List of Subjects

Environmental protection, Pesticides, pest, Bethoxazin, Polyxylenol teterasulfide.

Dated: January 12, 2004.

Frank Sanders,

Director, Antimicrobials Division, Office of Pesticide Programs.

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BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0409; FRL–7339–3]

Amicarbazone; 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–2003–0409, must be received on or before February 23, 2004.

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

FOR FURTHER INFORMATION CONTACT: Joanne I. Miller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–6224; e-mail address: *Miller.Joanne@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does This Action Apply to Me?

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

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

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Copies of This Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket ID number OPP–2003–0409. 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 in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

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

C. How and To Whom Do I Submit Comments?

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

is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

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

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket/*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2003–0409. 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–2003–0409. 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–2003–0409.

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–2003–0409. 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: January 12, 2004.

Lois Rossi,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

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

PP 0F6131

Arvesta Corporation

EPA has received a pesticide petition (0F6131) from Arvesta Corporation, 100 First Street, Suite 1700, San Francisco, CA 94105 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 amicarbazone (4-amino-4,5-dihydro-*N*-(1,1-dimethylethyl)-3-(1-methylethyl)-5-oxo-1H-1,2,4-triazole-1-carboxamide, DA amicarbazone *(N*-(1,1-dimethylethyl)-4,5-dihydro-3-(1-methylethyl)-5-oxo-1H-1,2,4-triazole-1-carboxamide) and iPr-2-OH DA amicarbazone *(N*-(1,1-dimethylethyl)-4,5-dihydro-3-(1-hydroxy-1-methylethyl)-5-oxo-1H-1,2,4-triazole-1-carboxamide) in or on the raw agricultural commodities corn grain, at 0.05 parts per million (ppm); corn forage at 0.8 ppm; corn stover at 0.5 ppm; alfalfa forage at 0.04 ppm; alfalfa hay at 0.06 ppm; cotton undelinted seed at 0.04 ppm; cotton gin by-product at 0.2 ppm; cottonseed meal at 0.01 ppm; cottonseed refined oil at 0.01 ppm; cottonseed hulls at 0.01 ppm; soybean forage at 2.5 ppm; soybean hay at 7.0 ppm, soybean seed at 0.6 ppm, soybean meal at 0.25 ppm; soybean hulls at 0.2 ppm; soybean oil at 0.01 ppm; wheat forage at 0.6 ppm; wheat hay at 0.9 ppm; wheat grain at 0.09 ppm; wheat straw at 0.4 ppm; wheat bran at 0.08 ppm; wheat shorts at 0.06 ppm; wheat flour at 0.05 ppm; wheat middlings at 0.05 ppm; wheat germs at 0.05 ppm; sugarcane at 0.15 ppm; sugarcane molasses at 0.8 ppm; meat (cattle, sheep, goats, horses, hogs) at 0.01 ppm; meat byproducts (cattle, sheep, goats, horses, hogs) at 0.2 ppm; and milk at 0.01 ppm respectively. 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 major metabolic pathway of amicarbazone involved the deamination of the triazole amino group followed by hydroxylation at the tertiary carbon of the isopropyl group to give iPr-2-OH DA amicarbazone. The iPr-2-OH DA amicarbazone was the major metabolite in all three corn matrices. Hydroxylation of the isopropyl methyl gave iPr-1-OH DA amicarbazone, which under went O-glucosidation. Another pathway involved hydroxylation of the t-butyl and isopropyl groups to give tBu-iPr-2-diOH DA amicarbazone. In addition, DA amicarbazone formed an glucoside. The hydroxylated DA amicarbazone formed several minor O-glucosides.

2. *Analytical method*—i. *Plant*. An analytical method was developed to determine the residues of amicarbazone in plant matrices. The method was validated in corn forage, corn fodder, corn grain, and corn processed commodities. The corn matrices were extracted with water containing 0.05% H3PO4 using a Dionex Accelerated Solvent Extractor (ASETM) at 150 °C and 1,500 psi. Following the addition of a mixture of deuterated internal standards, the material was purified by solid phase extraction (spe). The purified analytes were analyzed by high performance liquid chromatography-electrospray ionization/massspectrometry (LC-ES/MS/MS). The limit of quantitation (LOQ) of the method was 0.010 ppm. The recoveries from the various crop matrices fortified at 0.01 ppm with amicarbazone and related plant metabolites ranged from 70% to 119%. The recoveries from the various crop matrices fortified at 0.05 ppm with amicarbazone and related plant metabolites ranged from 74% to 97%. The limit of detection (LOD) in corn matrices (forage and grain) was 0.001 ppm. The LOD in corn fodder was 0.006 ppm. An alternative method was developed and validated in mustard green leaves, turnip tops, wheat forage, wheat hay, wheat grain, wheat straw, alfalfa, cotton, and soybean. The matrices were extracted in 0.1% acetic acid in acetonitrile/water (4:1), filtered and diluted using additional in acetonitrile/water (4:1). An aliquot of the extract was purified by solid-phase extraction and concentrated to an aqueous remainder. Methanol was added and the extract diluted with aqueous 5 mM ammonium bicarbonate. The samples were analyzed using LC-MS/MS in positive-ion selected reaction monitoring (+SRM) mode and quantified using a known amount of deuterated internal standard which was added to the initial sample extract.

The LOQ of the method was 0.010 ppm. The recoveries from the various crop matrices fortified at 0.01 ppm with amicarbazone and related plant metabolites ranged from 79% to 104%. The recoveries from the various crop matrices fortified at 0.10 ppm with amicarbazone and related plant metabolites ranged from 106% to 119%. The LOD in matrices ranged from 0.0011 to 0.0097 ppm.

ii. *Animal*. An analytical method was developed to measure the residues of amicarbazone in cattle tissue and milk. The amicarbazone residues were extracted from the tissue samples by accelerated solvent extraction (ASE). The extract was treated with potassium permanganate which oxidized the residues of interest to a common moiety, iPr-2-OH DA amicarbazone. The iPr-2-OH DA amicarbazone was removed from the reaction mixture by using C-18 solid-phase extraction (spe). The isolated analyte was detected by liquid chromatography/tandem mass spectroscopy (lc/ms/ms) and quantified against a known amount of a deuterated internal standard. Recoveries of a mixture of amicarbazone, DA amicarbazone, and iPr-2-OH DA amicarbazone from all tissues and milk (0.010 ppm and 0.100 ppm fortifications) were measured. For animal matrices, recoveries of 0.010 ppm of the amicarbazone component mixture ranged from 62% to 93%. The recoveries of 0.100 ppm of the amicarbazone component mixture from animal matrices ranged from 69% to 87%. For milk, the recoveries of 0. 010 ppm and 0.100 ppm of the amicarbazone component mixture ranged from 79% to 103%. The method LOQ is 0.010 ppm. The method LOD is 0.005 ppm.

3. *Magnitude of residues*. A total of 24 field trials were conducted in two different growing seasons to evaluate the quantity of amicarbazone residues in corn forage, fodder, and grain following a single application of amicarbazone. The residues of amicarbazone and two metabolites DA and iPr-2-OH DA, were quantitated by lc/ms/ms. The LOQ was 0.01 ppm for all RACs. The highest average field trial amicarbazone residues in corn were 0.55 ppm in forage, 0.43 ppm in fodder, and 0.02 ppm in grain. In decline trials, amicarbazone residues did not vary appreciably with time. Twelve trials each for alfalfa and cotton and 20 trials each for soybean and wheat were conducted to evaluate the quantity of amicarbazone residues in these rotational crops, following plant back intervals of 0 month (wheat), 1 month (soybean), 4 months (alfalfa, and 12 months (cotton). The LOQ was 0.01 ppm for all RACs. The highest average field trial amicarbazone residues were 0.02 and 0.04 ppm (alfalfa forage and hay), 0.03 and 0.16 ppm (cotton undelinted seed and gin byproduct), and 1.18, 4.35 and 0.57 ppm (soybean forage, hay and seed), 0.47, 0.87, 0.07, and 0.39 (wheat forage, hay, grain and straw).

One field trial was conducted to evaluate the quantity of amicarbazone residues in sugarcane, molasses, bagasse, and refined sugar in support of an import tolerance. Following an application at 5x the maximum expected rate for amicarbazone on sugarcane, the highest average field trial amicarbazone residues in sugarcane

were 0.11 ppm in cane, 0.78 ppm in molasses, 0.44 ppm in bagasse, and <0.01 ppm in refined sugar.

A processing study was conducted to evaluate the quantity of amicarbazone in corn processed products. The residues of amicarbazone and two metabolites DA and iPr-2-OH DA, were quantitated by LC/MS/MS. The LOQ was 0.01 ppm for corn grain and all corn processed commodities. Total amicarbazone residues in corn grain were <0.01 ppm. Except for the residue (0.01 ppm) in meal, which showed a slight concentration (1.1x), amicarbazone residues in all other processed commodities (starch, grits, flour, and refined oil) were less than the residue in corn grain.

Processing studies on the rotational crops cottonseed, soybean, and wheat were also conducted following an application at 5x (cottonseed) or 1x (soybean and wheat) the maximum expected labeled rate on corn. Total amicarbazone residues in all cotton seed fractions (meal refined oil and hulls) were <0.01 ppm. Amicarbazone residues in soybean grain hulls and deodorized oil were less than the residue in soybean grain. The residues in soybean grain meal (0.21 ppm), showed a slight concentration (1.2x). Amicarbazone residues in wheat grain flour, middlings and germs were less than the residue in grain. The residue in wheat bran (0.06 ppm) and shorts (0.05 ppm), showed a slight concentration (1.5x and 1.2x, respectively).

B. Toxicological Profile

1. *Acute toxicity*—i. Amicarbazone is minimally toxic to fasted rats following a single oral administration. The oral LD_{50} is 1,300 and 1,015 milligrams/kilogram body weight (mg/kg/bwt) for males and females, respectively.

ii. Amicarbazone is minimally toxic to rats following a single dermal application. The dermal LD_{50} is >2,000 mg/kg for both males and females.

iii. An acute inhalation study with rats demonstrated minimal toxicity following a 4–hour exposure to the test compound as a respirable dust. The inhalation LC_{50} is >2.242 mg/L for both males and females.

iv. A primary eye irritation study in rabbits showed no positive ocular effects, and only very slight, reversible irritation.

v. A dermal irritation study in rabbits showed that amicarbazone is not irritating to the skin.

vi. Amicarbazone has no skin sensitizing potential under the conditions of the buehler topical closed-patch technique in guinea pigs.

2. *Genotoxicity*. The genotoxic potential of amicarbazone was studied in bacterium and mammalian cells with the aid of various *in vitro* test systems (*salmonella* microsome test, hypoxanthine guanine phophoribosyl transferase (HGPRT) test with Chinese Hamster V79 cells, and a cytogenetic study with Chinese hamster V79 cells) and one *in vivo* test (micronucleus test). None of the tests revealed any evidence of a mutagenic or genotoxic potential of amicarbazone. The compound did not induce point mutation, DNA damage, or chromosome aberration.

3. *Reproductive and developmental toxicity*—i. In a two-generation reproduction toxicity study, Sprague-Dawley rats were administered dietary levels of amicarbazone at levels of 0, 100, 500, and 1,000 ppm. The no observed adverse effect levels (NOAELs) for reproductive parameters was established at 100 ppm (equivalent to 7 mg amicarbazone/kg/ bwt day) based on a decrease in pup weight at 500 and 1,000 ppm. The systemic NOAELs established for both parental males and females was 100 ppm based on decreased food consumption, decreased body weight, and increased liver/body weight observed in the 1,000 ppm group and to a lesser extent in the 500 ppm group.

ii. Two developmental toxicity studies were conducted with amicarbazone in the Sprague-Dawley rat. In the first study, gravid dams were administered 0, 15, 100, or 300 mg/kg bwt/day on days 6 through 19 of gestation. Maternal effects were observed at the 100 and 300 mg/kg dose levels, and included decreased food consumption, decreased body weight, and increased liver weight. No test compound-related maternal effects were noted in the 15 mg/kg dose group. An increase in nonviable fetuses and decreased fetal weight were observed in the 300 mg/kg dose level, and an increase in fetal skeletal variations was noted in the 100 and 300 mg/kg dose groups. A supplemental study was conducted to substantiate the developmental NOAEL of 15 mg/kg from the initial study. In the subsequent study gravid Sprague-Dawley rats were administered amicarbazone at 0, 5, and 15 mg/kg bwt/day on gestation days 6 through 19. No test compound-related maternal or developmental effects were observed at any dose level. Based on the findings from both rodent studies, there is no teratogenic potential for amicarbazone in the rat, and both the maternal and developmental NOAELs were established at 15 mg/kg bwt/day.

