The official public docket is the collection of materials that is available for public viewing at the EPA Docket Center, Rm. B102-Reading Room, EPA West, 1301 Constitution Ave., NW., Washington, DC. The EPA Docket Center is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The EPA Docket Center Reading Room telephone number is (202) 566–1744 and the telephone number for the OPPT Docket, which is located in EPA Docket Center, is (202) 566–0280.

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.

II. Meeting Procedures

For additional information on the scheduled meeting, the agenda of the NAC/AEGL Committee, or the submission of information on chemicals to be discussed at the meeting, contact the DFO listed under **FOR FURTHER INFORMATION CONTACT**.

The meeting of the NAC/AEGL Committee will be open to the public. Oral presentations or statements by interested parties will be limited to 10 minutes. Interested parties are encouraged to contact the DFO to schedule presentations before the NAC/AEGL Committee. Since seating for outside observers may be limited, those wishing to attend the meeting as observers are also encouraged to contact the DFO at the earliest possible date to ensure adequate seating arrangements. Inquiries regarding oral presentations and the submission of written statements or chemical-specific information should be directed to the DFO.

III. Future Meetings

Another meeting of the NAC/AEGL Committee is scheduled for September 21–23, 2004, in Washington, DC.

List of Subjects

Environmental protection, Chemicals, Hazardous substances, Health.

Dated: May 17, 2004.

Charles M. Auer,

Director, Office of Pollution Prevention and Toxics.

[FR Doc. 04–11671 Filed 5–25–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0119; FRL–7357–4]

Mepanipyrim, N-(4-methyl-6-prop-1-ynlypyrimidin-2-yl) aniline]; 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–2004–0119, must be received on or before June 25, 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: Mary Waller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–9354; e-mail address: *waller.mary@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 code 111)
- Animal Production (NAICS code 112)
- Food Manufacturing (NAICS code 311)
- Pesticide Manufacturing (NAICS code 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 identification (ID) number OPP–2004–0119. 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.1. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or on paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

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

C. How and to Whom Do I Submit Comments?

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

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

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket/*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2004–0119. 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–2004–0119. 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–2004–0119.

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–2004–0119. 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: May 6, 2004.

Lois Rossi,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner's summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by K-I Chemical U.S.A., Inc. and represents the view of the petitioner. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

K-I Chemical U.S.A., Inc.

PP 8E5017

EPA has received a pesticide petition PP 8E5017 from K-I Chemical U.S.A., Inc., 11 Martine Ave., 9th Floor, White Plains, NY, 10606 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180, by establishing import tolerances for residues of menpanipyrim N- (4-methyl-6-prop-1-ynlypyrimidin-2-yl) aniline in or on the raw agricultural commodities grape at 2.0 parts per million (ppm); grape, raisin at 4.0 ppm; strawberry at 1.5 ppm; and tomato 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 FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residues of mepanipyrim in plants is adequately understood. Metabolism studies on apples, grapes, and tomatoes have been conducted. The major residue is comprised of unchanged parent compound with small amounts of the metabolite 1 (2-anilino-6-methylpyrimidin-4-yl)-2-propanol and other metabolites. Parent compound and 1(2-anilino-6-methylpyrimidin-4-yl)-2-propanol are the only residues of concern.

2. *Analytical method*. An analytical method for measuring residues of mepanipyrim and the metabolite 1(2-anilino-6-methylpyrimidin-4-yl)-2-propanol has been submitted to EPA. The analytical method utilizes gas chromatography with a thermionic nitrogen specific detector (NPD). A confirmatory method utilizes an alternate chromatographic column. The confirmatory method is also, quantitative. These methods can be used for gathering residue data and for enforcement purposes.

3. *Magnitude of residues*. Residue field trials were conducted in representative countries that will export the majority of the treated commodities to the United States.

Grape residue field trials were conducted in Austria, France, Germany, Italy, Portugal, and Spain. Combined residues of mepanipyrim and its regulated metabolite were all less than the proposed 2.0 ppm tolerance for grapes.

Strawberry residue field trials were conducted in Belgium, France and Spain. Combined residues of mepanipyrim and its regulated metabolite were all less than the proposed 1.5 ppm tolerance.

