(NTIS) ATTN: Order Desk 5285 Port Royal Road, Springfield, VA 22161. Telephone: 1–800–553–6847. When requesting a document from NTIS, please provide its name and NTIS Publication Number (PB). The NTIS Publication for this version of the Pesticide Data Submitters List is PB 2000–102113.

2. *Electronically:* The Pesticide Data Submitters List is available of EPA's World Wide Web (WWW) site on the Internet. The Internet address of EPA's web site is www.epa.gov. To Access the Data Submitters List from the EPA Home Page, select ''Databases and Software.'' From the next page, select ''Media Specific.''

The Pesticide Data Submitters list may be found by searching for the keywords ''datasubmitterslist'' from the EPA Home Page, or may be access directly on the EPA web site, by going directly to the address listed below. Note that this address is case sensitive. http://www.epa.gov./oppmsd1/datasubmitterslist/index.html.

II. What Action is the Agency Taking?

The Pesticide Data Submitters List is a compilation of names and addresses of registrants who wish to be notified and offered compensation for use of their data. It was developed to assist pesticide applicants in fulfilling their obligation as required by sections 3(c)(1)(f) and 3(c)(2)(D) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and 40 CFR part 152 subpart E regarding ownership of data used to support registration. This notice announces the availability of an updated version of the Pesticide Data Submitters List which supersedes and replaces all previous versions.

List of Subjects

Environmental protection, Administrative practice and procedure, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: April 5, 2000.

Richard D. Schmitt,

Acting Director, Information Resources and Services Division, Office of Pesticide Programs.

[FR Doc. 00–10189 Filed 4–25–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–938; FRL–6554–2]

Notice of Filing a Pesticide Petition to Establish a Tolerance for Certain Pesticide Chemicals in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the amended filing of a pesticide petition proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by docket control number PF–938, must be received on or before May 26, 2000.

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

FOR FURTHER INFORMATION CONTACT: By mail: Susan Stanton, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–5218; e-mail address: stanton.susan@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	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'' 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–938. 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–938 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, Ariel Rios Bldg., 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–938. 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 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 supports 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: April 14, 2000.

James Jones,

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.

Novartis Crop Protection, Inc.

PP 7F4924

Amended Pesticide Petition

On June 5, 1998, EPA published a notice that it had received a pesticide petition (PP 7F4924) from Novartis Crop Protection, Inc., P.O. Box 18300, Greensboro, NC 27419 proposing tolerances for the herbicide clodinafop-propargyl (propanoic acid, 2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-2-propynyl ester; CGA–184927) in or on the raw agricultural commodities of wheat. EPA has received an amendment to PP 7F4924 from Novartis Crop Protection, Inc., P.O. Box 18300, Greensboro, NC 27419 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 to increase, as requested by EPA, the original proposed tolerances; thereby establishing tolerances for the combined residues of clodinafop-propargyl and its acid metabolite, CGA–193469 ((R)-2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-propanoic acid), in or on the raw agricultural commodities wheat, grain at 0.1 ppm; wheat, forage at 0.1 ppm; wheat, hay at 0.1 ppm and wheat, straw at 0.5 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 clodinafop-propargyl in wheat is understood for the purposes of the proposed tolerances. Two studies, one with the racemic mixture of the R (+) and S (−) forms and the other with the pure R (+) form (CGA–184927 pyridyloxy labeled), gave similar results. Metabolism involves hydrolysis of the parent to the resulting acid followed by conjugation, arylhydroxylation at the 6 position of the pyridyl ring followed by sugar conjugation, and cleavage of the pyridinyloxy-phenoxy ether bridge which forms the breakdown products 2-(4-hydroxyphenoxy) propanoic acid and 2-hydroxy-3-fluoro-5-chloropyridine.

2. *Analytical method.* Novartis has submitted practical analytical methods for the determination of clodinafop-propargyl and its major plant metabolite CGA–193469 in wheat raw agricultural commodities (RACs). Clodinafop-

propargyl is extracted from crops with acetonitrile, cleaned up by solvent partition and solid phase extraction and determined by column switching high performance liquid chromotography (HPLC) with ultra violet (UV) detection. CGA–193469 is extracted from crops with an acetone-buffer (pH=3) solution, cleaned up by solvent partition and solid phase extraction, and determined by HPLC with UV detection. The limits of quantitation (LOQ) for the methods are 0.02 ppm for clodinafop-propargyl in grain and forage, 0.05 ppm for clodinafop-propargyl in straw, and 0.05 ppm for CGA–193469 in forage, straw and grain.

