A copy of these fact sheets, which provide a summary description of these pesticides, use patterns and formulations, science findings, and the Agency's regulatory position and rationale, may be obtained from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161.

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are available for public inspection in the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Rm. 119, CM #2, Arlington, VA 22202 (703–305–5805). Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of Information Office (A-101), 401 M St., SW., Washington, DC 20460. Such requests should: (1) Identify the product name and registration number and (2) specify the data or information desired.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: July 31, 1998.

Janet L. Andersen,

Director, Biopesticides and Pollution Prevention Division, Office of Pesticide Programs.

[FR Doc. 98–22011 Filed 8–14–98; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–819 FRL–6018–2]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–819 must be received on or before September 16, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Joanne I. Miller	Rm. #227, CM #2, 703–305–6224, e-mail:miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Cynthia Giles-Parker	Rm. #247, CM #2, 703-305-7740,e-mail:giles-parker.cynthia@epamail.epa.gov.	Do.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals 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 these petitions contain 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.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–819] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:
opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comments and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number FRL–6018–2 and appropriate petition number. Electronic comments on notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Food additives, Feed additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated:August 4, 1998.

Arnold E. Layne,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing

them 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.

1. BASF Corporation

PP 6F4640 and 3F4270

EPA has received pesticide petitions (PP 6F4640 and 3F4270) from BASF Corporation, P.O. Box 13528, Research Triangle Park, NC 27709-3528 proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of bentazon (3-isopropyl-1H-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide) and its 6- and 8-hydroxy metabolites in or on the raw agricultural commodities succulent peas at 3.0 parts per million (ppm) and flax seed at 1.0 ppm. Bentazon is currently registered for use in succulent peas with a 30-day preharvest interval (PHI) and a tolerance has been established at 0.5 ppm. The proposed increase in tolerance will allow for a reduction in the preharvest interval (PHI) to 10 days. EPA has determined that the petitions contain 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 petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residue in plants is adequately understood. Bentazon is rapidly metabolized, conjugated and incorporated into natural plant constituents. Metabolism involves the hydroxylation of bentazon at the 6- and 8-position. The terminal residues of regulatory concern are bentazon, 6-hydroxy bentazon, and 8-hydroxy bentazon (as specified in 40 CFR 180.355 (a)).

2. *Analytical method*. Adequate enforcement methods are available in the Pesticide Analytical Manual (PAM) Vol. II for the determination of residues of bentazon and its 6- and 8-hydroxy metabolites in/on plant commodities and for the determination of bentazon and AIBA metabolite in animal commodities. The methods involve quantitation by gas chromatography with flame photometric or nitrogen-specific Coulson conductivity detectors. The limit of quantitation is 0.05 ppm in animal tissues and eggs, 0.02 ppm in milk, and 0.05 ppm in plants. Residue data submitted in support of the succulent pea and flax petitions were collected using modifications of the available PAM Vol. II methods. These modified methods, along with the methods listed in PAM Vol. II are adequate for bentazon data collection and tolerance enforcement.

3. *Magnitude of residues*. Ten garden pea field trials were conducted in 7 States. Experimental plots were treated with two applications of bentazon at a rate of 1.0 lb ai/A/application. Samples of pea pods and vines were harvested from each treated plot 10 days after the second application. Samples were analyzed for the combined residues of bentazon and its 6- and 8-hydroxy metabolites. Analysis of treated samples showed that the maximum total combined residue was 2.9 ppm in pods and 26.6 ppm in vines.

Flax field trials were conducted in North Dakota (1 trial), South Dakota (2 trials), and Minnesota (1 trial). Experimental plots of flax were treated with two applications of bentazon at a rate of 1.0 lb ai/A/ application. Samples of flax seed and straw were harvested at normal maturity, resulting in a PHI range of 43 to 47 days. The maximum combined residue (bentazon and its 6- and 8-hydroxy metabolites) in flax seed samples was 0.63 ppm and in flax straw was 4.9 ppm. In the processing study, there was no concentration of residue in flax meal. In the flax petition (PP 3F4270) tolerances were proposed for the combined residue of bentazon and its 6- and 8-hydroxy metabolites in or on flax seed at 1.0 ppm and flax straw at 6.0 ppm. Since this submission was made the regulations have changed and flax straw has been removed as a raw agricultural commodity (Residue Chemistry Test Guidelines, OPPTS 860.1000, August 1996) and a tolerance is no longer required. Therefore, the tolerance statement for PP 3F4270 has been amended proposing to establish a tolerance for the combined residues of the herbicide bentazon and its metabolites in/on flax seed only. The flax straw tolerance proposal has been removed.

B. Toxicological Profile

1. *Acute toxicity*. Technical bentazon has been evaluated for acute toxicity effects. A summary of the acute toxicity studies follows:

Acute oral LD_{50} (rat)	1,100 mg/kg; M&F	Toxicity category III
Acute dermal LD_{50} (rat)	>2,500 mg/kg	Toxicity category III
Eye irritation (rabbit)	Slight irritation	Toxicity category III
Acute inhalation LC_{50} (rat)	>4.8 mg/l	Toxicity category IV
Dermal irritation (rabbit)	Minimal	Toxicity category III
Dermal sensitization (guin. pig)		Sensitizer.

2. *Genotoxicty*. Bentazon was not mutagenic in the tests for gene mutations, which were reverse mutation assays in *S. typhimurium* and in *E. coli* WP2 uvrA as well as forward mutation assays with *in vitro* Chinese hamster ovary cell (HGPRT) cultures. Bentazon was also negative in the mouse micronucleus test for assessing structural chromosomal aberrations and the unscheduled DNA synthesis assay with primary mouse hepatocytes for detecting DNA damage.