Tomato residue field trials were conducted in Italy and Spain. Combined residues of mepanipyrim and its regulated metabolite were all less than the proposed 0.5 ppm tolerance. Grape and tomato crops both have processed commodities. Grape processed commodities are grape, juice; grape, raisin; and grape, wine. Tomato processed commodities are tomato paste and tomato puree. These processed commodities could be imported into the United States. Grape and tomato processing studies indicate that mepanipyrim residues concentrate in grape, raisin but do not concentrate in other processed commodities of grape or in the processed commodities of tomato. Tolerances are not required for grape, juice derived from mepanipyrim treated grape or from tomato, paste and tomato, puree derived from mepanipyrim treated tomato. A tolerance of 4.0 ppm is needed for the processed commodity grape, raisin.

No livestock feed items are associated with the crops for which tolerances are proposed in this petition. Therefore, no livestock residue tolerances are being proposed.

B. Toxicological Profile

1. *Acute toxicity*. Mepanipyrim has a very-low order of acute toxicity demonstrated by an acute oral LD_{50} in rats (both sexes) greater than 5,000 milligrams/kilogram/body weight (mg/kg/bwt).

2. *Genotoxicity*. A battery of *in vitro* and *in vivo* tests were conducted to determine the genotoxic potential of mepanipyrim. Mepanipyrim did not produce lethal DNA damage in three strains of *E. coli*: WP2, WP67, and CM871. Mepanipyrim was active in the Ames reverse gene mutation assay, with or without metabolic activation, employing five strains of *Salmonella typhimurium* (TA 98, TA 100, TA 1538, TA 1535, and TA 1537) and one strain of *E.coli* (WP2). Mepanipyrim did not produce unscheduled DNA synthesis in cultured human cells (HeLa S-3) either in the presence or absence of S-9 metabolic activation. *In vivo* chromosomal aberration assays (CD-1 mouse micronucleus and CD rat clastogenicity) were both negative when compared to the positive control, chlorambucil. *In vitro* chromosomal aberrations were assayed in Chinese hamster ovary (CHO-K1) cells, with and without metabolic activation S-9 mixture. Mepanipyrim did not show clastogenic activity in the activated assay; however, a questionable increase in aberrant cell frequency was produced in the non-activated assay. This increase of aberrant cell frequency occurred only

where the number of analyzable metaphases was significantly reduced. Mepanipyrim was negative in an *in vitro* specific locus gene mutation assay in cultured Chinese hamster (V79) cells as the hypoxanthine-guanine-phosphoribosyl transferase locus. In summary, mepanipyrim was not genotoxic and did not induce heritable effects in the assays conducted.

3. *Reproductive and developmental toxicity*. A developmental toxicity study was conducted in rabbits at doses of 0, 10, 30, and 90 mg/kg bwt/day. Doses of 30, and 90 mg/kg bwt/day produced marginal reductions in body weight gain and an increased incidence of premature delivery or abortion on days 28 and 29 of gestation. There was an increased percentage of small and extra small anterior fontanelle, an increased percentage of anomalous interparietal bones fissured or reduced, and an increased percentage of incompletely ossified and unossified centrales in all dosed groups. However, there was also an increased incidence of enlarged medium anterior fontanelle and posterior fontanelle in control fetuses. All indices were within the range of historical controls reported for 15 studies. In view of the percentage of variations that were evident across all groups, including controls, these sporadic increased incidences are not considered to be compound related. The developmental no adverse effects level (NOAEL) is considered to be 90 mg/kg bwt/day; the NOAEL for the study is 10 mg/kg bwt/day based on maternal toxicity at higher doses.