3. *Magnitude of residues*. Both Canadian and U.S. spring wheat residue trials were conducted. Twelve residue trials were conducted from 1989–1992 in the major spring wheat growing areas of Manitoba, Alberta and Saskatchewan, which share compatible crop zones with the major spring wheat growing areas of the United States (MT, ND, SD, MN). Nine trials were conducted in 1989–91 with a tank mix of clodinafop-propargyl and a safener as separate EC formulations, and three trials in 1992 were conducted with clodinafop-propargyl and the safener as a pre-pack EC formulation. All trials had a single post-emergence application of clodinafop-propargyl at a rate of 80 gram active ingredient/hectacre (g a.i./ha).

In 1998, an additional six spring wheat trials were conducted in the major growing areas of the United States. In these trials, clodinafop-propargyl was applied as a single application of a 240EC formulation at a rate of 70 g a.i./ha. Samples of 30–day forage and hay, and mature straw and grain treated 60 days prior to harvest were taken for analysis. Grain treated at an exaggerated rate in one trial was processed under simulated commercial processing conditions.

At pre-harvest intervals (PHIs) of 30 days for forage and hay in the U.S. trials, and 60–97 days for mature straw and grain in all trials, no detectable residues of clodinafop-propargyl were found. Residues of the metabolite CGA–193469 were detected in mature straw from four trials, with a maximum Highest Average Field Trail (HAFT) residue of 0.35 ppm. Separate decline studies on green forage in both the United States and Canada showed no detectable residues of clodinafop-propargyl or the metabolite CGA–193469 beyond the 7 days after application interval. No residues of clodinafop-propargyl or the metabolite CGA–193469 were found in mature grain or grain processed fractions in any trial.

A freezer storage stability study indicated reasonable stability of both analytes for a period of 1 year, with clodinafop-propargyl showing a decline to 56% in grain and 47% in straw after 2 years. CGA–193469 remained stable for at least 2 years.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral and dermal LD_{50} values for clodinafop-propargyl are 1,829 milligrams/kilograms (mg/kg) and greater than 2,000 mg/kg for rats of both sexes, respectively. Its acute inhalation LC_{50} in the rat is greater than 2.33 milligram/liter (mg/L), the highest attainable concentration. Clodinafop-propargyl is slightly irritating to the eyes, minimally irritating to the skin of rabbits, but was found to be sensitizing to the skin of the guinea pig. This technical will carry the EPA signal word ''Caution.''

2. *Genotoxicity*. The mutagenic potential of clodinafop-propargyl was investigated in six independent studies covering different end points in eukaryotes and prokaryotes *in vivo* and *in vitro*. These tests included: Ames reverse mutation with *Salmonella typhimurium* and Chinese hamster V79 cells *in vitro*; chromosomal aberrations using human lymphocytes *in vitro* and the mouse micronucleus test *in vivo*; and DNA repair using rat hepatocytes and human fibroblasts *in vitro*. Clodinafop-propargyl was found to be negative in all these tests and, therefore, is considered devoid of any genotoxic potential at the levels of specific genes, chromosomes, or DNA primary structure.

3. *Reproductive and developmental toxicity*. Dietary administration of clodinafop-propargyl over 2–generations at levels as high as 1,000 ppm did not affect mating performance, fertility, or litter sizes. Body weight was reduced in parental animals at 500 and 1,000 ppm. The physiological developmental and the survival of the pups during the last week of the lactation period were slightly reduced at levels equal to or greater than 500 ppm during the first generation only. Target organs were liver (adults) and kidney (adults and pups). The treatment had no effect on reproductive organs. The NOAEL for toxicity to the parental rats and offspring was 50 ppm, corresponding to a mean daily intake of 3.2 mg/kg clodinafop-propargyl. The NOAEL for reproductive toxicity was 1,000 ppm (64.2 milligram/kilogram body weight/day (mg/kg bw/day)).

In a developmental toxicity study in rats, the highest dose level of 160 mg/kg resulted in reduced body weight gain of the dams and signs of retarded fetal body weight and incomplete ossification of vertebrae and sternebrae. No teratogenic activity of the test article was detected. Novartis concluded that the NOAEL for dams and fetuses was 40 mg/kg/day. The EPA's Hazard Identification Assessment Review Committee (HIARC) concluded that based on an increase in bilateral distension and torsion of the ureters and delayed ossification in the fetuses, the developmental LOAEL was 40 mg/kg/day and the NOAEL was 5 mg/kg/day.