3. *Reproductive and developmental toxicity. Teratogenicity study—Rat*. In pregnant Wistar rats gavaged with 0, 40, 100, or 250 mg/kg/day of bentazon on gestation days 6-15, the maternal toxicity NOEL was over 250 mg/kg/day. The developmental toxicity NOEL was 100 mg/kg/day. The LOEL was 250 mg/kg/day based upon an increase in postimplantation loss and a reduction of fetal body weights. In addition, there was an indication of delayed skeletal ossification of phalangeal nuclei of fore- and hind-limb digits, sternebrae, and cervical vertebrae. The delayed skeletal development was considered to be due

to a delayed maturation as indicated by the decreased fetal weight at this dose.

4. *Teratogenicity study— Rabbit.* When pregnant Chinchilla rabbits were gavaged with 75, 150, or 375 mg/kg/day, on gestation days 6-18, the maternal toxicity NOEL was 150 mg/kg/day. The maternal LOEL was 375 mg/kg/day due to the occurrence in a single doe of a partial abortion, embryonic resorptions, and the absence of living fetuses. The developmental toxicity NOEL was over 375 mg/kg/day.

5. *Reproduction, 2-generation study— Rat.* A reproductive NOEL at 200 ppm (approximately 15 mg/kg/day; lowest dose tested (LDT)) was found in a 2-generation study in Wistar rats. Doses were 0, 200, 800, or 3,200 ppm bentazon in the diet. Higher levels of 800 ppm (reproductive LOEL) and 3,200 ppm (approximately 62 and 249 mg/kg/day, respectively) were associated with a decrease in the body weights of the pups during lactation. For parental toxicity, the NOEL was 800 ppm, and the LOEL was 3,200 ppm based on reductions in food consumption and weight gain, and increased incidence of renal mineralization and liver microgranuloma.

6. *Subchronic toxicity*—i. *90-day feeding study— Rat.* In a 13-week dietary feeding study in Wistar rats, the doses were 0, 400, 1,200, or 3,600 ppm in the diet. The systemic toxicity NOEL was 1,200 ppm (equivalent to 60 mg/kg/day). The LOEL was 3,600 ppm (180 mg/kg/day; highest dose tested (HDT)) based on reductions in body weight gain, increased thromboplastin and prothrombin times, diuresis, clinical chemistry changes (e.g. increases in albumin, A/G ratios, and sodium), and increased kidney and liver weights. In addition, females in the 3,600 ppm group showed suggestive evidence for the presence of lung thrombi and dilated uterine horns.

ii. *21-day dermal.* In a 21-day dermal study in rabbits, the doses were 0, 250, 500 and 1,000 mg/kg/day applied daily for 6 hours. There were no clinical signs of systemic toxicity at any dose level tested. The no adverse effect level (NOAEL) was > 1,000 mg/kg/day for male and female rabbits.

7. *Chronic toxicity*—i. *Chronic feeding study- non-rodent—Dog.* Administration of bentazon in the feed of beagle dogs for 1 year at levels of 0, 100, 400, or 1,600 ppm resulted in a systemic toxicity NOEL of 100 ppm (approximately 3.2 mg/kg/day) and a LOEL of 400 ppm (approximately 13.1 mg/kg/day). Adverse toxicological effects at the two HDT consisted of clinical signs of toxicity (emaciation, dehydration, loose and/or bloody stools, pale mucous membranes, and reduced activity), hematological changes suggestive of anemia (decreased red cells, hemoglobin and hematocrit, abnormal red cell morphology, and increased reticulocytes, platelets, leukocytes, and partial thromboplastin time), depressed body weight gains, intestinal inflammation, and congestion of the small intestine and spleen. The anemia appeared to be due to blood loss from the gastrointestinal tract.

ii. *Chronic feeding/oncogenicity study— Rat.* Fischer 344 rats were given 0, 200, 800, or 4,000 ppm bentazon in the diet in a 2-year combined chronic toxicity-carcinogenicity study. The systemic toxicity NOEL was 200 ppm, equivalent to 10 mg/kg/day LDT. Adverse effects were observed at levels of 800 ppm (40 mg/kg/day; LOEL) and 4,000 ppm (200 mg/kg/day) and consisted of increases in prothrombin time and partial thromboplastin time, increases in urine volume, blood urea nitrogen, and kidney weight along with reduced urinary specific gravity, a reduction in body weight gain, and a decrease in thyroid gland weight. No compound-related increase in tumors was observed.

iii. *Oncogenicity study— Mouse.* B6C3F1 mice were fed 0, 100, 400, or 2,000 ppm bentazon in a 2-year combined chronic toxicity-carcinogenicity study. The systemic toxicity NOEL was 100 ppm, equivalent to 15 mg/kg/day LDT. Adverse effects were observed at levels of 400 ppm (60 mg/kg/day; LOEL) and 2,000 ppm (300 mg/kg/day). There were an increased prothrombin time, calcification of the tunica albuginea of the testes, hyperplasia of pancreatic islet cells and liver, slight increase in mortality, reduced weight gain, areas of hemorrhage in the liver and heart, and increased weights of the kidney, thyroid gland, and pituitary gland. No compound- related increase in tumors was observed.

8. *Animal metabolism*. The qualitative nature of the residue in animals is adequately understood. Bentazon and its metabolite 2 amino-N-isopropylbenzamide (AIBA) are the regulated terminal residues in animal tissues, eggs and milk.