A developmental toxicity study was conducted in pregnant Charles River CD rats at doses of 0, 30, 150, and 750 mg/kg bwt/day, administered from day 6 though day 15 of gestation. There were no adverse effects on body weight gain, fetal growth, or morphological development at any dose. The only marginal non-dose related effects were slight increases in unilateral hydronephrosis and hydroureter at 150 and 750 mg/kg. However, these are not considered compound-related based upon the incidence in bilateral hydronephrosis and hydroureter which were increased in controls relative to all treated groups. At 750 mg/kg bwt/day there was a non-significant increase in intramuscular hemorrhage of the hind limb and subcutaneous hemorrhage of the lower jaw. The effect observed in the hind limb, although, not statistically significant, was outside the historical control range, whereas all other effects were within the historical control range of 137 studies reported. The developmental NOAEL is considered to be 750 mg/kg bwt. The NOAEL for the study is 750 mg/kg bwt/day.

A range-finding reproduction study was conducted at 200; 1,000; 2,500; and 5,000 ppm using 6 male and 6 female Charles River rats and evaluating the effects on a single litter per mating. Adult body weight gain was decreased at doses of 1,000; 2,500; and 5,000 ppm in the diet. No adverse effects on reproductive parameters were determined. A NOAEL of 200 ppm was assessed for this study.

A 2-generation reproduction study was conducted in Charles River CD rats using 28 males/females per dose. No reproductive effects were evident at doses up to and including 2,000 ppm. Liver weights were increased in parent and offspring, as well as histopathological changes at 1,000 ppm (i.e., hepatocytic fatty vacuolation). Tubular germinal epithelial degeneration was observed in F_2A and F_2B males at 1,000 ppm, with interstitial cell hyperplasia at 150 ppm. An overall NOAEL for the study was not demonstrated due to adverse effects on the liver at 150 ppm.

In a second 2-generation reproduction study in Charles River CD rats, 32 males/females were given 0, 50, or 150 ppm in the diet. The fertility index was low in control and low-dose groups (i.e., 69%), with 88% pregnant in the high dose group. All reproductive parameters which were evaluated were unaffected at all dose levels. Liver weights were increased in male and female F_1 and F_2 offspring at 150 ppm, as well as hepatocytic periacinar vacuolation in males. A NOAEL for general toxicity is considered to be 50 ppm, with 150 ppm a NOAEL for reproductive parameters.

4. *Subchronic dietary toxicity.* Short-term exposure of rats and dogs to mepanipyrim technical resulted in the following effects.

In a 13–week oral study with rats dosed at 0, 50, 100, 200, and 800 ppm, there were increased absolute and relative liver weights in both sexes. Pathological examination revealed no specific lesions. In a second 13–week dietary study in specific pathogen free rats dosed at 0; 1,600; and 4,000 ppm, decreased body weight gain was observed in both sexes at 4,000 ppm. Hematological examinations conducted at 13 weeks revealed decreased hematocrit (Hct), hemoglobin levels (Hgb), mean cell volume (MCV), and mean cell hemoglobin (MCH) in both sexes which were significantly less than controls at 4,000 ppm. Reticulocyte count, however, was increased at 4,000 ppm. There were also, significant increases in absolute and relative liver and kidney weights in both sexes at 4,000 ppm. The livers of both sexes at the 4,000 ppm level had a yellow pigment, showed fatty changes and granulation of the liver cells. The NOAEL in the 13–week oral rat studies is 13.8 mg/kg bwt/day in males and 15.3 mg/kg bwt/day in females (200 ppm).

In a 13–week oral study with mice dosed at 0; 100; 1,000; 3,000; and 7,000 ppm pathological examination revealed no abnormal gross findings in liver and kidney, although, absolute and relative liver and kidney weights were significantly increased in both sexes at 3,000 ppm. Histologic observations were limited to few organs and compound-related effects were not demonstrated. The NOAEL in the mouse is 18.8 mg/kg bwt/day in males and 22 mg/kg bwt/day for females.

In a 13–week oral study with beagle dogs dosed at 0, 15, 50, or 150 mg/kg bwt/day, body weight gain for high dose females was significantly decreased ($p<0.001$). Relative organ weight increases were observed at the highest dose, as well as alanine amino-transferase (ALT), which was increased in both sexes. Hematological examination revealed no treatment related effects. A brown pigment positively identified as lipofuscin by Schmorl's stain was seen in liver cells of both sexes at the 15 mg/kg bwt/day dose. A NOAEL was not demonstrated in this study. The study was repeated at 7.5 and 15 mg/kg bwt/day and the NOAEL was determined to be 7.5 mg/kg bwt/day based on the formation of lipofuscin in the liver.