iii. Two developmental toxicity studies were conducted with amicarbazone in the himalayan rabbit. In the first study, gravid does were administered 0, 5, 20, or 70 mg/kg bwt/day on gestation days 6 through 28 post-coitum. A maternal NOAEL of 5 mg/kg bwt/day was established based on decreased body weight at dose levels of 20 and 70 mg/kg bwt/day. The NOAEL for developmental parameters was 20 mg/kg bwt/day based on a marginal decrease in fetal weight and a corresponding marginal effect on fetal skeletal ossification. A supplemental developmental toxicity study was conducted in the rabbit to confirm the absence of treatment-related malformations. In this study gravid does were administered amicarbazone at 0 or 70 mg/kg bwt/day on gestation days 6 through 28. Decreased feed consumption, decreased water consumption, and decreased body weight were observed (as in the first study) in the 70 mg/kg bwt/day group. Also as noted in the previous study, fetal weight was decreased and an accompanying effect on fetal skeletal ossification was observed. Based on the findings from both rabbit studies, there is no teratogenic potential for amicarbazone in the rabbit, and thematernal and developmental NOAELs are 5 and 20 mg/kg bwt/day, respectively.

4. *Subchronic toxicity*—i. A subchronic dermal toxicity study was conducted in the Sprague-Dawley rat in which doses of 0, 200, 500, or 1,000 mg/kg bwt/day were applied to males (22 days) and females (21 days). There were no effects at any dose level. The NOAEL was 1,000 mg/kg bwt/day (the limit dose for this study type).

ii. A 90–day feeding study was conducted in which Fischer 344 rats were exposed to 0,100, 250, 500, 1,000, 2,500, or 5,000 ppm amicarbazone in the diet for 13 weeks. Body weight gain was reduced at dietary levels of 1,000 ppm and greater in both males and females. Hematology and clinical chemistry parameters were affected in the males and females of the 1,000, 2,500, and 5,000 ppm groups. No gross pathological alterations were described in any group. Through approximately 13 weeks of continuous and repeated dietary exposure to amicarbazone, the toxicological response of the rat could be broadly characterized as involving structural and/or functional alterations in liver-, thyroid-, pancreatic-, and hematologic-related (spleen and bone marrow) endpoints. There were no adverse compound-related effects in the various parameters associated with these target organs at doses up to and including 500 ppm (equivalent to 33 mg

amicarbazone/kg bwt/day) in both the males and females.

iii. In a dose range-finding toxicity study, CD-1 mice were continuously exposed to 0, 25, 50, 100, 250, 500, 1,000, 2,500, 5,000, or 7,000 ppm amicarbazone in the diet for 6 weeks. Effects observed during the study included decreased body weight (7,000 ppm males only), affected clinical chemistry parameters (500–7,000 ppm, males and/or females, depending on endpoint), and alterations in hematology endpoints (2,500–7,000 ppm, males and/or females, depending on endpoint). Organ weight effects were limited to significantly increased liver weights, noted in both the males and females at 500 ppm and above. Compound-related histopathology included hepatocytomegaly (500–7,000 ppm), thyroid follicular cell hypertrophy (5,000–7,000 ppm), and splenic pigmentation (5,000–7,000 ppm). No effects were noted in either the males or females of the 250 ppm level.

iv. A 90–day feeding study in the dog at levels of 0, 200, 800, and 2,000 ppm amicarbazone established a NOAEL of 200 ppm (equivalent to 6.74 mg/kg bwt/day) in the males and a NOAEL of 200 ppm (equivalent to 6.28 mg/kg bwt/day) in the females. Effects observed at 2,000 ppm and to a lesser extent in the 800 ppm group included elevated liver and thyroid weights, decreased thymus weight, and affected clinical chemistry and hematology parameters. Compound-related histopathology was noted in the liver, gall bladder, and thyroid of males and/or females (depending on endpoint) of the 2,000 ppm level. The NOAEL was established in the females based on a slight induction of hepatic enzymes at the 200 ppm dietary level. In contrast affected hepatic enzymes were only observed in the males of the 800 and 2,000 ppm groups.