9. *Endocrine disruption*. No special studies investigating potential estrogenic or endocrine effects of bentazon have been conducted. However, the standard battery of required studies has been completed. These studies include an evaluation of the potential effects on reproduction and development, and an evaluation of the pathology exposure. These studies are generally considered to be sufficient to detect any endocrine effects but no such effects of the endocrine organs following repeated or long-term were noted in any of the studies.

10. *Neurotoxicity*. No specific neurotoxicity studies have been conducted with bentazon. However, the results of acute, subchronic and chronic studies with bentazon in different animal species did not indicate evidence of any neurotoxic potential. It is assessed as being very unlikely that bentazon would pose a specific neurotoxic hazard.

C. Aggregate Exposure

EPA has performed analyses to determine the risks from aggregate exposure to bentazon residues. For purposes of assessing the potential dietary exposure, EPA has estimated aggregate exposure based on the Theoretical Maximum Residue Contribution (TMRC) from: (i) all existing bentazon tolerances; and (ii) all existing tolerances plus the proposed increase in tolerance in succulent peas. The TMRC is a ''worst case'' estimate of dietary exposure since it is assumed that 100% of all crops for which tolerances are established are treated and that pesticide residues are at the tolerance levels.

EPA published a dietary risk assessment for bentazon based on existing uses supported through reregistration in the Reregistration Eligibility Decision (RED) for bentazon dated January 27, 1995. EPA also published an aggregate risk assessment for bentazon based on existing tolerances plus a proposed increase in tolerance in succulent peas in a final rule in the FR 33563 (FRL 5720-4) (June 20, 1997). This final rule established a time-limited tolerance for bentazon and its metabolites in/on succulent peas at 3 ppm in connection with EPA's granting an emergency exemption under section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act authorizing use of bentazon in/on succulent peas with a 10-day PHI in Minnesota, and Wisconsin. BASF used information/data from these documents and performed additional analyses in developing the following aggregate risk assessment.

1. *Dietary exposure*. The TMRC for the overall U.S. population from existing bentazon tolerances supported through reregistration is estimated at 0.000651 mg/kg bwt/day, which represents 2.2% of the RfD. The TMRC for the overall U.S. population from the existing bentazon tolerances plus the proposed increase in tolerance for succulent peas is estimated at 0.001079 mg/kg bwt/day, which represents 3.6%

of the RfD. Thus, dietary exposure to residues of bentazon in or on food from the proposed tolerance increase in succulent peas will increase the TMRC by 1.4% of the RfD for the overall U.S. population.

The TMRC from existing bentazon tolerances supported through reregistration for the most highly exposed subpopulation (non-nursing infants, <1- year old) is estimated at 0.002444 mg/kg bwt/day, which represents 8.1% of the RfD. The TMRC from the existing bentazon tolerances plus the proposed increase in tolerance for succulent peas for non-nursing infants (<1-year old) is estimated at 0.003755 mg/kg bwt/day, which represents 12.5% of the RfD. Dietary exposure to residues of bentazon in or on food from the proposed tolerance increase in succulent peas will increase the TMRC by 4.4 % of the RfD for non-nursing infants (<1-year old). These exposure assessments rely on very conservative assumptions- 100% of crops will contain bentazon residues and those residues would be at the level of the tolerance- which results in an overestimate of human exposure.

BASF believes that there will be no impact on the TMRC as a result of the use of bentazon in flax. No flax product is consumed by man as food and therefore the proposed tolerance will not directly impact the TMRC.

2. *Drinking water.* To account for the exposure from drinking water, BASF used an exposure level of 20 ppb as previously used in the final rule establishing a time-limited tolerance for bentazon and its metabolites in/on succulent peas. This is a very conservative estimate since it is unlikely that a person would be exposed to this level daily for a life-time. BASF estimates that consumption of 2 liters of water per day by a 70 kg adult at a water exposure level of 20 ppb would result in an additional consumption of approximately 2.2% of the RfD. Using these very conservative estimates for food (3.6%) and water (2.2%) results in a total of 5.8% of the RfD for the U.S. population. Thus, BASF believes that even if all the water consumed by a person over a lifetime contained bentazon at 20 ppb there would still be nearly a twenty-fold level of safety.

3. *Non-dietary exposure.* In the final rule establishing a time-limited tolerance for bentazon and its metabolites in/on succulent peas, EPA discussed short- and intermediate-term exposure. According to EPA, short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. Although residential exposure data are not available for ornamentals and ornamental turf uses of bentazon, EPA noted that large MOEs were calculated for acute aggregate risk (>= 7,000) and occupational exposure (> 6,000 for the most highly exposed group, aerial mixer loader) and that EPA believes that short- and intermediate-term aggregate risk is likely to be below EPA's level of concern.

Therefore, BASF believes that the proposed use of bentazon in succulent peas in this petition also will not exceed the EPA's level of concern for short- and intermediate exposure, since this use is identical to the section 18 use of Bentazon. BASF also believes that there will be no impact on short- and intermediate-term exposure as a result of the use of bentazon in flax since flax is a minor agricultural use with no flax product consumed by man as food.

D. Cumulative Effects

BASF has considered the potential for cumulative effects of bentazon and other substances that have a common mechanism of toxicity. BASF is unaware of any data indicating that some other active ingredient produces toxic effects by a mechanism similar to that of bentazon and that would result in cumulative toxicity. Thus, BASF is considering only the potential risks of bentazon.