5. *Chronic toxicity—i. Chronic toxicity/oncogenicity in rat*. Rats were administered mepanipyrim in the diet for 104 weeks at doses of 0; 50; 150; 2,000; and 4,000 ppm. Males and females at 2,000 ppm had significant decreases in body weight gain, Hct, Hgb, MCV, and MCH, also, cholesterol, triglyceride, phospholipids, and non-esterified fatty acid. Significant increase in relative and absolute liver, kidney, and spleen weights were determined in males and females at 2,000 ppm. Yellowish enlarged livers occurred in males and females at 2,000 ppm, as well as fatty changes which were increased. There was an increased incidence of transitional cell hyperplasia in kidneys of males at 2,000 ppm. The incidence of hepatocellular adenoma was significantly increased in females at the high dose. The NOAEL for the study was 50 ppm (2.45 mg/kg bwt in males and 3.07 mg/kg bwt in females).

ii. *Chronic toxicity in the dog.* Mepanipyrim was administered to dogs for 52–weeks at doses of 0, 2.5, 7.5, and 50 mg/kg bwt/day. Body weight gain was decreased in high-dose females.

Animals receiving 50 mg/kg bwt/day demonstrated significantly increased relative liver weights in both sexes and hepatocellular enlargement in females. Alkaline phosphatase (AP) and ALT were also significantly increased in high-dose male and females. Hematologic examination revealed a significant increase in neutrophils and lymphocytes manifested as a ''left-shift'' in the M:E ratio of males and females. Pigmentation in hepatocytes and Kupffer cells, identified as lipofuscin, was increased in high-dose males and females. The NOAEL for the study was 7.5 mg/kg bwt/day.

iii. *Chronic toxicity/oncogenicity in the mouse.* B6C3F1 mice were administered mepanipyrim in the diet continuously for 2 years at dose levels of 0; 70; 350, 3,500; and 7,000 ppm. Males and females showed increased relative liver weights at 3,500 ppm. Male mice also had decreased body weights at 7000 ppm. Hematocrit and hemoglobin were decreased in males at 7,000 ppm. Several effects were observed in the liver, including: Increased hepatic nodules (both sexes) at 3,500 ppm; increased swelling of liver cells in males at 3,500 ppm and in females at 7,000 ppm; and increased foci/hyperplasia in males and females at 3,500 ppm. Incidences of hepatocellular adenoma and carcinoma were increased in both sexes at 3,500 ppm. A NOAEL was demonstrated for non-neoplastic effects in both males and females at 350 ppm, equal to 56 mg/kg bwt/day in males and 68 mg/kg bwt/day in females.

Ancillary (non-good labotatory practice) studies were conducted to explore the compound-related effects on the liver in rodents.

''Studies on fatty liver induced by mepanipyrim in rats.'' Young adult Fischer 344 rats were dosed at 4,000 and 8,000 ppm for 3 weeks. Various blood and liver examinations were conducted. The results indicate that serum lipid concentrations decreased in conjunction with the induction of fatty liver by mepanipyrim treatment.

''*Study on the possible oxidative damage to DNA by mepanipyrim.*'' Mepanipyrim was administered to rats and mice in a single-oral dose, and in the diet for 3 and 6 weeks. Livers were removed at pre-determined times after each compound administration regimen, and the DNA extracted. Individual samples were assayed for 8-hydroxyquanine (8-OHdG) by high performance liquid chromatography and enzyme-linked immunosorbant assay. No significant increase in the 8-OHdG (an indicator of oxidative DNA damage) was observed in rat livers or in the 3 and 6 week exposure periods in mice.

''*Microsomal mixed function oxidase activity in liver of rats and mice administered with mepanipyrim.*'' Mepanipyrim was administered for 3 weeks to rats at dose levels of 150 and 4,000 ppm and to mice at 350 and 7,000 ppm. The study revealed that at the 4,000 and 7,000 ppm dose levels the microsomal drug-metabolizing enzyme aminopyrine N-demethylase increased slightly in the rat and mouse livers. Aniline hydroxylase activity was unchanged in both species.

