561 utilized the proposed tolerances in wheat forage, wheat hay, wheat straw, wheat grain, meat, and meat byproducts (cattle, sheep, goats, horses, hogs), and milk of 1.5, 0.15, 0.05, 0.01, 0.05, and 0.002 ppm respectively. Other assumptions were that 100% of the target crop would be treated with MKH–6561 and that no loss of residue would occur due to processing or cooking. For chronic exposures, a reference dose (RfD) of 0.43 mg/kg/day was assumed based on and NOAEL of 43 mg/kg bwt/day from the combined chronic toxicity/oncogenicity study in the rat. A safety factor of 100 was used based on interspecies extrapolation (10x) and intraspecies variability (10x). Using these conservative assumptions, dietary residues of MKH–6561 contribute 0.000219 mg/kg/day (0.1% of the RfD) for children 1 to 6 years old, the most sensitive sub-population. For the U.S. population, the exposure was 0.000098 mg/kg/day (0.02% of the RfD). For acute dietary exposure, the same conservative assumptions were made. A NOAEL of 100 mg/kg bwt/day from the developmental toxicity study in rabbits and an safety factor of 100 were used in the acute dietary assessment. The safety factor of 100 was based on interspecies extrapolation (10x) and intraspecies variability (10x). Acute dietary exposure at the 95th percentile was negligible for all population subgroups. For children 1 to 6 years old (the most sensitive sub-population,) and for the U.S. population, <0.1% of the acute RfD was consumed at the 95th percentile.

ii. *Drinking water.* Estimates of chronic dietary exposure to residues of MKH–6561 utilized the proposed tolerances in wheat forage, wheat hay, wheat straw, wheat grain, meat, and meat byproducts (cattle, sheep, goats, horses, hogs), and milk of 1.5, 0.15, 0.05, 0.01, 0.05, and 0.002 ppm respectively. Other assumptions were that 100% of the target crop would be treated with MKH–6561 and that no loss of residue would occur due to processing or cooking. For chronic exposures, an RfD of 0.43 mg/kg/day was assumed based on and NOAEL of 43 mg/kg bwt/day from the combined chronic toxicity/oncogenicity study in the rat. A safety factor of 100 was used based on interspecies extrapolation (10x) and intraspecies variability (10x). Using these conservative assumptions, dietary residues of MKH–6561 contribute 0.000219 mg/kg/day (0.1% of the RfD) for children 1 to 6 years old, the most sensitive sub-population. For the U.S. population, the exposure was 0.000098 mg/kg/day (0.02% of the RfD). For acute dietary exposure, the same conservative assumptions were made. A NOAEL of 100 mg/kg bwt/day from the developmental toxicity study in rabbits and an safety factor of 100 were used in the acute dietary assessment. The safety factor of 100 was based on interspecies extrapolation (10x) and intraspecies variability (10x). Acute dietary exposure at the 95th percentile was negligible for all population subgroups. For children 1 to 6 years old (the most sensitive sub-population,) and for the U.S. population, <0.1% of the acute RfD was consumed at the 95th percentile.

2. *Non-dietary exposure.* There are no current non-food uses for BAY MKH–6561 registered under the Federal Insecticide, Fungicide, and Rodenticide Act, as amended. No non-food uses are proposed for BAY MKH6561 and no non-dietary exposures are expected for the general population.

D. Cumulative Effects

BAY MKH–6561 is a sulfonamide herbicide. There is no information to suggest that any chemical in this class of herbicides has a common mechanism of mammalian toxicity or that chemicals in this class produce similar effects so it is not appropriate to combine exposures of BAY MKH–6561 with other herbicides. Bayer Corporation is considering only the potential risk of BAY MKH–6561.

E. Safety Determination

1. *U.S. population.* As presented previously, the exposure of the U.S. general population to MKH–6561 is low, and the risks, based on comparisons to the RFD, are minimal. The margins of safety from the use of MKH–6561 are well within EPA's acceptable limits. Bayer Corporation concludes that there is a reasonable certainty that no harm will result to the U.S. population from aggregate exposure to MKH–6561 residues.

2. *Infants and children.* The complete toxicological data base including the developmental toxicity and 2–generation reproduction studies were considered in assessing the potential for additional sensitivity of infants and children to residues of BAY MKH–6561. The developmental toxicity studies in rats and rabbits revealed no increased sensitivity of rats or rabbits to *in-utero* exposure to BAY MKH–6561. The 2–generation reproduction study did not reveal any increased sensitivity of rats to *in-utero* or postnatal exposure to BAY MKH–6561. Furthermore, none of the other toxicology studies revealed any data demonstrating that young animals were more sensitive to BAY MKH–6561 than adult animals. The data taken collectively clearly demonstrate that application of a FQPA uncertainty factor for increased sensitivity of infants and children is not necessary for BAY MKH–6561.

F. International Tolerances

There are currently no international Codex tolerances established for BAY MKH–6561. It is not currently registered in any other countries. There are no harmonized maximum residue levels at the European Union level at present.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–2002–0172 FRL–7191–1]

Notice of Filing of Pesticide Petitions to Establish Tolerances for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket ID number OPP–2002,–0172, must be received on or before September 20, 2002.

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 ID number OPP–2002–0172 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Shaja R. Brothers, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–3194; e-mail address: brothers.shaja@epa.gov.

SUPPLEMENTARY INFORMATION: