use of the pesticide during the conditional registration period will not cause unreasonable adverse effects; and that use of the pesticide is in the public interest. The Agency has considered the available data on the risks associated with the proposed use of *N*-(2,3-dichloro-4-hydroxyphenyl)-1-methyl cyclohexanecarboxamide, and information on social, economic, and environmental benefits to be derived from such use. Specifically, the Agency has considered the nature and its pattern of use, application methods and rates, and level and extent of potential exposure. Based on these reviews, the Agency was able to make basic health and safety determinations which show that use of *N*-(2,3-dichloro-4-hydroxyphenyl)-1-methyl cyclohexanecarboxamide during the period of conditional registration will not cause any unreasonable adverse effect on the environment, and that use of the pesticide is, in the public interest.

Consistent with section 3(c)(7)(C) of FIFRA, the Agency has determined that these conditional registrations are in the public interest. Use of the pesticides are of significance to the user community, and appropriate labeling, use directions, and other measures have been taken to ensure that use of the pesticides will not result in unreasonable adverse effects to man and the environment.

III. Conditionally Approved Registrations

EPA issued a notice, published in the **Federal Register** of February 23, 1999 (64 FR 8815)(FRL–6062–1), which announced that Tomen Agro Inc., 100 First St., Suite 1610, San Francisco, CA 94105, had submitted applications to register the products Fenhexamid Technical and Elevate 50 WDG Fungicide (EPA File Symbols 66330–GA and 66330–GL) containing the active ingredient *N*-(2,3-dichloro-4-hydroxyphenyl)-1-methyl cyclohexanecarboxamide at 97.8% and 50% respectively. These products were not previously registered.

The applications were approved on May 21, 1999, for one technical and one end-use product:

1.Fenhexamid Technical for manufacturing use only; for disease control in grapes, strawberries, and ornamentals (EPA Registration Number 66330–36.

2. Elevate 50 WDG Fungicide for agricultural and horticultural use only; for use to control *Botrytis* diseases of grapes, strawberries, and ornamentals (EPA Registration Number 66330–35).

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests.

Dated: October 13, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 99–28638 Filed 11–2–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–897; FRL–6389–1]

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 initial 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–897, must be received on or before December 3, 1999.

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'' section. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–897 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: James Tompkins, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–5697; and e-mail address: tompkins.james@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	Crop production
	112	Animal production
	311	Food manufacturing
	32532	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 in the ''FOR FURTHER INFORMATION CONTACT'' section.

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–897. 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–897 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, 401 M St., SW., 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–897. 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 in the ''FOR FURTHER INFORMATION CONTACT'' section.

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: October 26, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

The petitioner summary of the pesticide petitions are 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 views of the petitioner. EPA is publishing the petition summary verbatim without editing it in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

E.I. DuPont de Nemours & Company

PP 7F4849 and 9F6039

EPA has received pesticide petitions (9F6039 and an amended petition 7F4849) from E.I. DuPont de Nemours and Company, Barley Mill Plaza, P.O. Box 80083, Wilmington, DE 19880-0038 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 a tolerance for residues of azafenidin, 2-[2,4-dichloro-5-(2-propynyloxy)phenyl]-5,6,7,8-tetrahydro-1,2,4-triazolo[4,3-a]pyridin-3(2H)-one] in or on the raw agricultural commodities (RAC) crop groupings of pome fruits at 0.02 ppm, the crop grouping stone fruits at 0.02 ppm, the crop grouping of tree nuts including pistachios at 0.02 ppm, and almond hulls at 0.5 ppm 9F6039. On December 3, 1997 (62 FR 63942) (FRL–5756–1), EPA issued a notice proposing to amend 40 CFR part 180 by establishing tolerances for residues of azafendin in or on the raw agricultural commodities (RAC) crop grouping citrus, grapes, sugarcane, and sugarcane molasses (7F4849). DuPont has amended PP 7F4849 by proposing the amend 40 CFR part 180 by establishing tolerances for residues of the herbicide azafenidn, 2-[2,4-dichloro-5-(2-propynyloxy)phenyl]-5,6,7,8-tetrahydro-1,2,4-triazolo[4.3-a]pyridin-3(2H)-one in or on the crop grouping citrus at 0.1 ppm, and the RAC citrus oil at 0.50 ppm, grapes at 0.02 ppm, sugarcane at 0.05 ppm, and sugarcane molasses 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 qualitative nature of the residues of azafenidin in pome fruit, stone fruit, and tree nuts is