''*Promoting activities of mepanipyrim liver carcinogenesis initiated with dimethylnitrosamine in rats.*'' Rats were fed a diet containing 1,000 and 5,000 ppm mepanipyrim for 6 weeks after having been injected with nitrosodiethylamine. Mepanipyrim has a weak promoting effect evidenced by the induction of gamma-glutamyl transpeptidase foci in the liver.

6. *Animal metabolism.* A rat metobolism study was conducted with 106 rats divided into 13 dose groups. No radioactivity was noted in expired CO_2 or other expired volatiles. The majority of radioactivity was excreted in the feces. Urine was the other major route of excretion. The same residues, parent and metabolites, were found in both urine and feces. Most of the radioactivity had been excreted by 24 hours after dosing. The majority of the radioactivity in blood was acetonitrile extractable at 5–8 hours after dosing and declined to zero at 120 hours. In bile duct cannulated rats, a significant amount (50–70%) of the dose was excreted in bile. The percentage of dose excreted in feces was reduced to 3–4% at 120 hours.

7. *Metabolite toxicology.* No toxicologically significant metabolites were detected in plant and rat metabolism studies.

8. *Endocrine disruption.* No specific tests have been conducted with mepanipyrim to determine whether mepanipyrim may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. There is no evidence at this time that mepanipyrim causes endocrine effects, and no reason to suspect that it does based upon the information available and mode of action of this class of compounds.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food* The Theoretical Maximum Residue Concentration (TMRC) of mepanipyrim in or on grape, strawberry, tomato, and their processed commodities (grape, juice; grape, wine; grape, raisin; tomato, paste; and tomato, puree) are as follows: 0.000936 mg/kg bodyweight/day for the general U.S. population; 0.000429 mg/kg bodyweight/day for non-nursing infants; 0.00178 mg/kg body weight/day of children 1–6 years of age; and 0.00118 mg/kg bodyweight/day of children 7–12 years of age.

The TMRC values are based on the assumption that all of the grape, strawberry, and tomato and their processed commodities will bear residues at the proposed tolerance levels for the raw agricultural commodities. These chronic dietary exposure estimates are very conservative because they assume that 100% of all grape, strawberry, and tomato are imported. Imported grapes, strawberry, and tomato actually comprise less than 10% of these commodities consumed in the United States. The estimates also assume that all imported grape, strawberry, and tomato and their processed products are treated with mepanipyrim and that residue levels on all of the imported commodities are at the proposed tolerance level.

Dietary exposure to residues of mepanipyrim will be from grape, strawberry, tomato, and their processed products and also, from grape, and wine. There are no livestock or poultry feed items associated with these raw commodities. Thus, there will be no dietary exposure to mepanipyrim residues in meat, milk, poultry, and eggs. There are no other tolerances or exemptions from a tolerance for mepanipyrim in the United States.

ii. *Drinking water.* There are neither tolerances nor registration for the use of this chemical in the United States. Therefore, there will be no exposure to mepanipyrim from residues in drinking water.

2. *Non-dietary exposure.* This petition is for a tolerance on imported grape, strawberry, and tomato. There is no approved registered use for mepanipyrim in the United States, and none is being sought. Therefore, the potential for non-dietary exposure is not pertinent to this petition.

D. Cumulative Effects

This chemical is in the anilinopyrimidine class. EPA consideration of a common mechanism of toxicity is not appropriate at this time because EPA has not made a determination that mepanipyrim and other substances may have a common mechanism of toxicity that would have a cumulative effect. K-I Chemical U.S.A., Inc., is considering only the potential risk of mepanipyrim in its cumulative-exposure assessment.

Evidence from rodent studies indicate that mepanipyrim may be oncogenic at

high doses in rodent livers. In the 2–year mouse study, at doses of 3,500 and 7,000 ppm, hepatocellular adenoma and hepatocellular carcinoma of the liver of both sexes were statistically significantly increased above those seen in the controls. A slight increase in hepatocellular adenomas was observed in female rats dosed at 4,000 ppm in the 2–year rat study. No increase was noted at lower doses or in the male rats. Additionally, the tumors did not lead to a shortening of the lifespan of affected animals and there was no decrease in the time-to-tumor versus the concurrent control animals. In the chronic toxicity portion of the rat study, there was also, the observation of hepatic perilobular lipogenesis.

