ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0035; FRL–7293–9]

Butafenacil; 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–2003–0035, must be received on or before March 28, 2003.

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: Jim Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–5697; e-mail address: *tompkins.jim@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:

- 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 ID number OPP–2003–0035. 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, 1921 Jefferson Davis Hwy., 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 on 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–2002–0035. 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–2003–0035. 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–2003–0035.

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, 1921 Jefferson Davis Hwy., Arlington, VA, Attention: Docket ID number OPP–2003–0035. 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: February 12, 2003.

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 FFDCA section 408(d)(3). The summary of the petition was prepared by Syngenta Crop 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.

Syngenta Crop Protection, Inc.

PP 1F6309

EPA has received a pesticide petition (PP 1F6309) from Syngenta Crop Protection, Inc., Greensboro, NC 27419 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180, by establishing a tolerance for residues of butafenacil in or on the raw agricultural commodity cotton, undelinted seed at 0.5 parts per million (ppm) and cotton, gin byproducts at 13 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the

submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolic pathway of butafenacil in cotton after defoliation applications is understood. The data support the selection of the residue of concern for tolerance setting.

2. *Analytical method*. Syngenta Crop Protection, Inc. has submitted practical analytical methodology for detecting and measuring levels of butafenacil in or on raw agricultural commodities. This method is based on crop-specific cleanup procedures and determination by liquid chromatography with a liquid chromatography/mass spectroscopy (LC/MS) detector. The limit of quantitation is 0.01 ppm for butafenacil for all crops tested, including cotton. The limit of quantitation for metabolites is also 0.01 ppm except for cotton gin trash where the limit of quantitation is 0.05 ppm. The analytical method was validated by determination of recoveries for fortified samples.

3. *Magnitude of residues*. A residue program was performed with butafenacil on the full geography required to support use on cotton.

B. Toxicological Profile

1. *Acute toxicity*. Butafenacil technical and the 100 EC formulation (0.83 lb active ingredient/gallon (ai/gal) have very low order of acute toxicity by oral, dermal, and inhalation exposure routes. Butafenacil technical is mildly irritating to the eye and non-irritating to the skin. The 100 EC formulation is moderately irritating to the eye and skin. Neither the technical nor the formulation are skin sensitizers. The rat dermal LD_{50} is >5,000 milligram/kilogram (mg/kg). The rat dermal LD_{50} is >4,000 mg/kg and the rat inhalation LD_{50} is >5.1 milligrams per liter (mg/L) air. The end-use formulation of butafenacil has a similar low acute toxicity profile.

2. *Genotoxicity*. Butafenacil has been tested for its potential to induce gene mutation and chromosomal changes in five different test systems. Butafenacil technical did not induce point mutations in bacteria (ames assay in *salmonella typhimurium* or *escherichia coli*), and was not genotoxic in an *in vitro* unscheduled DNA synthesis assay in rat hepatocytes. Chromosome aberrations were not observed in an *in vitro* test using Chinese hamster ovary cells and there were no clastogenic or aneugenic effects on mouse bone marrow cell *in vivo* in a mouse micronucleus test. There was a borderline positive response in the gene mutation test in V79 cells *in vitro* at the highest concentration in the presence of metabolic activation, which proved to be cytotoxic. This effect was considered to be an isolated finding and not to be of relevance when assessing the overall mutagenic potential of butafenacil. To substantiate this finding, a corresponding *in vivo in-vitro* DNA repair study on rat hepatocytes was performed. The results of this test show no mutagenic potential of butafenacil. Consequently, it can be concluded that butafenacil is not genotoxic.