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, and 1,250/1,000 ppm. Decreased body weight gain was noted in the males and females of the mid and high-dose groups. Also observed in these groups were affected clinical chemistry parameters, including increased serum cholesterol (males and females) and increased thyroxine and triiodothyronine (males only). At the interim sacrifice (1–year), an increase in liver weights was observed in the males (500 and 1,200 ppm) and females (500 and 1,000 ppm). Evaluation of other organ/body weight ratios suggests that other organ weight changes were attributable to the decreases in body weight gain. Histopathological considerations included a decrease in the background incidence of hepatic vacuolation in the 1–year, 1,250 ppm, males. No other remarkable histopathology findings were noted and no evidence of any test compound-induced neoplastic response was noted in any tissue examined. Through approximately 2 years of continuous and repeated dietary exposure to the test substance, the toxicological response of the rat was principally characterized by alterations in body weight gain as well as structural and/or functional alterations in liver-related endpoints. Based on the lack of an adverse compound-related effect in the liver at a dose of 50 ppm in males and females, a systemic chronic toxicity NOAEL of 2.3 mg amicarbazone/kg bwt/day was established for the rat (specifically, 2.3 and 2.7 mg amicarbazone/kg bwt/day for male and female rats, respectively).

ii. In a chronic toxicity study in the mouse, CD-1 mice were continuously exposed to 0, 100, 1,500, or 4,000 ppm amicarbazone in the diet for 18-months. Compound-related effects were limited to organ weight changes, including pronounced increases in liver weights in the 1,500 and 4,000 ppm males and females, and decreased kidney weights in 4,000 ppm males and females. Histopathological considerations included an increased incidence of splenic pigmentation in 1,500 and 4,000 ppm males and 4,000 ppm males and females as well as hepatocellular hypertrophy in all doses tested. The hypertrophy was indicative of an adaptive response by the liver to an increased need to facilitate the metabolism and excretion of an exogenously administered test substance. While the response at 100 ppm (equivalent to 16 and 18 mg/kg bwt/day for the males and females, respectively) could be characterized as a slight physiologically adaptive response, morphological evidence demonstrated an increasingly severe response at 1,500 and 4,000 ppm, suggesting that the animals had been pushed near physiological limit. There was no evidence of a compound-induced neoplastic response in any tissue examined.

iii. A 1–year feeding study in dogs at dietary levels of 0, 75, 100, 300, and 1,200 ppm amicarbazone established a NOAEL of 75 ppm for both males and females (equivalent to 1.6 and 1.8 mg/kg bwt/day for the males and females, respectively). Mild neurological signs (described as secondary neuromuscular in nature) were noted in the 1,200 ppm females: Three at 6 months and one at 12 months. No other females, and no males were affected. Clinical pathology parameters, including triglyceride, cholesterol, albumin, globulin, and several hepatic enzymes,were, in general, affected in both the males and females of the 1,200 ppm group, to a lesser extent in the 300 ppm group, and in some cases in the 100 ppm group. Hematology parameters, including platelets, hemoglobin, hematocrit, and eosinophils, were affected primarily in the 1,200 ppm group, and to a lesser extent in the 300 and 100 ppm groups. Terminal body weight was unaffected by treatment and there were no gross lesions ascribed to the test compound. Compound-related effects on organ weights were limited to the liver and thymus. Relative and absolute liver weights were increased in the 300 and 1,200 ppm males and the 1,200 ppm females, and absolute and relative thymus weights were decreased in the 1,200 ppm males. Compound-related micropathology lesions were limited to minimal to slight diffuse thymic a trophy in all 1,200 ppm males and one 1,200 ppm female. There was no evidence of a compound-induced neoplastic response in any tissue examined.