E. Safety Determination

1. *U.S. population*— i. *Acute risk.* In the final rule establishing a time-limited tolerance for bentazon and its metabolites in/on succulent peas, EPA performed an acute dietary risk assessment and selected the NOEL of 100 (mg/kg/day), based on developmental effects of increased postimplantation loss and decreased fetal body weight at the LOEL of 250 mg/kg/day, from the developmental toxicity study in rats. EPA used tolerance level residues and assumed 100% crop-treated. EPA has identified women of child bearing age (females 13+ years old) as the most sensitive subpopulation. The resulting high-end exposure estimate of 0.01125 mg/kg/day, results in a dietary (food only) MOE of 8,888 for females 13+ years old which EPA considered acceptable. EPA used available monitoring data for groundwater to calculate a water exposure estimate of 3 x 10-3 mg/kg/day for adults. Adding this water exposure to the food exposure resulted in a MOE of 7,000 for females 13+ years.

In the final rule establishing a time-limited tolerance for bentazon and its metabolites in/on succulent peas the following items are noted: (a) the acute drinking water component of the risk calculations presented are relevant to subpopulations with high-end exposure within the United States (FL and CA); (b) because the calculated risk, based on high-end exposure is acceptable, the overall risk assessment is protective of the whole U.S. population; and (c) in the best scientific judgment of the Office of Pesticide Programs, the aggregate acute risk (food and water) from the currently registered uses and section 18 (succulent peas) use of bentazon does not exceed EPA's level of concern.

Therefore, BASF believes that the proposed use of bentazon in succulent peas in this petition also will not exceed the EPA's level of concern for acute exposure, since this use is identical to the section 18 use of bentazon. BASF also believes that there will be no impact on acute exposure as a result of the use of bentazon in flax. No flax product is consumed by man as food and therefore the proposed tolerance will not impact the MOE. Furthermore, flax is considered a minor crop with <100,000 acres harvested in the US in 1996. Therefore, BASF believes that the impact on groundwater exposure will be negligible as a result of bentazon use in flax and should not impact the MOE.

ii. *Short- and intermediate-term risk.* In the final rule establishing a time-limited tolerance for bentazon and its metabolites in/on succulent peas, EPA discussed short- and intermediate-term exposure. According to EPA, short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. Although residential exposure data are not available for ornamentals and ornamental turf uses of bentazon, EPA noted that large MOEs were calculated for acute aggregate risk (>= 7,000) and occupational exposure (> 6,000 for the most highly exposed group, aerial mixer loader) and that EPA believes that short- and intermediate-term aggregate risk is likely to be below EPA's level of concern.

Therefore, BASF believes that the proposed use of bentazon in succulent peas in this petition also will not exceed the EPA's level of concern for short- and intermediate exposure, since this use is identical to the section 18 use of Bentazon. BASF also believes that there will be no impact on short- and intermediate-term exposure as a result of the use of bentazon in flax since flax is a minor agricultural use with no flax product consumed by man as food.

iii. *Chronic risk.* Using the conservative TMRC exposure

assumptions described above, BASF has concluded that aggregate exposure to bentazon from food will utilize 5.8% of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is non-nursing infants which is discussed below. EPA generally has no concern for exposure below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to bentazon, including all anticipated dietary exposure and all other non-occupational exposure, BASF does not expect the aggregate exposure to exceed 100% of the RfD. BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to bentazon residues.

iv. *Cancer risk*. Bentazon was classified as a ''Group E'' carcinogen, which denotes evidence of non-carcinogenicity for humans, by the Agency's Health Effects Division Carcinogenicity Peer Review Committee, June 26, 1991.

2. *Infants and children*— i. *Developmental toxicity testing.* Developmental toxicity was observed in a developmental toxicity study using rats but was not seen in a developmental toxicity study using rabbits.

ii. *Developmental toxicity study— Rat*. From the rat developmental toxicity study, the maternal (systemic) NOEL was 250 mg/kg/day, the HDT. The developmental (fetal) NOEL was 100 mg/kg/day, based on increased postimplantation loss and decreased fetal body weight at the LOEL of 250 mg/kg/day.

iii. *Developmental toxicity study— Rabbit*. From the rabbit developmental toxicity study, the maternal (systemic) NOEL was 150 mg/kg/day, based on abortion and embryonic resorptions at the LOEL of 375 mg/kg/day. The developmental (fetal) NOEL was 375 mg/kg/day, the HDT.

iv. *Reproductive toxicity study— Rat*. From the rat reproductive study, the parental (systemic) NOEL was 62 mg/kg/day, based on increased incidences of kidney mineralization and liver microgranules at the LOEL of 249 mg/kg/day. The reproductive (pup) NOEL was 15 mg/kg/day, based on decreased body weight gain at the LOEL of 62 mg/kg/day.

v. *Pre- and post-natal sensitivity*. In the rat teratology study, fetal effects were observed at the high dose of 250 mg/kg/day in the absence of apparent maternal toxicity. However, it should be noted that very few general toxicity parameters are investigated for the maternal animals in rat teratology studies. Essentially body weight, food consumption and clinical signs are all that are determined. Bentazon typically does not produce any significant effects on these parameters at doses around 250 mg/kg/day. However, other factors indicating toxicity to adult animals were observed at a lower dose of 180 mg/kg/day in the 90-day rat feeding study. These effects consisted of increased thromboplastin and prothrombin times, diuresis, clinical chemistry changes (e.g. increases in albumin, A/G ratios, and sodium) and increased kidney and liver weights. The NOEL in this 90-day rat feeding study was determined to be 60 mg/kg/day. A conclusion can be drawn that the true NOEL for this study lies between 60 and 180 mg/kg/day. Since the effects stated above were well defined and characterized for the endpoints discussed, the data would suggest that the apparent NOEL would be in the range of 80-120 mg/kg/day. Therefore, the maternal NOEL and developmental NOEL in the rat study are similar if the same parameters are measured in the rat developmental study as are measured in the 90-day rat feeding study. Thus, since toxicity to adult animals is observed at doses which are similar to or lower than that which produced developmental toxicity, it can be concluded that bentazon does not produce selective toxicity to fetuses.