In a developmental toxicity study in rabbits, mortality was observed in dams at dose levels of 125 and 175 mg/kg. No teratogenic or fetotoxic effects were noted. Novartis concluded that the maternal NOAEL was 25 mg/kg/day and the fetal NOAEL was 175 mg/kg/day. The HIARC considered that the developmental NOAEL was 125 mg/kg/day due to significant mortality at 175 mg/kg/day.

4. *Subchronic toxicity*. A 90–day feeding study in rats at 1,000 ppm resulted in reduced body weight gain, increased liver weights, hematological changes, and increased serum activities of the alkaline phosphatase. Target organs were liver (increased weight), thymus (atrophy) and spleen (reduced weight). The changes were reversible during 4 weeks of recovery. The NOAEL was 15 ppm (0.92 mg/kg in males and 0.94 mg/kg in females). The EPA HIARC suggested the NOAEL in female rats was 8.24 mg/kg bw/day.

In a 90-day feeding study in mice, 400 ppm resulted in reduced activity, one death, markedly increased activities of aminotransferases, alkaline phosphatase, and albumin concentration, increased liver weights, hepatocellular hypertrophy, and single cell necroses in all mice. Other findings included intrahepatic bile duct proliferation, Kupffer cell hyperplasia, and higher incidence of inflammatory cell infiltration. These findings were considered to be secondary to the hepatocyte necrosis. The NOAEL of 6 ppm was equivalent to a daily dose of 0.9 mg/kg in males and 1.05 mg/kg in females.

In a 90–day study in beagle dogs, levels of 500 and 1,000 ppm fed over 2 weeks clearly exceeded a maximum tolerated dose and led to mortality and severe toxicity. Effects at 50 and 200 ppm were limited to dermatitis and clinical chemistry changes, which were generally mild and transient. The NOAEL of 10 ppm was equivalent to a mean daily intake of 0.36 mg/kg in males. The HIARC concluded that in

females the NOAEL was 50 ppm (1.9 mg/kg bw/day).

5. *Chronic toxicity*. In a 12-month feeding study in dogs, 500 ppm resulted in transient dermatitis and reduced body weight gain. Two females were more severely affected and showed inappetence, body weight loss, tremors, and severe dermatitis, and necessitated an interruption of the treatment in order to avoid mortality. Histopathology revealed slight hepatocellular hypertrophy in one male and one female. The NOAEL of 100 ppm was equivalent to a mean daily intake of 3.38 mg/kg in males and 3.37 mg/kg in female.

Lifetime dietary administration of clodinafop-propargyl to mice resulted in reduced body weights and reduced survival in males treated at 250 ppm. Severe hepatotoxicity was noted at 100 and 250 ppm in both sexes. Based on markedly increased liver weights, enhanced serum activities of hepatic enzymes and hepatocellular necroses, dietary levels of 100 ppm and 250 ppm clearly exceeded maximum tolerated doses in males and females, respectively. The increased incidence of benign liver tumors that occurred in males treated at 250 ppm was, therefore, considered a toxicologically irrelevant response as the livers of these animals were damaged significantly and this finding was not interpretable. The toxicity to liver can be associated with the peroxisomal proliferating activity of clodinafop-propargyl in the mouse. Despite this mode of action, the incidence of hepatocellular carcinoma, in these clearly compromised mice, remained within the historical control range, although the incidence was slightly increased in comparison to the concomitant controls. Tumor incidences in females were generally low and well within the range of the historical controls. The NOAEL of 10 ppm was equivalent to a mean daily dose of 1.10 mg/kg in males and 1.25 mg/kg in females.