3. *Reproductive and developmental toxicity*. In rat and rabbit teratology studies there was no evidence of teratogenicity. Delayed fetal development was apparent only at maternally toxic doses of butafenacil technical in rabbits. In the rabbit study (with doses of 0, 10, 100, 1,000 mg/kg), 1,000 mg/kg/day caused a mean body weight loss from days 12 to 16, decreased food consumption during the dosing period and an increase in the incidence of post-implantation loss, almost exclusively in the form of early resorptions. This increase in post-implantation loss, which was restricted to the top dose, was considered to be secondary to the maternal toxicity occurring at this dose level, and not a direct effect by butafenacil. Slightly reduced fetal body weights at 1,000 mg/kg/day were considered secondary to maternal effects. The incidence and type of external, visceral and skeletal findings were not affected by treatment. There was no indication of developmental toxicity in rabbit offspring at 100 mg/kg/day. The no observed adverse effect level (NOAEL) for both maternal and developmental toxicity was established at 100 mg/kg/day in rabbits.

In the rat teratogenicity study 0, 10, 100, 1,000 mg/kg, there was no observation of maternal toxicity. Body weight and food consumption were comparable in all groups. Reproduction and fetal parameters were not impaired. The incidence and type of external, visceral and skeletal findings were comparable in all dose groups. No treatment-related findings were noted. In conclusion, butafenacil was not teratogenic and not toxic to the progeny. Maternal parameters were not affected. The NOAEL for both maternal and developmental toxicity was >1,000 mg/kg/day, the highest dose level tested.

In a rat multi-generation study, butafenacil technical was administered in feed at concentrations of 0, 30, 300, or 1,000 ppm. The dose in mg/kg/day spans a wide range over the duration of the study as animals gain weight and go through gestation and lactation. The ranges are 1.5–3.3, 15.5–31.9, and 50.9–101.6 for males and 1.7–6.3, 16.8–65.4, and 49.8–215.8 mg/kg/day for females at the 30, 300, or 1,000 ppm dietary levels, respectively. Butafenacil had no effect on reproductive parameters for either the F0 or F1 generation of parent animals. Parental body weight gain and food consumption were reduced at 300 and 1,000 ppm in both the F0 and F1 males and in F1 females. Increased incidence of liver pathology was observed in males and females in the F0 and F1 generations, including bile duct hyperplasia in both sexes at 300 and 1,000 ppm, hepatocellular hypertrophy in males at 1,000 ppm, and foci of necrosis in both sexes at 1,000 ppm and males at 300 ppm. Body weight gain was reduced during the lactation period at 300 and 1,000 ppm in offspring of the F0 generation and at 1,000 ppm in offspring of the F1 generation.

In conclusion, the NOAEL for systemic toxicity in both sexes and both generations of rats was 30 ppm (range = 1.5–3.3 mg/kg/day in males and 1.7–6.3 mg/kg/day in females). The grand mean test item intake (mean of all weekly means for both sexes, both generations, all time points) at this dose level was 2.48 mg/kg/day. There were no effects on the reproductive parameters and the NOAEL for reproductive toxicity was >1,000 ppm. Offspring effects were observed only at dose levels that also produced parental toxicity. There is no evidence that developing offspring are more sensitive than adults to the effects of butafenacil.

4. *Subchronic toxicity*. In a 90–day subchronic neurotoxicity study in rats, butafenacil was not neurotoxic when administered in the diet for 13 weeks at concentrations resulting in average daily test substance intakes of 0, 7.8, 23.5, or 74.4 mg/kg/day for males or at 0, 8.7, 26.0, or 78.9 mg/kg/day for females. There were no treatment-related neurobehavioral or motor activity effects, no macroscopic findings and no microscopic findings in central or peripheral nervous tissue. All animals survived until scheduled sacrifice and there were no treatment-related clinical observations. Histopathology of the liver revealed effects in animals of both sexes from the top dose group. In addition, one male at 23.5 mg/kg/day showed single cell necrosis of hepatocytes. In conclusion, subchronic dietary administration of butafenacil to rats did not produce neurotoxic effects at any dose level. The NOAEL for liver toxicity was 7.8 mg/kg/day for males and 26.0 mg/kg/day for females.

5. *Chronic toxicity*. Butafenacil technical was not oncogenic in rats or

mice. A summary of results of chronic toxicity studies in rats, mice, and dogs indicates that the primary target organ from chronic exposure is liver, with effects on hematology parameters and body weight.