No treatment-related developmental (fetal) toxicity was observed in the rabbit teratology study despite testing to a maternally toxic level.

In the rat reproduction study, pup effects were observed at the high and mid doses of approximately 249 and 62 mg/kg/day, respectively, with parental toxicity observed at the high dose only. However, the only effect on offspring at both the mid and high doses was a slight decrease in pup weight during the lactation period. These marginal to slight differences from control were demonstrated to be transient. The F1 pups were kept on the treated diets at the mid and high dose levels after lactation. By 4 weeks of age, the F1 pup weights were the same for the mid and high doses and control. At the mid dose, there was no effect on body weight of the F1 generation animals through 123 days of treatment prior to mating.

In summary, there was no developmental toxicity observed in the rabbit teratology study, there was no selective toxicity to fetuses in the rat teratology study, and the only effect noted in the reproductive toxicity study at a dose below the parental toxicity was a slight and transient decrease in pup weight. Based on these results no additional safety factor is required for protection of infants and children.

BASF believes that the RfD used to assess safety to children should be the same as that for the general population, 0.03 mg/kg/day. Using the conservative exposure assumptions described above, BASF has concluded that the most sensitive child population is that of non-nursing infants (<1- year old). BASF calculates the exposure to bentazon residue from all existing tolerances plus the proposed increase in tolerance in succulent peas and the tolerance for flax seed to be approximately 12.5% of the RfD for non-nursing infants (< 1- year old).

F. International Tolerances

1. *Succulent peas*. There is a Codex MRL of 0.2 ppm for bentazon and its metabolites established in/on garden peas (young pods), a Canadian MRL for parent only of 0.1 ppm (negligible) established in/on peas, and a Mexican limit for parent (presumed) of 0.05 ppm established in/on green peas.

2. *Flax*. No maximum residue level (MRL) has been established for bentazon in/on flax by the Codex Alimentarius Commission. Austria has established a tolerance level for bentazon (including its hydroxy metabolites) in/on linseed (seed) of 1.5 ppm. Canada has a maximum residue level for parent only of 0.1 ppm in/on linseed. (Joanne I. Miller)

2. Novartis Crop Protection, Inc.

PP 8F4955

EPA has received a pesticide petition (PP 8F4955) from Novartis Crop Protection, Inc., PO Box 18300, Greensboro, NC 27419 proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of CGA-279202 in or on the raw agricultural commodity on pome fruit at 0.4, cucurbit vegetables at 0.25, grapes at 1.5, peanuts at 0.02, peanut hay at 4.0, apple pomace at 1.5 and imported bananas at 0.1 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of CGA-279202 in plants (cucumbers,

apples, wheat and peanuts) is well understood. Identified metabolic pathways are substantially similar in plants and animals (goat, rat and hen). Novartis proposes CGA-279202, per se, as the residue of concern for tolerance setting purposes.

2. *Analytical method*. Novartis Crop Protection Inc. has submitted practical analytical methodology for detecting and measuring levels of CGA-279202 in or on raw agricultural commodities. The limit of detection (LOD) for each analyte of this method is 0.08 ng injected, and the limit of quantitation (LOQ) is 0.02 ppm. The method is based on crop specific cleanup procedures and determination by gas chromatography with nitrogen-phosphorus detection.

3 *Magnitude of residues—Residue trials*. CGA-279202 was applied to apples in 10 States and to pears in 4 States for a total of 19 field trials. Twelve field trials were conducted in the following 8 representative peanut-growing States: Alabama, Florida, Georgia, North Carolina, Oklahoma, South Carolina, Texas, and Virginia. Eighteen cucurbit field trials in 10 States were successfully harvested, including 8 cucumber, 5 cantaloupe, and 5 summer squash field trials. Twelve field trials in 5 States, accounting for 94% of the U.S. grape production, were conducted to generate residue data on grapes, raisins, and raw and pasteurized juice. Thirteen banana field trials were conducted in Costa Rica, Ecuador, Colombia, Guatemala, Mexico, Honduras, and Puerto Rico.

B. Toxicological Profile

1. *Acute toxicity*. Studies conducted with the technical material of CGA-279202 include a rat acute oral toxicity study with a LD_{50} >5,000 mg/kg; a mouse acute oral toxicity study with a LD_{50} >5,000 mg/kg; a rabbit acute dermal toxicity study with a LD_{50} >2,000 mg/kg; a rat acute dermal toxicity study with a LD_{50} >2,000 mg/kg; a rat acute inhalation toxicity study with a LC_{50} >4.65 mg/L; a rabbit eye irritation study showing slight irritation (Category III); a rabbit dermal irritation study showing slight irritation (Category IV); a Guinea pig dermal sensitization study with the Buehler's method showing negative findings; a Guinea pig dermal sensitization study with the maximization method showing some positive findings.