Dietary treatment of rats with concentrations over 2 years resulted in initial inappetence in males and reduced body weight development in both sexes treated at 750 ppm. The main target organ of toxicity was the liver. Changes in plasma protein and lipid levels, strongly enhanced serum activities of liver enzymes, increased liver weights, and severe liver necroses were observed at dietary doses of 300 and 750 ppm in males and at 750 ppm in females. The degenerative lesions provide strong evidence that these dose levels exceeded a maximum tolerated dose (MTD). Top dose group males showed a higher incidence of prostate adenoma, while prostate hyperplasia was reduced. However, the total incidence of proliferative changes in the prostate remained unchanged indicating a progression from prostate hyperplasia to adenoma. Females treated at the same high dose had higher incidences of ovary tubular adenoma. The slightly enhanced incidences of these lesions are likely a consequence of the severe disturbance of the general metabolic balance due to excessive liver toxicity. In fact, male rats fed 750 ppm exhibited a marked increase in peroxisomalβ oxidation, and an increase in cytochrome P450 4A1/ A3 and 4A2 in their livers. Further, a decrease in cytochrome P450 isoenzymes including CYP 2A, CYP 3A, and male-specific CYP 2C11 was observed. The total oxidation rate of testosterone, aromatase (CYP 19A1) activity plasma estradiol concentration and plasmaβ—dihydrotestosterone are altered at this level of treatment. Clodinafop-propargyl is a potent peroxisome proliferator in the rat liver and this peroxisomal prolifering activity manifests itself by altering Cytochrome P450-dependent monooxygenses which are involved in steroid hormone homeostasis. The NOAEL of 10 ppm was equivalent to a mean daily dose of 0.32 mg/kg in males and 0.37 mg/kg in females. The EPA HIARC concluded that based on hepatocellular hypertrophy and kidney findings, the NOAEL was 1 ppm (0.031 in males and 0.034 in females.

Carcinogenicity. The EPA HIARC recommended, based on the increased incidence of prostate and ovarian tumors in rats and hepatocellular tumors in mice, that the Cancer Assessment Review Committee review clodinafop-propargyl. A Q1* value based on the combined incidence of liver tumors in male mice has been calculated by the EPA Science Analysis Branch. The Q1* value estimate is $1.29 \times E^{-1}$ $(mg/kg/day)^{-1}$ in human equivalents.

6. *Animal metabolism*. In rats, clodinafop-propargyl was rapidly absorbed through the gastrointestinal tract. Absorption through the skin of rats is considerably slower with 15% of a dermally applied dose being absorbed within 8 hours. The EPA HIARC estimated the dermal absorption rate for clodinafop-propargyl to be 2.5% derived by taking the ratio of the LOAEL from the 28-day oral toxicity study in rats (5 mg/kg/day) and 28-day dermal toxicity study in rats (200 mg/kg/day). Female rats excreted single doses more rapidly than males. Most likely due to enzyme induction, differences were much less pronounced after repeated treatment. Both sexes excreted clodinafop-propargyl with urine and feces mainly in the form of its propionic acid derivative, CGA–193469. Simultaneous administration of the safener, cloquintocet-mexyl, did not alter the rate of excretion of clodinafop-propargyl or its metabolite pattern.

7. *Metabolite toxicology*. Clodinafop-propargyl acts as a typical peroxisome proliferator in the rodent liver, which is most likely induced by its propionic acid derivative metabolite, CGA–193469. Like other known well-characterized substances with this property, CGA–193469 caused peroxisome proliferation *in vitro* in hepatocytes of the mouse and rat, but not of the Guinea pig, marmoset, or human. In addition, clodinafop-propargyl was unable to activate the PPAR α-dependent human ACYL CoA oxidase promoter which further supports the evidence that humans are refractory to peroxisome proliferation and related changes. The scientific evidence available amply demonstrates that exposure to substances that produce tumors by a peroxisome proliferator mode of action does not represent a risk of tumor development in man. Novartis, therefore, has concluded that clodinafop-propargyl is not a carcinogen of relevance to humans.

8. *Endocrine disruption*. No special studies investigating potential estrogenic or endocrine effects of clodinafop-propargyl have been conducted. However, the standard battery of required studies has been completed. These studies include an evaluation of the potential effects on reproduction and development and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure. Although prostate adenomas and ovarian adenomas were observed to be statistically increased in rats at the highest feeding level with clodinafop-propargyl, this feeding level clearly exceeded the MTD and the livers in these rats were severely compromised. These findings in the endocrine organs were considered to be secondary to the severe liver effects.