adequately understood for the purposes of registration. Similar metabolic pathways were previously demonstrated in the three dissimilar crops of grapefruit, grapes, and sugarcane. The primary metabolic pathway begins with rapid O-dealkylation and production of hydroxyl derivatives, with subsequent formation of glucoside conjugates.

2. *Analytical method*. There is an independently validated practical analytical method available using gas chromatography (GC) and mass selective detection (MS) to measure levels of azafenidin in or on pome fruits, stone fruits, and tree nuts, with limits of quantitation (LOQ) that will allow for monitoring of crop residues at or above tolerance levels.

3. *Magnitude of residues*. Crop field trial residue data from pome fruit, stone fruit and tree nut studies show that the proposed tolerances on these commodities will not be exceeded when Milestone* is used as directed. Excessive application rates made to pome fruit and stone fruit in field trial residue studies demonstrated that azafenidin does not concentrate in the processed commodities of these crops.

B. Toxicological Profile

1. *Acute toxicity*. Technical azafenidin has been placed in acute toxicology category III based on overall results from several studies. Results from the following studies indicate toxicology category III: acute dermal toxicity (LD_{50} > 2,000 milligrams/kilograms (mg/kg); rabbits) and eye irritation (effects reversible within 72 hours; rabbits). Acute oral toxicity (LD_{50} > 5,000 mg/kg; rats), acute inhalation toxicity (LC_{50} > 5.4 milligrams per liter (mg/L), rats) and skin irritation (slight effects resolved within 48 hours; rabbits) results were assigned toxicology category IV. Technical azafenidin is not a dermal sensitizer.

An acute neurotoxicity study was conducted in rats administered azafenidin via gavage at 0, 100, 300, or 900 mg/kg. Azafenidin was not neurotoxic at any dose. The systemic no observed adverse effect level (NOAEL) was 100 mg/kg for males and females based on reduced food consumption and body weights at 300 mg/kg and above.

2. *Genotoxicity*. Technical azafenidin was negative for genotoxicity in a battery of *in vitro* and *in vivo* tests. These tests included the following: mutagenicity in bacterial (Ames test) and mammalian Chinese hampster ovary/hypoxanthine guanine phophoribosyl transferase (CHO/HGPRT assay) cells; *in vitro* cytogenetics (chromosomal aberration in human lymphocytes); *in vivo* cytogenetics (bone marrow micronucleus assay in mice); and unscheduled DNA synthesis (UDS) in rat primary hepatocytes.

3. *Reproductive and developmental toxicity*—i. A 2-generation reproduction study was conducted in rats with dietary technical azafenidin concentrations of 0, 5, 30, 180, or 1,080 ppm. The NOAEL was 30 ppm (1.7 to 2.8 mg/kg/day for P_1 and F_1 males and females and their offspring). This was based on the following effects at 180 ppm (10.1 to 17.8 mg/kg/day for P_1 and F_1 males and females and/or their offspring): slight reductions in mean body weights for F_1 males and females; reductions in mean gestation body weight gain and implantation efficiency; slightly increased gestation lengths; decreased offspring survival, body weights and other indices of offspring health; and increased incidence of diarrhea among F_1 parental males.

ii. A developmental study was conducted in rats administered technical azafenidin by gavage at 0, 3, 8, 16, or 24 mg/kg/day. Azafenidin was not teratogenic. The NOAEL was 16 mg/kg/day based on the following observations at 24 mg/kg/day: reduced maternal body weight, increased resorptions, reductions in litter size and fetal weights and increased sternebral variations. The maternal effects consisted of transient body weight reductions; however, the nature of these effects suggested that fetal resorptions contributed to weight reductions.