2. *Genotoxicty*. No genotoxic activity is expected of CGA-279202 under *in-vivo* or physiological conditions. The compound has been tested for its potential to induce gene mutation and chromosomal changes in 5 different test systems. The only positive finding was seen in the *in vitro* test system (Chinese hamster V79 cells) as a slight increase in mutant frequency at a very narrow range (250 - 278 µg/ml) of cytotoxic and precipitating concentrations (compound solubility in water was reported to be 0.61 µg/ml; precipitate was visually noted in culture medium at 150 µg/ml). The chemical was found to be non-mutagenic in the *in vivo* system or all other *in vitro* systems. Consequently, the limited gene mutation activity in the V79 cell line is considered a nonspecific effect under non-physiological *in vitro* conditions and not indicative of a real mutagenic hazard.

3. *Reproductive and developmental toxicity*. FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database on CGA-279202 relative to pre- and post-natal effects for children is complete.

In assessing the potential for additional sensitivity of infants and children to residues of CGA-279202, Novartis considered data from teratogenicity studies in the rat and the rabbit and a 2-generation reproduction studies in the rat. The teratogenicity studies are designed to evaluate adverse effects on the developing embryo as a result of chemical exposure during the period of organogenesis. Reproduction studies provide information on effects from chemical exposure on the reproductive capability of mating animals and systemic and developmental toxicity from in-utero exposure.

In the rat teratology study, reductions in body weight gain (bwtg) and food consumption were observed in the dam at 100 mg/kg. No teratogenic effects or any other effects were seen on pregnancy or fetal parameters except for the increased incidence of enlarged thymus, which is a type of variation, at 1,000 mg/kg. The developmental NOEL was 100 mg/kg.

In the rabbit teratology study, body weight loss and dramatically reduced food consumption were observed in the dam at ≧250 mg/kg. No teratogenic effects or any other effects were seen on pregnancy or fetal parameters except for the increase in skeletal anomaly of fused sternebrae-3 and -4 at the top dose level of 500 mg/kg. This finding is regarded as a marginal effect on skeletal development that could have resulted from the 40-65% lower food intake during treatment at this dose level. The developmental NOEL was 250 mg/kg.

In the 2-generation rat reproduction study, bwtg and food consumption were decreased at ≧750 ppm, especially in females during lactation. Consequently, the reduced pup weight gain during lactation (≧750 ppm) and the slight delay in eye opening (1,500 ppm) are judged to be a secondary effect of maternal toxicity. No other fetal effects or any reproductive changes were noted. The low developmental NOEL, 50 ppm (5 mg/kg), seen in this study was probably due to the lack of intermediate dose levels between 50 and 750 ppm. Based on an evaluation of the dose-response relationship for pup weight at 750 ppm and 1,500 ppm, the NOEL should have been nearly ten-fold higher if such a dose was available.

Based on all these teratology and reproduction studies, the lowest NOEL for developmental toxicity is 5 mg/kg while the lowest NOEL in the subchronic and chronic studies is 2.5 mg/kg/day (from the rat chronic study). Therefore, no additional sensitivity for infants and children to CGA-279202 is suggested by the data base.

4. *Subchronic toxicity*. In subchronic studies, several mortality related changes were reported for the top dose in dogs (500 mg/kg) and rats (800 mg/kg). At these dose levels, excessive toxicity has resulted in body weight loss and mortality with the associated and nonspecific changes in several organs (such as atrophy in the thymus, pancreas, bone marrow, lymph node, and spleen) which are not considered specific target organs for the test compound. In the dog, specific effects were limited to hepatocellular hypertrophy at ≧150 mg/kg and hyperplasia of the epithelium of the gall bladder at 500 mg/kg. Target organ effects in the rat were noted as hepatocellular hypertrophy (≧200 mg/kg) and the related liver weight increase (≧50 mg/kg). In the mouse, target organ effects included single cell necrosis (≧300 mg/kg) and hypertrophy (1,050 mg/kg) in the liver and extramedullary hematopoiesis (≧300 mg/kg) and hemosiderosis in the spleen (1,050 mg/kg).

In general, definitive target organ toxicity, mostly in the liver, was seen at high feeding levels of over 100 mg/kg for an extended treatment period. At LOEL, no serious toxicity was observed other than mostly non-specific effects including a reduction in body weight and food consumption or liver hypertrophy.

5. *Chronic toxicity*. The liver appears to be the major primary target organ based on the chronic studies conducted in mice, rats, and dogs. It was identified as a target organ in both the mouse and

the dog studies with CGA-279202. However, no liver effect was seen in the chronic rat study which produced the lowest NOEL of 2.5 mg/kg based on reduced bwtg and food consumption seen at higher dose levels (HDL). The compound did not cause any treatment-related increase in general tumor incidence, any elevated incidence of rare tumors, or shortened time to the development of palpable or rapidly lethal tumors in the 18-month mouse and the 24-month rat studies. Dosages in both studies were sufficient for identifying a cancer risk. In the absence of carcinogenicity, Novartis believes that a Reference Dose (RfD) rapproach is appropriate for quantitation of human risks.

6. *Animal metabolism*. CGA-279202 is moderately absorbed from the gastrointestinal tract of rats and is rapidly distributed. Subsequent to a single oral dose, the half life of elimination is about 2-days and excretion is primarily via bile. CGA-279202 is extensively metabolized by the rat into about 35 metabolites, but the primary actions are on the methyl ester (hydrolysis into an acid), the methoxyimino group (O-demethylation), and the methyl side chain (oxidation to a primary alcohol). Metabolism is dose dependent as it was almost complete at low doses but only about 60% complete at high doses.