C. Aggregate Exposure

1. *Dietary exposure*. Chronic and acute dietary exposure were calculated for the use of clodinafop-propargyl and the corresponding hydrolysis product, CGA–193469 on wheat. Analyses were conducted using the Dietary Exposure Evaluation Model (DEEM™) by Novigen Sciences and the 1994–96 Continuing Survey of Food Intake (CSFII). Chronic and acute tier three assessments were conducted to account for the consumption of commodities containing

wheat grain and residues were adjusted with a projected percent of crop treated value of 4%. Residues of parent clodinafop-propargyl were below the limit of quantitation (LOQ) of 0.02 ppm in all grain samples. Residues of the acid (CGA–193469) were also below the LOQ (LOQ = 0.05 ppm) in grain. Since no residues were observed in any of the samples, a statistical limit of detection (sLOD) was calculated for parent and the corresponding acid metabolite and one-half of the sLOD of each were summed and entered into the chronic and the acute assessments. Although wheat fractions may be fed to livestock and poultry, calculation of dietary burden with subsequent transfer to animal commodities shows secondary residues are extremely negligible and do not impact risk. Tolerances of 0.1 ppm are being proposed for clodinafop-propargyl and the acid metabolite, CGA–193469, for wheat grain, forage, and hay and 0.5 ppm for straw. Tolerances for meat, milk and eggs are not required.

i. *Food*—a. *Chronic*. Chronic exposure was compared to a chronic reference dose (RfD) of 0.00003 mg/kg/day based on a no-effect level of 0.03 mg/kg/day from a 2-year chronic toxicity/carcinogenicity study in rats and a 1,000X uncertainty factor. Exposure results are compared against the aforementioned reference dose as well as the Agency's $Q1^*$ value of 0.129. Since all residues in grain were below the LOQ, an sLOD was calculated for parent and CGA–193469. One-half sLOD values for parent clodinafop-propargyl and the corresponding acid were 0.0049 ppm and 0.0147 ppm, respectively. These values were summed and adjusted with a market share value of 4% for the calculation of exposure. The exposure results show that the U.S. population utilizes 4.3% of the chronic RfD. The most sensitive subpopulation is children (1–6 years old) with an exposure of 9.9% of the chronic RfD. Using the Agency's $Q1^*$ value of 0.129, a lifetime risk of 1.35×10^{-7} was calculated. These results indicate there is more than a reasonable certainty that exposure to residues of clodinafop-propargyl and its corresponding acid metabolite (CGA–193469) will result in no harm.

b. *Acute*. Acute exposure to females greater than 13 years old was compared to an acute reference dose (aRfD) of 0.005 mg/kg/day based on a NOAEL of 5 mg/kg/day from a developmental study in rats and a 1,000x uncertainty factor. As in the chronic assessment, one-half sLOD was used for parent clodinafop-propargyl and the corresponding acid (0.0049 ppm and 0.0147 ppm for parent and acid, respectively). These values were summed and zeroes were added to the residue distribution file corresponding to the percent of crop not treated (96% not treated). For all female populations in the DEEM™, exposure ranged from 3.0% – 4.2% of the aRfD at the 99.9th percentile of exposure. The most sensitive female population was nursing females (13+ years old) with an exposure of 4.2% of the aRfD (99.9th percentile). Acute exposure for the general population excluding females (> 13 years old), was compared to an aRfD of 0.025 mg/kg/day based on a NOAEL of 25 mg/kg/day from a developmental study in rabbits and a 1,000x uncertainty factor (UF). Acute exposure at the 99.9th percentile for the general population, children and males (all populations excluding females) ranged from 0.18% (seniors, 55+) to 0.62% of the aRfD for children (1–6 years old). These results demonstrate that there is a high degree of certainty of no harm resulting from acute exposure to dietary residues of clodinafop-propargyl.

ii. *Drinking water*. Another potential route of exposure to residues of pesticides includes drinking water. Field and laboratory study results have demonstrated that clodinafop-propargyl and its degradation products have slight to medium mobility in soil. However, due to rapid degradation of the product under field conditions and its low application rate, the potential for it to reach surface and ground water is considered to be negligible. Thus, drinking water exposure to clodinafop-propargyl and its degradation products was not included in the aggregate risk assessment. Also, since clodinafop-propargyl is not intended for uses other than the agricultural use on wheat, there is no potential for nonoccupational exposure.

The estimated exposures of clodinafop-propargyl and its main environmental degradate were combined and the hazards for both compounds were based on the RfD values determined for clodinafop-propargyl alone. The estimated water concentrations for clodinafop-propargyl and the degradate were estimated, weighted and combined based on applications rates adjusted for the maximum concentration of the degradate present in the aerobic soil metabolism studies.

The Screening Concentration in Ground Water (SCI–GROW) model was used to provide the estimated ground water concentration of the combined clodinafop-propargyl and degradate residues, 0.006688 ppb. The Pesticide Root Zone Model/Exposure Analysis Modeling Systems (PRZM/EXAMS) model using the Index Reservoir scenario and the Percent Cropped Area provided the estimated surface water concentrations of the combined clodinafop-propargyl and degradate residues for a wheat application in North Dakota. The estimated 90th percentile acute peak concentration for the combined residues was 0.792 part per billion (ppb). The estimated 36-year mean-yearly chronic concentration for the combined residues was 0.0519 ppb.