iii. A developmental study was conducted in rabbits administered technical azafenidin by gavage at 0, 12, 36, 100, or 300 mg/kg/day. Azafenidin was not teratogenic. The NOAELs for maternal and offspring toxicity were 12 and 100 mg/kg/day, respectively. The maternal NOAEL was based on reduced body weight at 36 and 100 mg/kg/day and mortality at higher doses. Excessive maternal toxicity at 300 mg/kg/day precluded assessment of developmental effects at this level. However, the developmental NOAEL was considered to be 100 mg/kg/day since there were no indications of fetal toxicity up to and including this dose level.

iv. A dermal pre-natal developmental toxicity study was conducted in rats administered technical azafenidin. The dose levels were 0, 5, 25, 50, and 100 mg/kg/day. The NOAEL was 5 mg/kg/day based on postimplantation losses with a corresponding decrease in viable litter size and fetal weight, visceral variations and increased skeletal malformations at all other dose levels. The maternal effects consisted of body weight gain reduction.

4. *Subchronic toxicity*—i. A 90-day study in mice was conducted at dietary concentrations of 0, 50, 300, 900, or 1,500 ppm. The NOAEL was 300 ppm (47.2 and 65.8 mg/kg/day for male and female mice, respectively). This was based on reduced body weight gain in males and microcytic and hypochromic anemia in males and females at 900 ppm (or 144 and 192 mg/kg/day for males and females, respectively).

ii. Technical azafenidin was administered in the diets of rats at 0, 50, 300, 900, or 1,500 ppm for 90 days. The NOAEL was 300 ppm (24.2 and 28.2 mg/kg/day for male and female rats, respectively). This was based on methemoglobinemia and microcytic and hypochromic anemia in males and females at 900 ppm (or 71.9 and 83.8 mg/kg/day for male and female rats, respectively).

iii. Dogs were administered technical azafenidin in their diets at 0, 10, 60, 120, or 240 ppm for 90 days. The NOAEL was 10 ppm (0.34 and 0.33 mg/kg/day for males and females, respectively). This was based on enlarged hepatocytes and increased serum alkaline phosphatase and alanine aminotransferase activities at 60 ppm (2.02 and 2.13 mg/kg/day for male and female dogs, respectively).

iv. A 90–day subchronic neurotoxicity study was conducted in rats at 0, 50, 750, or 1,500 ppm. There were no neurological effects observed in this study. The NOAEL for systemic toxicity was 50 ppm (3.0 mg/kg/day) and 750 ppm (54.5 mg/kg/day) for male and female rats, respectively. These were based on reduced food consumption and body weights and increased incidences of clinical signs of toxicity at the higher doses.

v. A 28–day dermal study was conducted in rats at 0, 80, 400, or 1,000 mg/kg/day. There was no dermal irritation or systemic toxicity among males or females at the highest dose tested (HDT). The NOAEL was > 1,000 mg/kg/day.

5. *Chronic toxicity*—i. An 18–month mouse study was conducted with dietary concentrations of 0, 10, 30, 300, or 900 ppm technical azafenidin. This product was not oncogenic in mice. The systemic NOAEL was 300 ppm (39.8 and 54.1 mg/kg/day for males and females, respectively). This was based on hepatotoxicity among males and reduced body weights and food efficiency among females at 900 ppm (or 122 and 163 mg/kg/day for males and females, respectively).

ii. A 2–year chronic toxicity/oncogenicity study was conducted in rats fed diets that contained 0, 5, 15, 30, 300, or 900 ppm technical azafenidin.

This product was not oncogenic in rats. The systemic NOAEL was 300 ppm (12.1 and 16.4 mg/kg/day males and females, respectively). The NOAEL was defined by microcytic, hypochromic and hemolytic anemia and mortality at 900 (or 35.2 and 50.2 mg/kg/day for male and female rats, respectively).

iii. Technical azafenidin was administered for 1–year to dogs at dietary concentrations of 0, 5, 10, 120, and 360 ppm. The NOAEL was 10 ppm (0.30 mg/kg/day for males and females). This was based on observations of altered hepatocyte morphology, hydropic degeneration and elevated alanine aminotransferase and alkaline phosphatase at 30 ppm (0.86 and 0.87 mg/kg/day for male and female dogs, respectively) and above.