In the goat, elimination of orally administered CGA-279202 is primarily via the feces. The major residues were the parent compound and the acid metabolite (CGA-321113) plus its conjugates. In the hen, CGA-279202 is found as the major compound in tissues and in the excreta, but hydroxylation of the trifluormethyl-phenyl moiety and other transformations, including methyl ester hydrolysis and demethylation of the methoxyimino group, are also seen. In conclusion, the major pathways of metabolism in the rat, goat, and hen are the same.

7. *Metabolite toxicology*. Metabolism of CGA-279202 has been well characterized in plants, soil, and animals. In plants and soil, photolytically induced isomerization results in a few minor metabolites not seen in the rat; however, most of the applied materials remained as parent compound as shown in the apple and cucumber studies. All quantitatively major plant and/or soil metabolites were also seen in the rat. The toxicity of the major acid metabolite, CGA-321113 (formed by hydrolysis of the methyl ester), has been evaluated in cultured rat hepatocytes and found to be 20-times less cytotoxic than the parent compound. Additional toxicity studies were conducted for several minor metabolites seen uniquely in plants and/or soil. The studies indicate that these metabolites, including CGA-357261, CGA-373466, and NOA-414412, are not mutagenic to bacteria and are of low acute toxicity (LD_{50} >2,000 mg/kg). In conclusion, the metabolism and toxicity profiles support the use of an analytical enforcement method that accounts for parent CGA-279202.

8. *Endocrine disruption*. CGA-279202 does not belong to a class of chemicals known for having adverse effects on the endocrine system. Developmental toxicity studies in rats and rabbits and reproduction study in rats gave no indication that CGA-279202 might have any effects on endocrine function related to development and reproduction. The subchronic and chronic studies also showed no evidence of a long-term effect related to the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. For the purposes of assessing the potential dietary exposure under the proposed tolerances for the residue of CGA-279202 and its metabolites, Novartis has estimated aggregate exposure based upon the Theoretical Maximum Residue Concentration (TMRC). The values range from 0.0031 ppm in milk to 1.5 ppm in grapes and include tolerances for various crops; pome fruit - 0.4 ppm for the raw agricultural commodities (RAC); cucurbits - 0.25 ppm for the RAC; grapes - 1.5 ppm for the RAC; peanuts - 0.02 ppm for the RAC; banana - 0.1 ppm for the RAC. The TMRC is a ''worst case'' estimate of dietary exposure since it assumes 100% of all crops for which tolerances are established are treated and that pesticide residues are at the tolerance levels, resulting in an overestimate of human exposure.

2. *Food*—i. *Chronic*. The RfD of 0.025 mg/ kg/day is derived from the 24-month rat NOEL of 2.5 mg/kg/day. Even under worst-case assumptions, dietary exposure analysis for CGA-279202 in the most exposed population (non-nursing infants <1-year old) shows the percent RfD utilization to be only 18.9%. Although tolerances in meat and milk are not required for these uses, anticipated residues in meat and milk were also included in this exposure analysis. For average U.S. populations (48 States), dietary exposure for CGA-279202 shows a minimal utilization of 3.4% of the RfD.

ii. *Acute*. For CGA-279202, the appropriate NOEL for acute exposure is 2,000 mg/kg/day from the acute oral neurotoxicity study in rats. Acute dietary exposure analysis predicted the general population will be exposed to less than 0.0045 mg/kg/day of CGA-279202, which corresponds to a MOE of 44,237 at the 99.9 percentile. Children 1-6 years constitute the sub-population with the highest predicted exposure. Predicted acute exposure for this subgroup is less than 0.026 mg/kg/day, corresponding to a MOE of at least 7,797 for 99.9% of the individuals.

3. *Drinking water*. The potential for exposure to CGA-279202 through drinking water (surface or ground water) is low; this is due to the strong binding affinity of CGA-279202 to soil and to its low use rates (0.04-0.125 lb ai/acre/application). The highest average (56-days) surface water concentration due to runoff predicted by the GENEEC model is 0.06 ppb, resulting from application on turf. Assuming a daily water consumption rate of 2 L/day for an adult (70 kg), this would lead to an adult intake of 0.0000017 mg/kg/day which is only 0.007% of the chronic reference dose of 0.025 mg/kg/day. Assuming a three-fold increase in water consumption per unit body weight for children, the potential exposure increases only to 0.02% of RfD for this population subgroup. Estimated concentrations for treating other crops or for ground water are even lower and do not indicate any cause for concern.

4. *Non-dietary exposure*. Non-dietary exposure to CGA-279202 is considered negligible as the chemical is intended primarily for commercial and agricultural use. Exposure due to professional use on turf is considered negligible. For workers handling this chemical, acceptable margins of exposure (in the range of thousands) have been obtained for both acute and chronic scenarios.

D. Cumulative Effects

Consideration of a common mechanism of toxicity is not appropriate at this time since there is no information to indicate that toxic effects produced by CGA-279202 would be cumulative with those of any other types of chemicals. Furthermore, the oximinoacetate is a new type of fungicide and no compound in this general chemical class currently has a significant market share. Consequently, Novartis is considering only the potential exposure to CGA-279202 in its aggregate risk assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data base for CGA-279202,

Novartis has calculated aggregate exposure levels for this chemical. The calculation shows that only 3.4% of the RfD will be utilized for the U.S. population based on chronic toxicity endpoints. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Novartis concludes that there is a reasonable certainty that no harm will result from aggregate exposure to CGA-279202 residue.