Concerning the acute and chronic exposures to clodinafop-propargyl and the degradate, an additional 10×-safety factor has been proposed by the EPA HIARC for the protection of infants and children. This additional safety factor was applied to the acute and chronic non-cancer RfD values for all sub-populations as a worse case estimate of exposure. This resulted in an acute RfD for females 13+ years of 0.005 mg/kg/day and 0.025 mg/kg/day for all other subgroups. This also resulted in a chronic RfD for infants and children of 0.00003 mg/kg/day. A chronic lifetime cancer risk exposure of 0.129 mg/kg/day has also been proposed by the EPA. This was applied to the adult population exposures only.

For ground water, the acute dietary assessment provided drinking water levels of comparison (DWLOC) ranging from 140 to 873 ppb. The estimated ground water concentration, 0.006688 ppb, represented from 0.0008% to 0.0048% of the acute RfD for all sub-populations. The chronic dietary exposures provided DWLOC values of 0.16 ppb (infants), 0.31 ppb (children), and 0.2026 to 0.2363 ppb (lifetime cancer risk for adults). The estimated ground water concentration represented 3.98%, 1.93% and 2.5 to 2.9% of the chronic risk, respectively.

For surface water, the acute dietary assessment provided DWLOC values ranging from 140 to 873 ppb. The estimated acute surface water concentration, 0.792 ppb, represented from 0.09% to 0.57% of the acute RfD for all sub-populations. The chronic dietary exposures provided DWLOC values of 0.16 ppb (infants), 0.31 ppb (children), and 0.2026 to 0.2363 ppb (lifetime cancer risk for adults). The estimated surface water concentration, 0.0519 ppb, represented 31%, 15% and 19.1–to–22.3% of the chronic risk, respectively. Therefore, the acute and chronic drinking water exposures for clodinafop-propargyl and its main environmental degradate did not exceed the exposures allowed by the risk cup.

2. *Non-dietary exposure*. Exposure to clodinafop-propargyl for the mixer/loader/ground-boom/aerial applicator

and flagger was calculated using the Pesticide Handlers Exposure Database (PHED). It was assumed that the product would be applied 6 days per year by ground-boom application to a maximum of 80 acres per day by the grower, 15 days per year by ground-boom application to a maximum of 80 acres per day by the commercial ground-boom applicator and 15 days per year to a maximum of 350 acres per day by the aerial applicator, at a maximum use rate of 28.3 grams active ingredient per acre. For purposes of this assessment, it was assumed that an applicator would be wearing a long sleeved shirt and long pants and the mixer/loader would, in addition, wear gloves. Daily doses were calculated for a person weighting 70 kg assuming 100% dermal penetration. Short-term and intermediate-term dermal and inhalation risk assessments were performed. Doses and endpoints used for risk assessments were based on Agency determined toxicological endpoints recommended by the HIARC. The NOAEL of 50 mg/kg/day from the 28–day rat dermal study was used for short- and intermediate-term dermal risk assessments. The NOAEL of 5 mg/kg/day from the developmental toxicity study in rats was used for short-term inhalation risk assessments. The NOAEL of 0.9 mg/kg/day based on a subchronic oral toxicity study in rats was used for intermediate-term inhalation risk assessments. Based on the use pattern of clodinafop-propargyl, no long-term dermal or inhalation exposure is expected to occur and long-term risk assessments are not required.

Large margins of exposure (MOEs) exist for all occupational exposure scenarios. Short-term dermal exposure MOEs ranged from 4.0E+03 for the commercial open mixer-loader to 1.8E+05 for the commercial or grower ground-boom enclosed-cab applicator. Intermediate-term dermal exposure MOEs ranged from 9.7E+04 for the commerical open mixer-loader to 1.1E+07 for the grower ground-boom enclosed-cab applicator. Short-term inhalation exposure MOEs ranged from 3.6E+04 for the commercial open mixer-loader to 1.7E+06 for the commercial or grower ground-boom enclosed-cab applicator. Intermediate-term inhalation exposure MOEs ranged from 1.6E+05 for the commercial open mixer-loader to 1.8E+07 for the grower ground-boom enclosed-cab applicator.