6. *Animal metabolism*. In a metabolism and disposition study, amicarbazone (MKH 3586); (4-amino-4,5-dihydro-*N*-(1,1-dimethylethyl)-3-(1-methylethyl)-5-oxo-H-1,2,4-triazole-1-carboxamide), was administered as a single oral dose, 5 mg/kg/bwt, to four male Fischer rats. The test compound was radio-labeled at the 3-position of the triazolinone ring. After oral administration to rats, triazolinone-3-14C amicarbazone was rapidly absorbed and metabolized. Recovered radioactivity ranged from 88% to 95% of the theoretical dose. The majority (54% to 68%) of the radioactive residue was excreted in the urine, and the remainder (20% to 38%) of the radioactive residue was excreted in feces. No appreciable portion of the TRR was found in the tissues, residual carcass, or respired gases. A total of 17 metabolites arising from amicarbazone were detected in excreta; 10 metabolites could be identified. Approximately 73% of the dose was identified in the urine and feces. All individual metabolites representing >1% of the dose were identified. All unidentified residues in excreta were characterized. The main pathways for degradation and excretion of amicarbazone were direct conjugation with glucuronic acid to form amicarbazone-GA, an *N*-glucuronide, which was excreted mainly in the feces and deamination to form DA amicarbazone with subsequent

oxidation to form a variety of hydroxylated metabolites which were excreted in the urine.

7. *Metabolite toxicology—i.* Amicarbazone-triazolinone was tested for eye and dermal irritation, skin sensitization, and mutagenicity. In an acute eye irritation study in the rabbit, the test compound demonstrated corneal opacity (grade 1) in all animals at 1 and 24–hours with one animal demonstrating effects up to 4 days following exposure. No effects on the iris or conjunctiva were observed. The results of a dermal irritation study in the rabbit indicate that the test compound is not a dermal irritant. The guinea pig maximization test was utilized to test the skin sensitization potential of the test compound. No dermal effects were noted following the challenge dose indicating that the test compound exhibits no skin-sensitization potential. Mutagenicity was investigated using the *salmonella*/microsome plate incorporation test. Following incubation with five *salmonella* typhimurium LT2 mutants, no evidence of mutagenic activity of the test compound was seen.

ii. In a similar battery of tests, amicarbazone-oxadiazolinone was evaluated as above. In the eye irritation study corneal opacity and irritation to the iris were observed up to 21 days after treatment. The conjunctiva were not affected by instillation of the test compound. Dermal irritation was observed up to 24-hours following exposure to the test compound. Based on the findings of the guinea pig maximization test, the test compound does not exhibit skin sensitizing properties. Similarly, the test compound did not demonstrate any mutagenic potential following evaluation using the *salmonella*/microsome plate incorporation test.

8. *Endocrine disruption*. There is no evidence to suggest that amicarbazone has an effect on the endocrine system. Studies in this database include evaluation of the potential effects on reproduction and neonatal 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 amicarbazone.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Estimates of chronic dietary exposure to residues of amicarbazone utilized the proposed tolerances in corn forage, corn grain, meat, meat byproducts, fat and milk (of cattle, sheep, goats, horses, hogs) of 0.8, 0.05, 0.01, 0.2, 0.01 and 0.01 ppm respectively. The assumption was made that 7% of the target crop would be treated with amicarbazone. Processing factors were used in estimating the residue levels of amicarbazone in processed commodities. Potential secondary residues in livestock tissues and milk were calculated by multiplying the tissue-to-feed ratios determined in the cattle feeding study by a calculated dietary burden based on actual field residue data. Potential exposures from field rotational crops were considered negligible compared to the above-mentioned exposures. For chronic exposures, an reference dose (RfD) of 0.016 mg/kg/day was assumed based on and NOAEL of 1.6 mg/kg bwt/day from the chronic toxicity feeding study in dogs. A safety factor of 100 was used based on interspecies extrapolation (10x) and intraspecies variability (10x). Using these assumptions, dietary residues of amicarbazone contribute 0.000000 mg/kg/day (0.0% of the RfD for children 1 to 6 years old, and for the U.S. population. For acute dietary exposure, the same assumptions were made. A NOAEL of 5 mg/kg bwt/day from the behavioral and physiological toxicity study in rats with a safety factor of 100 was used in the acute dietary assessment. The safety factor of 100 was based on interspecies extrapolation (10x) and intraspecies variability (10x) and the acute (aRfD) was 0.05 mg/kg bwt/day. At the 95th percentile for the U.S. population, amicarbazone contributes 0.000023 mg/kg bwt/day (0.05% of the aRfD) toward the RfD. For children 1 to 6 years old (the most sensitive subpopulation) amicarbazone contributes 0.000042 mg/kg bwt/day (0.08% of the aRfD) toward the aRfD.