In a 12–month chronic oral toxicity study, dogs were fed capsules containing butafenacil that resulted in daily test substance intakes of 0, 20, 100, 500, or 1,000 mg/kg/day. The administration of butafenacil caused findings only at 500 and 1,000 mg/kg/day. These effects consisted of loss in the body weight of male animals at 1,000 mg/kg/day. Hematology parameters were slightly affected at 500 and 1,000 mg/kg/day. Based on body weight loss at 1,000 mg/kg/day, the increase in relative liver weight at 1,000 mg/kg/day and the hematological effects at 500 and 1,000 mg/kg/day, the maximum tolerance dose (MTD) was achieved at 1,000 mg/kg/day and the NOAEL for chronic toxicity in dogs is 100 mg/kg/day.

In an 18–month oncogenicity study, mice were fed diets containing butafenacil that resulted in average daily test substance intakes of 0, 0.12, 0.36, 1.18, 6.78 mg/kg/day. The treatment of mice with butafenacil for 18 months revealed effects on hematology parameters in males at 1.18 and 6.78 mg/kg/day, increased liver weights at 6.78 mg/kg/day in both sexes and histopathological findings indicating that the liver was the target organ of toxicity. The MTD was achieved at 6.78 mg/kg/day. Dose responsive non-neoplastic changes in the liver occurred at 1.18 mg/kg/day in males and at 6.78 mg/kg/day in both sexes. Butafenacil was not carcinogenic in this study. Based on the hematology and liver effects, the NOAEL for chronic toxicity in mice was established at 0.36 mg/kg/day in males and 1.20 mg/kg/day in females.

In a 2–year chronic toxicity and carcinogenicity study, rats were fed diets containing butafenacil that resulted in average (sexes combined) daily test substance intakes of 0, 0.42, 1.22, 4.10, or 12.2 mg/kg/day. Treatment had no effect on survival and there were no treatment-related clinical signs. There were no effects on food consumption and body weight. Hematology and clinical chemistry data were comparable in all groups. Necropsy revealed no changes in organ weights.

The treatment of rats with butafenacil for 24 months indicated the liver as the target organ, with non-neoplastic histopathological findings in the liver in both sexes at 4.10 and 12.2 mg/kg/day. Based on the liver effects, the MTD was achieved at 12.2 mg/kg/day. No increased incidence of tumor formation was noted, indicating that butafenacil was not carcinogenic in this study. Based on the liver effects at 4.10 and 12.2 mg/kg/day, the NOAEL was established at 1.14 mg/kg/day (1.14 mg/kg/day in males and 1.30 mg/kg/day in females).

6. *Animal metabolism*. The major initial metabolic processes in rat involve the hydrolysis of the allyl ester to form the free carboxylic acid compounds. Parent compound was of significant abundance only in the feces from the high dose group. Subsequent metabolic routes involve reduction, hydroxylation, and opening of the uracil ring. The phenyl and uracil rings remain connected and all major metabolites have the unchanged phenyl structure.

7. *Metabolite toxicology*. Toxicity studies, including acute oral, mutagenicity, and 28–day feeding studies were conducted with major metabolites found in environmental studies. An acute oral and a mutagenicity test were conducted. The acute oral LD_{50} was at least >2,000 mg/kg and all mutagenicity studies were negative. The 28–day feeding study was conducted with major metabolites at 0, 300, 2,000, and 10,000 ppm. The target organ was confirmed as the liver for all test materials. Based on the data from the studies and reasons cited, none of these metabolites is considered to be of toxicological concern.

8. *Endocrine disruption*. Butafenacil does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There is no evidence that butafenacil has any effect on endocrine function in development or reproductive studies. Furthermore, histological investigation of endocrine organs in chronic dog, mouse, and rat studies did not indicate that the endocrine system is targeted by butafenacil.