Although there are no residential uses of clodinafop-propargyl, there is potential for residential exposure to spray drift resulting from aerial application. No standard operating procedure exists for performing this risk assessment; however, a very conservative risk assessment was performed assuming dermal exposure equal to total deposition to outside clothing for a flagger as well as inhalation exposure equivalent to a pesticide flagger, as reflected in PHED. A dermal absorption factor of 2.5%, as estimated by HIARC, was assumed. Offsite drift was assumed to be 15% and the area assumed to be adjacent to the sensitive area was one acre. Large MOEs exist for this potential exposure scenario. Dermal exposure MOEs were 6.0E+07 for a 15 kg child and 2.8E+08 for a 70 kg adult. Inhalation MOEs were 2.3E+07 for a 15 kg child and 1.1E+8 for a 70 kg adult.

D. Cumulative Effects

A cumulative exposure assessment for effects of clodinafop-propargyl and other substances with the same mechanism of action is not appropriate because there is ample evidence to indicate that humans are not sensitive to the effects of clodinafop-propargyl and other peroxisome proliferators. Thus, the calculations outlined below were done for clodinafop-propargyl alone.

E. Safety Determination

1. *U.S. population.* Acute and chronic dietary exposure is minimal for clodinafop-propargyl and the corresponding acid metabolite. Both chronic and acute exposure estimates showed that less than 10% of the RfD is utilized in all populations.

Exposure through the consumption of drinking water is minimal from both surface water and ground water model estimates and in all cases less than 35% of the risk cup is utilized. The estimated water concentrations are very conservative since conservative model parameters were assumed.

There are no residential uses of clodinafop-propargyl that would result in non-dietary exposure. However, there is a remote possibility that spray drift resulting from aerial application could lead to residential exposure. Since exposure from spray drift would be an unlikely event, it is not appropriate to include non-dietary exposure into the aggregate assessment. Therefore, it is concluded that there is more than a reasonable certainty that no harm will result from aggregate exposure to residues of clodinafop-propargyl.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of clodinafop-propargyl, data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat have been considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from chemical exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to a chemical on the reproductive capability of mating animals and data on systemic toxicity.

Retarded fetal body weight and incomplete ossification of vertebrae and sternebrae were observed at a maternally toxic dose of 160 mg/kg/day in rats; however, no developmental toxicity of the test article was detected. Novartis believes that the NOAEL for dams and fetuses was 40 mg/kg/day. The EPA's HIARC concluded that based on an increase in bilateral distension and torsion of the ureters and delayed ossification in the fetuses, the developmental LOAEL was 40 mg/kg/day and the NOAEL was 5 mg/kg/day. Although mortality was observed in rabbit dams at dose levels of 125 and 175 mg/kg, no teratogenic or fetotoxic effects were noted. The maternal NOAEL was 25 mg/kg/day and the fetal NOAEL was 175 mg/kg/day.

Clodinafop-propargyl fed over 2-generations to rats at levels as high as 1,000 ppm did not affect mating performance, fertility, or litter sizes. Body weight was reduced in parental animals at 500 and 1,000 ppm. Physiological developmental and the survival of the pups during the last week of the lactation period were slightly reduced at levels equal to or greater than 500 ppm during the first generation only. Target organs were liver (adults) and kidney (adults and pups). The NOAEL for toxicity to the parental animals and offspring was 50 ppm, corresponding to a mean daily intake of 3.2 mg/kg bw/day of clodinafop-propargyl. The NOAEL for reproductive toxicity was 1,000 ppm (64.2 mg/kg bw/day).

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 database. Based on the current toxicological data requirements, the database relative to prenatal and postnatal effects for children is complete. The results from the 2-generation reproduction study and the rabbit developmental toxicity study would indicate there is no additional sensitivity of infants and children to clodinafop-propargyl. The HIARC selected the developmental NOAEL of 5 mg/kg/day from the rat developmental toxicity as opposed to the maternal NOAEL of 40 mg/kg bw/day. Therefore, the HIARC recommended the 10x safety

factor should be retained based on this increased susceptibility.