ii. *Drinking water*. The Tier I screening models GENEEC and SCI-GROW were used to determine potential levels of human exposure from drinking water sources. Given the proposed application pattern and course soil use restriction, the risk of human exposure from ground water is predicted to be lower than that for surface water. The Tier I models predict residues of amicarbazone resulting from typical agricultural use would be higher in surface water than ground water. However, even when potential surface water exposure is evaluated using the Tier I screening model GENEEC, the risk via drinking water is very low. GENEEC was used to predict an acute surface water concentration of amicarbazone of 19.8 g/L assuming a 70 kg adult drinks 2 liters of water/day containing 19.8 g/L, the acute exposure would be 5.66E-04 mg/kg/day for adults. Assuming a 10 kg child drinks 1 liter/day containing 19.8 g/L, the exposure would be 1.98E-03 mg/kg/day. Based on the NOAEL of 5 mg/kg/day from the behavioral and physiological toxicity study in rats and assuming an uncertainty factor of 100, the acute population adjusted dose (aPAD) is 0.05 mg/kg/day. Therefore, based on the contribution from drinking water alone, 1.1% of the aPAD is consumed for adults and 4.0% of the aPAD for children. At the levels calculated here, acute exposure from amicarbazone via drinking water inadults or children is far below the level of concern. GENEEC predicted a chronic (average 56–day) surface water concentration of amicarbazone to be 15.4 g/L. Assuming a 70 kg adult consumes 2 L of water per day containing 15.4 g/L amicarbazone residues for a period of 70 years, the chronic exposure would be 4.40E-04. Assuming a chronic NOAEL of 1.6 mg/kg/day from the chronic toxicity feeding study in dogs and a 100-fold safety factor, residues of amicarbazone in surface water account for less than 3.0% of the chronic population adjusted dose (cPAD) (0.016 mg/kg/day). For children (10 kg consuming 1 L/day with 15.5 g/L of amicarbazone) the same calculation translates to only 9.6% of the cPAD. Amicarbazone screening concentrations in ground water SCI-GROW were predicted to be much lower than in surface water generic expected environmental concentration (GENEEC). SCI-GROW predicted an amicarbazone concentration of less than 1 g/L at the maximum seasonal use rate. Therefore the potential contribution to human exposure from drinking water from ground water sources is even less than that from surface water. At the levels predicted by EPA's current Tier I screening models, both acute and chronic exposure from amicarbazone via drinking water in adults and children is predicted to be well below any reasonable level of concern.

2. *Non-dietary exposure*. There are no current non-food uses for amicarbazone registered under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended. No non-food uses are proposed for amicarbazone and no non-dietary exposures are expected for the general population.

D. Cumulative Effects

Amicarbazone falls into the category of triazolinone herbicides. There is no information to suggest that any members of this class of herbicides has a common mechanism of mammalian toxicity or even produce similar effects, so it is not appropriate to combine exposures of amicarbazone with other herbicides. Arvesta Corporation is considering only the potential risk of amicarbazone.

E. Safety Determination

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

2. *Infants and children.* The complete toxicological data base, including the developmental toxicity and two-generation reproduction studies were considered in assessing the potential for additional sensitivity of infants and children to residues of amicarbazone. The developmental toxicity studies in rats and rabbits did not indicate any increased sensitivity of rats or rabbits to *in-utero* exposure to amicarbazone. The two-generation reproduction study did not reveal any increased sensitivity of rats to prenatal or postnatal exposure to amicarbazone. Furthermore, none of the other toxicology studies indicated any data demonstrating that young animals were more sensitive to amicarbazone than adult animals. The data taken collectively clearly demonstrate that application of an FQPA uncertainty for increased sensitivity of infants and children is unnecessary for amicarbazone.

F. International Tolerances

Amicarbazone is registered for use on corn and sugarcane in Brazil. The tolerance for these uses in 0.02 ppm.

[FR Doc. 04–1237 Filed 1–21–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[FRL–7612–1]

Brunswick Wood Preserving Superfund Site; Notice of Proposed Settlement

AGENCY: Environmental Protection Agency.