9. *Neurotoxicity*. In an acute neurotoxicity study in rats, butafenacil was administered orally by gavage at 0 or 2,000 mg/kg. All animals survived and body weight development and food consumption were not affected by treatment. There were no toxicologically relevant clinical signs nor changes in observations and functional tests conducted as part of the functional observational battery. No treatment-related effects on any of the different motor activity parameters were seen. Macroscopical and microscopical examination of the multiple areas of the central and peripheral nervous system, the eyes, optic nerves, and skeletal muscle of the male and female, control and treated animals did not reveal any treatment-related neuropathic changes. In conclusion, butafenacil was devoid of any acute neurotoxicity when administered to rats at a single oral dose of 2,000 mg/kg. The NOAEL was greater than 2,000 mg/kg body weight.

C. Aggregate Exposure

1. *Dietary exposure*. Dietary exposure from butafenacil potentially exists through both food commodities and drinking water. Each exposure pathway is addressed below.

i. *Food.* Chronic and acute dietary exposure evaluations for butafenacil were performed using average field trial residues and assuming 100% crop treated. Cotton is the only raw agricultural commodity included in the assessment. All dietary exposure evaluations were made using the Dietary Exposure Evaluation Model (DEEM) and the USDA's Continuing Survey of Food Intake By Individuals (1994–96). Chronic exposure was compared to a chronic NOAEL of 100.0 mg/kg body weight/day (bwt/day) from a 1–year dog study. The acute NOAEL is 100 mg/kg in a rabbit teratology study based on maternal body weight loss and increased post-implantation loss. A 100X-uncertainty factor was assumed for both chronic and acute values. Both chronic and acute exposures were expressed as a percent of a reference dose of 1.0 mg/kg/day.

Secondary residues in animal commodities were calculated by constructing diets for beef and dairy cattle, poultry and swine in order to calculate anticipated residues in meat, fat, milk and pork. The beef cattle diet was used to calculate meat, fat and organ meats. The dairy cattle diet was used to estimate residues in milk. The swine diet was used for secondary residues in pork commodities and the poultry diet was used for residues in poultry commodities. Each diet was calculated using averaged field trial residues. Beef (cattle and dairy), and swine transfer factors were derived from a lactating goat 14C-metabolism study.

The results were favorable in both acute and chronic assessment scenarios. Acute and chronic exposure values were negligible (less than 0.01% of the acute and chronic reference dose of 1 mg/kg bwt/day.

The major contributors to chronic exposure (children 1–6 years old) were milk, accounting for 48% of the total exposure, cottonseed oil accounting for 28%, and meat (beef) products accounting for 25% of the total. In the U.S. population, the percentage contribution to the chronic exposure from meat (beef) products and milk were each 34% and cottonseed oil

accounted for 31% of the total. Major sources of acute exposures for the U.S. population and children 1–6 years old included cottonseed oil and meat (beef) commodities. The %RfD for all populations was less than 0.01% of the reference dose (RfD) of 1.0 mg/kg bwt/day.

ii. *Drinking water—a. Acute drinking water exposure.* The estimated tier 1 maximum concentrations of butafenacil in surface water and ground water are 1.98 ppb and 0.000038 ppb, respectively. The acute RfD for butafenacil is 1.0 mg/kg bwt/day. From the acute dietary exposure analysis, acute food exposure from the uses of butafenacil were neglegible for all populations. Using this information, acute drinking water levels of comparison (DWLOC) were calculated for butafenacil. The lowest DWLOC was 10,000 ppb. Based on this analysis, butafenacil estimated environmental concentrations (EECs) do not exceed the calculated acute DWLOCs.

b. *Chronic drinking water exposure.* The estimated maximum concentrations of butafenacil in surface water and ground water are 0.033 ppb Day 56 EEC/3 from Generic Expected Environmental Concentration (GENEEC) and 0.000025 parts per billion (ppb) (SCI-GROW, maximum at 0.16 lb active ingredient/acre/year, respectively. The chronic RfD for butafenacil is 1.0 mg/kg bwt/day. From the chronic dietary exposure analysis, an exposure to butafenacil is negligible for all populations. Based on EPA's ''Interim Guidance for Conducting Drinking Water Exposure and Risk Assessments'' document (December 2, 1997), chronic drinking water levels of comparison were calculated for butafenacil. The lowest DWLOC was 10,000 ppb. Based on this analysis, butafenacil EECs do not exceed the calculated chronic DWLOCs.