6. *Animal metabolism*. The metabolism of azafenidin in animals (rat and goat) is adequately understood and is similar among the species evaluated. Azafenidin was readily absorbed following oral administration, extensively metabolized and rapidly eliminated in the urine and feces. The terminal elimination half-life in plasma was 40 hours in rats. Less than 1% of the administered dose was present in rat tissues at 120 hours. There were no volatile metabolites of azafenidin. The major metabolic pathways in the rat and goat consisted of rapid O-dealkylation and production of hydroxyl derivatives, subsequent formation of glucuronide and sulfate conjugates and elimination of these conjugates in feces and urine. There was no evidence of accumulation of azafenidin or its metabolites in the tissues of either species or in the goat's milk.

7. *Metabolite toxicology*. There is no evidence that the metabolites of azafenidin identified in animal or plant metabolism studies are of any toxicological significance. The existing metabolism studies indicate that the metabolites formed are unlikely to accumulate in humans or in animals that may be exposed to these residues in the diet. The fact that no quantifiable residues were found in edible portions of treated crops further indicates that exposures to and accumulation of metabolites are unlikely.

8. *Endocrine disruption*. No special studies investigating potential estrogenic or other endocrine effects of azafenidin have been conducted. However, the standard battery of toxicology studies required to support product registration has been completed. Studies in this battery included an evaluation of the potential effects on reproduction in the rat over 2-generations and effects on offspring development in two species. Evaluations of the pathology of the endocrine organs in subchronic and chronic studies at doses that far exceed likely human exposures have also been conducted in several species. Based on the results of these studies, the potential for azafenidin to impact the endocrine system has been adequately defined. There is no evidence to suggest that azafenidin has estrogenic properties or mimics the actions of other hormones in the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. It is proposed that azafenidin be defined as the residue for enforcement purposes. Monitoring for azafenidin residues in field samples will provide an adequate estimate of this compound in edible portions of treated crops.

i. *Food—Acute dietary exposure*. An acute dietary exposure assessment was made using the dietary exposure evaluation model (DEEM) computer software (version 6.73, Acute Module, Novigen Sciences, Inc, 1999). Acute dietary exposure was based upon the following crop uses: citrus, grapes, pome fruit, stone fruit, sugarcane, and tree nuts. Anticipated residues were estimated based on field trial data and assuming that 30% of every crop was treated. The predicted acute exposure for the U.S. population subgroup was 0.000158 mg/kg body weight day (bw/d). The population subgroup with the highest predicted level of acute exposure was the children age 1-6–year subgroup with an exposure of 0.000273 mg/kg bw/d (99.9th percentile). Based on an acute NOAEL of 16 mg/kg bw/d from an oral developmental toxicity study with rats, and a 100-fold safety factor, the acute reference dose (aRfD) would be 0.16 mg/kg bw/d. For the U.S. population the predicted exposure is equivalent to 0.10% of the aRfD. For the population subgroup children age 1-6–year, the exposure would be equivalent to 0.17% of the aRfD. Because the predicted exposures, expressed as percentages of the aRfD, are well below 100%, there is reasonable certainty that no acute effects would result from dietary exposure to azafenidin.

ii. *Chronic dietary exposure*. A chronic dietary exposure assessment was made using the DEEM computer software (version 6.74, Chronic Module, Novigen Sciences, Inc, 1999). Acute dietary exposure was based upon the following crop uses: citrus, grapes, pome fruit, stone fruit, sugarcane, and tree nuts. Anticipated residues were estimated based on field trial data and assuming that 30% of every crop was treated. The predicted chronic exposure for the U.S. population subgroup was 0.000007 mg/kg bw/d. The population subgroup with the highest predicted level of chronic exposure was the children age 1–6–year subgroup with an exposure of 0.000021 mg/kg bw/d. Based on a chronic NOAEL of 0.3 mg/kg bw/d from a 1-year chronic feeding study in dogs, and a 100-fold safety factor, the chronic reference dose (cRfD) would be 0.003 mg/kg bw/d. For the U.S. population the predicted exposure is equivalent to 0.2% of the cRfD. For the population subgroup children age 1-6–year, the exposure would be equivalent to 0.7% of the cRfD. Because the predicted exposures, expressed as percentages of the cRfD, are well below 100%, there is reasonable certainty that no chronic effects would result from dietary exposure to azafenidin.