2. *Infants and children*. Developmental toxicity, manifested as reduced weaning pup weight, enlarged thymus, or fused sternabrae, was observed in the teratology study and 2-generation rat reproduction studies at maternally toxic doses. All of these findings are judged to be non-specific, secondary effects of maternal toxicity. The lowest NOEL for developmental toxicity was established in the rat reproduction study at 5 mg/kg, a level that is likely to be an overly low estimate (as a result of dose gap) but is still higher than the chronic NOEL of 2.5 mg/kg on which the RfD is based. Using the same conservative exposure assumptions as employed for the determination in the general population, Novartis has calculated that the percent of the RfD that will be utilized by aggregate exposure to residues of CGA-279202 is only 19% for non-nursing infants less than 1-year old (the most impacted sub-population). Therefore, based on the completeness and reliability of the toxicity data base and the conservative exposure assessment, Novartis concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to CGA-279202 residues.

F. International Tolerances

No Codex MRLs have been established for residues of CGA-279202. (Janet Whitehurst).

[FR Doc. 98–22012 Filed 8–14–98; 8:45 am]

BILLING CODE 6560–50–F

FEDERAL EMERGENCY MANAGEMENT AGENCY

Agency Information Collection Activities: Submission for OMB Review; Comment Request

ACTION: Notice and request for comments.

SUMMARY: The Federal Emergency Management Agency has submitted the following proposed information collection to the Office of Management and Budget for review and clearance in accordance with the requirements of the Paperwork Reduction Act of 1995 (44 U.S.C. 3507).

Title: National Flood Insurance Program Biennial Report.

Type of Information Collection: Reinstatement, with change of a previously approved collection for which approval has expired.

OMB Number: 3067–0018.

Abstract: The Federal Emergency Management (FEMA) requires that communities participating in the National Flood Insurance Program submit a biennial report on progress made in local floodplain management. The use of a simple, standard format facilitates FEMA's reporting of response, thus enhancing the reports value as a management tool. The following three FEMA forms are used to collect data for the biennial report:

FEMA Form 81–28, Regular Program and Emergency Program (Minimally Floodprone). The hour burden estimate is 35 minutes per response.

FEMA Form 81–29, Regular Program (with Base Flood Elevations). The hour burden estimate is 1 hour per response.

FEMA Form 81–29A, Regular Program (No Special Flood Hazard Area). The hour burden estimate is 12 minutes per response.

Affected Public: State, Local or Tribal Government.

Number of Respondents: 9,089.

Estimated Total Annual Burden Hours: 7,546.

Frequency of Response: Biennially.

COMMENTS: Interested persons are invited to submit written comments on the proposed information collection to the Desk Officer for the Federal Emergency Management Agency, Office of Information and Regulatory Affairs, Office of Management and Budget, Washington, DC 20503 within 30 days of the date of this notice.

FOR FURTHER INFORMATION CONTACT: Requests for additional information or copies of the information collection should be made to Muriel B. Anderson, FEMA Information Collections Officer, Federal Emergency Management Agency, 500 C Street, SW, Room 311, Washington, DC 20472. Telephone number (202) 646–2625, FAX number (202) 646–3524 or email address at muriel.anderson@fema.gov.

Dated: August 10, 1998.

Reginald Trujillo,

Director, Program Services Division, Operations Support Directorate.

[FR Doc. 98–22048 Filed 8–14–98; 8:45 am]

BILLING CODE 6718–01–P

FEDERAL EMERGENCY MANAGEMENT AGENCY

[FEMA–1203–DR]

State of California; Amendment to Notice of a Major Disaster Declaration

AGENCY: Federal Emergency Management Agency (FEMA).

ACTION: Notice.

SUMMARY: This notice amends the notice of a major disaster for the State of California, (FEMA–1203–DR), dated February 9, 1998, and related determinations.

EFFECTIVE DATE: August 3, 1998.

FOR FURTHER INFORMATION CONTACT: Madge Dale, Response and Recovery Directorate, Federal Emergency Management Agency, Washington, DC 20472, (202) 646–3260.

SUPPLEMENTARY INFORMATION: The notice of a major disaster for the State of California, is hereby amended to include the following area among those areas determined to have been adversely affected by the catastrophe declared a major disaster by the President in his declaration of February 9, 1998:

Del Norte County for Individual Assistance (already designated for Public Assistance).

(The following Catalog of Federal Domestic Assistance Numbers (CFDA) are to be used for reporting and drawing funds: 83.537, Community Disaster Loans; 83.538, Cora Brown Fund Program; 83.539, Crisis Counseling; 83.540, Disaster Legal Services Program; 83.541, Disaster Unemployment Assistance (DUA); 83.542, Fire Suppression Assistance; 83.543, Individual and Family Grant (IFG) Program; 83.544, Public Assistance Grants; 83.545, Disaster Housing Program; 83.548, Hazard Mitigation Grant Program.)

Lacy E. Suiter,

Executive Associate Director, Response and Recovery Directorate.

[FR Doc. 98–22046 Filed 8–14–98; 8:45 am]

BILLING CODE 6718–02–P

FEDERAL EMERGENCY MANAGEMENT AGENCY

[FEMA–1223–DR]

Florida; Amendment No. 9 to Notice of a Major Disaster Declaration

AGENCY: Federal Emergency Management Agency (FEMA).

ACTION: Notice.

SUMMARY: This notice amends the notice of a major disaster for the State of Florida (FEMA–1223–DR), dated June 18, 1998, and related determinations.

EFFECTIVE DATE: July 22, 1998.