Using conservative exposure assumptions, dietary exposure to the most sensitive subpopulation, children (1–6 years old) utilizes 9.9% of the chronic reference dose. Chronic dietary exposure to infants (non-nursing, 1–6 years old) is 2.0% of the chronic RfD. Acute exposure for all infants and children is less than 1.0% of the acute RfD (0.62% of the RfD for the most sensitive subpopulation, children 1–6 years old). Exposure to drinking water for children (1–6 years old) utilizes 31% of the chronic RfD (surface water estimate). Children (1–6 years old) utilize 15% of the chronic RfD (surface water estimate). For acute exposure to drinking water, the worst case estimates (surface water) for infants show that only 0.57% of the aRfD is utilized and children (1–6 years old) utilize 0.27% of the aRfD. These results show that aggregate exposure to residues of clodinafop-propargyl in the diet and drinking water is negligible. Based on the completeness and reliability of the toxicity data and the conservative nature of the exposure assumptions, it is concluded that there is a more than reasonable certainty that no harm will result to infants and children from exposure to residues of clodinafop-propargyl.

F. International Tolerances

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRLs) established for residues of clodinafop-propargyl in or on raw agricultural commodities.

[FR Doc. 00–10432 Filed 4–25–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPP–66276; FRL–6552–8]

Notice of Receipt of Requests To Voluntary Cancel Certain Pesticide Registrations

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: In accordance with section 6(f)(1) of the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), as amended, EPA is issuing a notice of receipt of requests by registrants to voluntarily cancel certain pesticide registrations.

DATES: Unless a request is withdrawn, the Agency will approve these use deletions and the deletions will become effective on October 23, 2000.

FOR FURTHER INFORMATION CONTACT: By mail: James A. Hollins, Office of Pesticide Programs (7502C), Environmental Protection Agency, Ariel Rios Building, 1200 Pennsylvania Ave., NW., Washington, DC 20460. Office location for commercial courier delivery, telephone number and e-mail address: Rm. 224, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, telephone number: (703) 305–5761; e-mail: hollins.james@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 persons who produce or use 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 notice, 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'' 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*. Contact James A. Hollins at 1921 Jefferson Davis Highway, Crystal Mall #2, Rm. 224, Arlington, VA., telephone number (703) 305–5761. Available from 7:30 a.m. to 4:45 p.m., Monday thru Friday, excluding legal holidays.

II. What Action Is the Agency Taking?

This notice announces receipt by the Agency of applications from registrants to cancel some 163 pesticide products registered under section 3 or 24 of FIFRA. These registrations are listed in sequence by registration number (or company number and 24 number) in the following Table 1.

TABLE 1.—REGISTRATIONS WITH PENDING REQUESTS FOR CANCELLATION

Registration No.	Product name	Chemical name
000070–00179	Kill-Ko Seed Treater	*O,O*-Diethyl *O*-(2-isopropyl-6-methyl-4-pyrimidinyl) phosphorothioate cis-*N*-Trichloromethylthio-4-cyclohexene-1,2-dicarboximide
000070–00190	Kill-Ko Fruit Tree Spray	Methoxychlor (2,2-bis(*p*-methoxyphenyl)-1,1,1-trichloroethane) *O,O*-Dimethyl phosphorodithioate of diethyl mercaptosuccinate cis-*N*-Trichloromethylthio-4-cyclohexene-1,2-dicarboximide
000100 OR–98–0021	Supracide 25WP Insecticide-Miticide	*O,O*-Dimethyl phosphorodithioate, *S*-ester with 4-(mercaptomethyl)-2-
000100 OR–99–0022	Maxim – MZ Potato Seed Protectant	Gas cartidge (as a device for burrowing animal control) Zinc ion and manganese ethylenebisdithiocarbamate, coordination product 1*H*-Pyrrole-3-carbonitrile, 4-(2,2-difluoro-1,3-benzodioxol-4-yl)- (9CI)
000264 OR–81–0055	Rovral Fungicide	3-(3,5-Dichlorophenyl)-*N*-(1-methylethyl)-2,4-dioxo-1-imidazolidinecarboxamide
000264 WA–81–0052	Rovral Fungicide	3-(3,5-Dichlorophenyl)-*N*-(1-methylethyl)-2,4-dioxo-1-imidazolidinecarboxamide
000270–00053	Farnam Ready-To-Use Stable & Horse Fly Spray	Pine oil 2,2-Dichlorovinyl dimethyl phosphate
000270–00284	Security Brand Cygon* 2-E Systemic Insecticide	*O,O*-Dimethyl *S*-((methylcarbamoyl)methyl) phosphorodithioate
000270–00285	Security Brand Fungi-Gard	Tetrachloroisophthalonitrile
000270–00287	Security Brand Systemic Rose & Flower Booster	*O,O*-Diethyl *S*-(2-(ethylthio)ethyl) phosphorodithioate