2. *Non-dietary exposure*. There are no residential uses and therefore, no need for non-dietary exposure assessment for this use.

D. Cumulative Effects

The potential for cumulative effects of butafenacil and other substances that have a common mechanism of toxicity has been considered. Butafenacil is a member of the class of herbicides designated as uracil-derivatives. There is no reliable information to indicate that toxic effects produced by butafenacil would be cumulative with those of any other chemical including another pesticide. Therefore, Syngenta believes it is appropriate to consider only the potential risks of butafenacil in an aggregate risk assessment.

E. Safety Determination

1. *U.S. population*. Using the acute and chronic exposure assumptions and the proposed RfDs described above, the aggregate exposure, including drinking water to butafenacil to the U.S. population (48 contiguous states, all seasons) was calculated to be less than 0.01% of the RfD of 1.0 mg/kg bwt/day. Therefore, Syngenta concludes that there is reasonable certainty that no harm will result from the aggregate acute or chronic exposure to butafenacil residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of butafenacil, data from developmental toxicity studies in the rat and rabbit and a multi-generation reproduction study in the rat have been considered. In the rat and rabbit teratology studies there was no evidence of teratogenicity. Delayed fetal development was apparent only at maternally toxic doses of butafenacil technical in rabbits. In the rabbit study 1,000 mg/kg/day caused effects indicative of maternal toxicity. There was no indication of developmental toxicity in rabbit offspring at 100 mg/kg/day. The NOAEL for both maternal and developmental toxicity was established at 100 mg/kg/day in rabbits.

In the rat teratogenicity study there was no observation of maternal toxicity. Body weight and food consumption were comparable in all groups. Reproduction and fetal parameters were not impaired. Butafenacil was not teratogenic and not toxic to the progeny. Maternal parameters were not affected. The NOAEL for both maternal and developmental toxicity was ≥1,000 mg/kg/day, the highest dose level tested.

In a rat multi-generation study the NOAEL for systemic toxicity in both sexes and both generations of rats was 2.48 mg/kg/day. There were no effects on the reproductive parameters and the NOAEL for reproductive toxicity was ≥1,000 ppm. Offspring effects were observed only at dose levels that also produced parental toxicity. There is no evidence that developing offsprings are more sensitive than adults to the effects of butafenacil.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base. Based on the current toxicological requirements, the data base for butafenacil relative to prenatal and postnatal effects for children is complete. Further, for butafenacil, the developmental studies showed no increased sensitivity in fetuses as compared to maternal animals following in-utero exposures in rats and rabbits, and no increased sensitivity in pups as compared to the adults in the multi-generation reproductive toxicity study. Therefore, it is concluded, that an additional uncertainty factor is not warranted to protect the health of infants and children and that a RfD of 1.0 mg/kg bwt/day is appropriated for assessing aggregate risk to infants and children from butafenacil.

F. International Tolerances

There are no codex established for residues of butafenacil on cotton, undelinted seed or cotton, gin byproducts.

[FR Doc. 03–4386 Filed 2–25–03; 8:45 am]
BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0042; FRL–7293–4]

Issuance of an Experimental Use Permit

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has granted an experimental use permit (EUP) to the following pesticide applicant. An EUP permits use of a pesticide for experimental or research purposes only in accordance with the limitations in the permit.

FOR FURTHER INFORMATION CONTACT: Denise Greenway, Biopesticides and Pollution Prevention Division (7511C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–8263; e-mail address: *greenway.denise@epa.gov.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed to the public in general. Although this action may be of particular interest to those persons who conduct or sponsor research on pesticides, the Agency has not attempted to describe all the specific entities that may be affected by this action. If you have any questions regarding the information in this action, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.