iii. *Drinking water*. Surface water exposure was estimated using the PRZM/EXAMS models. Several USEPA standard scenarios were used (Florida citrus, Louisiana sugar cane, and New York grapes) along with standard methods for selecting input data. Ground water exposure was estimated using SCI-GROW. These are screening level models used for determining upper bound concentrations of pesticides in surface and ground water. PRZM/EXAMS and SCI-GROW use the soil/water partition coefficient , hydrolysis half life, and maximum label rate to estimate surface water concentration. The models and accompanying scenarios contain a number of very conservative underlying assumptions. Therefore, the concentrations derived from PRZM/EXAMS and SCI- GROW for drinking water are likely to be great overestimates. The predicted concentration for azafenidin in ground water under worst-case conditions was 2 parts per billion (ppb). The predicted peak concentration for azafenidin in surface water in a small non-flowing pond directly adjacent a treated citrus grove at the maximum rate was 24 ppb. The annual average concentration predicted for the same pond scenario was 4.72 ppb. EPA uses drinking water levels of comparison (DWLOCs) as a surrogate measure to capture risk associated with exposure to pesticides in drinking water. A DWLOC is the concentration of a pesticide in drinking water that would be acceptable as an upper limit in light of total aggregate exposure to that pesticide from food, water, and residential uses. A DWLOC will vary depending on the residue level in foods, the toxicity endpoint and with drinking water consumption patterns and body weights for specific subpopulations. The acute DWLOC for

azafenidin was calculated for the subpopulation of concern, children (ages 1-6 years) to be 1.6 parts per million (ppm). The estimated maximum concentration of azafenidin in surface water (24 ppb) derived from PRZM/EXAMS is much lower than the acute DWLOC. Therefore, one can conclude with reasonable certainty that residues of azafenidin in drinking water do not contribute significantly to the aggregate acute human health risk. The chronic DWLOCs are 0.1 ppm for the U.S. population and 0.03 ppm for the most sensitive subgroup, children (1-6 years). The DWLOCs are substantially higher than the PRZM/EXAMS estimated annual environmental concentration of 4.7 ppb for azafenidin in surface water. Therefore, one can conclude with reasonable certainty that residues of azafenidin in drinking water do not contribute significantly to the aggregate chronic human health risk.

2. *Non-dietary exposure*. Azafenidin is pending registration for use in weed control in selective non-food crop situations including certain temperate woody crops, and in non-crop situations including industrial sites and unimproved turf areas. Azafenidin is not to be used in on residential temperate woody plantings, or on lawns, walkways, driveways, tennis courts, golf courses, athletic fields, commercial sod operations, or other high maintenance fine turf grass areas, or similar areas. Any non-occupational exposure to azafenidin is likely to be negligible.

D. Cumulative Effects

The herbicidal activity of azafenidin is due to its inhibition of an enzyme involved with synthesis of the porphyrin precursors of chlorophyll, protoporphyrinogen oxidase. Mammals utilize this enzyme in the synthesis of heme. Although there are other herbicides that also inhibit this enzyme, there is no reliable information that would indicate or suggest that azafenidin has any toxic effects on mammals that would be cumulative with those of any other chemicals. In addition there is no valid methodology for combining the risks of adverse effects of overexposures to these compounds.