ACTION: Notice of proposed settlement.

SUMMARY: Under section 122(h)(1) of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), the Environmental Protection Agency (EPA) has entered into an Administrative Agreement (Agreement) at the Brunswick Wood Preserving Superfund Site (Site) located in Glynn County, Brunswick, Georgia, with Kerr-McGee Chemical L.L.C. EPA will consider public comments on the Agreement until February 23, 2004. EPA may withdraw from or modify the Agreement should such comments disclose facts or considerations which indicate the Agreement is inappropriate, improper, or inadequate. Copies of the Agreement are available from: Ms. Paula V. Batchelor, U.S. Environmental Protection Agency, Region 4, Superfund Enforcement & Information Management Branch, Waste Management Division, 61 Forsyth Street, SW., Atlanta, Georgia 30303, (404) 562–8887.

Written comment may be submitted to Greg Armstrong at the above address within 30 days of the date of publication.

Dated: November 19, 2003.

Rosalind H. Brown,

Chief, Superfund Enforcement & Information Management Branch, Waste Management Division.

[FR Doc. 04–1235 Filed 1–21–04; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[FRL–7612–4]

Proposed CERCLA Section 122(h) Administrative Agreement for Recovery of Past Costs for the Morgan Materials, Inc. Superfund Site, City of Buffalo, Erie County, NY

AGENCY: Environmental Protection Agency.

ACTION: Notice; request for public comment.

SUMMARY: In accordance with section 122(i) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended (''CERCLA''), 42 U.S.C. 9622(i), notice is hereby given by the U.S. Environmental Protection Agency (''EPA''), Region II, of a proposed administrative agreement pursuant to section 122(h) of CERCLA, 42 U.S.C. 9622(h), with the settling parties, Morgan Materials, Inc. (''Morgan''), and Donald Sadkin (collectively, the ''Settling Parties''), for recovery of past response costs concerning the Morgan Materials, Inc. Superfund Site (''Site'') located in the City of Buffalo, Erie County, New York. The settlement requires payments to the EPA Hazardous Substance Superfund which total $425,000: $300,000 from Morgan, and $125,000 from Donald Sadkin. The settlement includes a covenant not to sue the Settling Parties pursuant to section 107(a) of CERCLA, 42 U.S.C. 9607(a), for EPA's past response costs. For thirty (30) days following the date of publication of this notice, EPA will receive written comments relating to the settlement. EPA will consider all comments received and may modify or withdraw its consent to the settlement if comments received disclose facts or considerations that indicate that the proposed settlement is inappropriate, improper or inadequate. EPA's response to any comments received will be available for public inspection at EPA Region II, 290 Broadway, New York, New York 10007–1866.

DATES: Comments must be submitted on or before February 23, 2004.

ADDRESSES: To request a copy of the proposed settlement agreement, please contact the individual identified below. The proposed settlement is also available for public inspection at EPA Region II offices at 290 Broadway, New York, New York 10007–1866. Comments should reference the Morgan Materials, Inc. Superfund Site, City of Buffalo, Erie County, New York, Index No. CERCLA–02–2004–2002.

FOR FURTHER INFORMATION CONTACT: Brian Carr, Assistant Regional Counsel, New York/Caribbean Superfund Branch, Office of Regional Counsel, U.S. Environmental Protection Agency, 290 Broadway—17th Floor, New York, New York 10007–1866. Telephone: 212–637–3170.

Dated: January 7, 2004.

Kathleen Callahan,

Deputy Regional Administrator, Region 2.

[FR Doc. 04–1373 Filed 1–21–04; 8:45 am]

BILLING CODE 6560–50–P

FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collection(s) Being Reviewed by the Federal Communications Commission, Comments Requested

January 15, 2004.

SUMMARY: The Federal Communications Commission, as part of its continuing effort to reduce paperwork burden invites the general public and other Federal agencies to take this opportunity to comment on the following information collection(s), as required by the Paperwork Reduction Act (PRA) of 1995, Public Law 104–13. An agency may not conduct or sponsor a collection of information unless it displays a currently valid control number. No person shall be subject to any penalty for failing to comply with a collection of information subject to the