A complete battery of *in vitro* and *in vivo* mutagentcity studies were performed to evaluate mepanipyrim's ability to induce gene mutations, structural chromosomal aberrations, or other genotoxic effects. Mepanipyrim showed no evidence of genotoxic activity in any of the investigations performed.

While mepanipyrim is not genotoxic, mepanipyrim demonstrated an ability to induce gamma glutamyl transferase (GGT) positive liver cell foci and to induce the liver's metabolizing enzymes. Therefore, mepanipyrim may be a non-genotoxic carcinogen suggested by its ability to induce a proliferative effect in the liver which results in increases in spontaneously occurring liver neoplasia in both mice and rats. A threshold would exist in this case and no oncogenic response would be anticipated below such a threshold level. In the current studies, no hepatocellular tumors or liver toxicity were observed in mice at 350 ppm (56.0 mg/kg/day mepanipyrim) and in rats at 50 ppm (2.45 mg/kg/day mepanipyrim).

Based on the total information examined, mepanipyrim is considered a Group C carcinogen not requiring quantitative risk assessment.

E. Safety Determination

1. *U.S. population*. The reference dose (RfD) represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. For mepanipyrim, the RfD of 0.0245 mg/kg bwt/day is based on a NOAEL of 50 ppm or 2.45 mg/kg bwt/day from the chronic toxicity/oncogenicity study. Considering the extremely conservative estimates of exposure in comparison to the RfD of 0.0245 mg/kg, the chronic dietary exposure of the U.S. population will only utilize 3.8% of the RfD. This exposure is much less than 100% of the RfD and K-I Chemical U.S.A., Inc., concludes that there is a ''reasonable certainty to no harm'' from aggregate exposure to mepanipyrim residues.

2. *Infants and children*. The chronic dietary exposure estimates will utilize approximately 1.8% of the RfD for non-nursing infants less than 1–year of age, and approximately 7.3% of the RfD for children 1–6 years of age, and approximately 4.8% for children 7–12 years of age. The conservative exposure estimates for the infant and children populations are all well below the RfD for mepanipyrim.

F. International Tolerances

Registration of mepanipyrim is in progress in the European Union (EU). A provisional registration has been granted in several countries with temporary maximum residue levels (tMRL) set. These countries and tMRLs are: Austria, strawberry and grape (2 mg/kg); Belgium, strawberry (2); France, strawberry and grape (2), wine (0.2); Italy, strawberry (2), grape (3), wine and tomato (1); Luxembourg, strawberry (2), grape (3); Netherlands, strawberry (2); Portugal, strawberry and grape (2); Spain, strawberry and grape (2), tomato (1); and United Kingdom, strawberry (2).

Mepanipyrim is registered for crop uses in Switzerland, Japan, and Israel.

[FR Doc. 04–11562 Filed 5–25–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0353; FRL–7356–1]

Di-n-propylisocinchomeronate (MGK® Repellent 326); Availability of Reregistration Eligibility Decision Document for Comment

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces availability and starts a 30–day public comment period on the Reregistration Eligibility Decision (RED) document for the insect repellent di-n-propylisocinchomeronate (MGK® Repellent 326). The RED represents EPA's formal regulatory assessment of the human health and environmental data base of the subject chemical and presents the Agency's determination regarding which pesticidal uses are eligible for reregistration.

DATES: Comments, identified by docket ID number OPP–2003–0353, must be received on or before June 25, 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: Tawanda Spears, Special Review and Reregistration Division (7508C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–8050; e-mail address: *spears.tawanda@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed to the public in general. This action may, however, be of interest to persons who are or may be required to conduct testing of chemical substances under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) or the Federal Food, Drug, and Cosmetic Act (FFDCA); environmental, human health, and agricultural advocates; pesticides users; and members of the public interested in the use of pesticides. Since other entities may also be interested, 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 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 identification (ID) number OPP–2003–0353. 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