E. Safety Determination

1. *U.S. population*. Based on the completeness and reliability of the azafenidin toxicology database and using the conservative aggregate exposure assumptions presented earlier, it is concluded that azafenidin products may be used with a reasonable certainty of no harm relative to exposures from food and drinking water. The TMRC determined for the combined pending and proposed uses of azafenidin in citrus, grapes, pome fruit, stone fruit, sugar cane and tree nuts utilized only 0.2% of the cRfD (an exposure of 0.000007 mg/kg bw/d). The chronic calculated drinking water level of comparison DWLOCs of 0.1 ppm for the U.S. population is substantially higher than the PRZM/EXAMS estimated annual environmental concentration of 4.7 ppb for azafenidin. Therefore, one can conclude with reasonable certainty that chronic aggregate exposure will not exceed 100% of the cRfD. In a similar analysis of acute risk for the U.S. population, a predicted exposure of 0.000158 mg/kg bw/d, equivalent to 0.10% of the aRfD is determined. The aRfD For the U.S. population is based on an acute NOAEL of 16 mg/kg bw/d from an oral developmental toxicity study with rats, and a 100-fold safety factor. An acute DWLOC for azafenidin, calculated for the subpopulation of children (ages 1-6 yrs), was 1.6 parts per million (ppm). The estimated maximum concentration of azafenidin in water (24 ppb) derived from PRZM/EXAMS is again, much lower than this acute DWLOC. Therefore, one can conclude with reasonable certainty that residues of azafenidin in drinking water would not contribute significantly to the aggregate acute human health risk. In conclusion, there is a reasonable certainty of no harm to the general population resulting from either acute or chronic aggregate exposure to azafenidin.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of azafenidin, data from the previously discussed developmental and multigeneration reproductive toxicity studies were considered. Developmental studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during pre-natal development. Reproduction studies provide information relating to reproductive and other effects on adults and offspring from pre-natal and post-natal exposures to the pesticide. The rat reproduction and developmental studies indicated developmental effects in this species at exposures that produced minimal maternal effects. A clear dose-response and developmental NOAEL has been defined for these effects. FFDCA section 408 provides that EPA may apply an additional uncertainty factor for infants and children in the case of threshold effects to account for pre-natal and post-natal toxicity and the completeness of the database. The additional uncertainty factor may increase the margin of exposure (MOE) from the usual 100- up to 1,000-fold. Based on current toxicological data requirements, the database for azafenidin relative to pre-natal and post-natal effects for children is complete. In addition, the NOAEL of 0.3 mg/kg/day in the 1–year dog study and upon which the RfD is based is much lower than the NOAELs defined in the reproduction and developmental toxicology studies. Conservative assumptions utilized to estimate acute and chronic dietary exposures of infants and children to azafenidin demonstrated that only 0.17% of the aRfD and 0.7% of the cRfD were utilized. Chronic and acute drinking water levels of concern (DWLOC's) of 0.03 ppm and 1.6 ppm calculated for children age 1–6–years, were significantly greater than predicted chronic and acute water concentrations of 4.7 ppb and 24 ppb respectively. Based on these exposure estimates it may be concluded that there is reasonable certainty that no harm will result to infants and children from aggregate exposures to azafenidin.

F. International Tolerances

There are no established Canadian, Mexican or Codex MRLs for azafenidin. Compatibility is not a problem.

[FR Doc. 99–28728 Filed 11–2–99; 8:45 am]
BILLING CODE 6560–50–F

FEDERAL MARITIME COMMISSION

[Docket No. 99–21]

South Carolina Maritime Services, Inc. v. South Carolina State Ports Authority; Notice of Filing of Complaint and Assignment

Notice is given that a complaint was filed by South Carolina Maritime Services, Inc. (''Complainant''), against South Carolina State Ports Authority (''Respondent''). The complaint was served on October 27, 1999. Complainant alleges that Respondent violated sections 10(b)(10) and (d)(4) of the Shipping Act of 1984, 46 U.S.C. app. §§ 1709(b)(10) and (d)(4), by refusing to deal with gaming vessels and refusing to provide berthing space to Complainant for its ''cruises to nowhere'' and cruises to the Bahamas, yet providing berthing space to other vessels providing ''cruises to nowhere'' and cruises to the Bahamas.

This proceeding has been assigned to the office of Administrative Law Judges. Hearing in this matter, if any is held, shall commence within the limitations