chemistry and there are no reliable data to indicate that this chemical is structurally or toxicologically similar to existing chemical substances at this time. Therefore, it appears unlikely that azoxystrobin bears a common mechanism of activity with other substances. For the purposes of this tolerance action, it is not appropriate to assume that azoxystrobin has a common mechanism of toxicity with other substances.

E. Safety Determination

The chronic toxicity Reference Dose (RfD) for azoxystrobin is 0.18 mg/kg/day, based on the NOEL of 18.2 mg/kg/day from the rat chronic toxicity/carcinogenicity feeding study in which decreased body weight and bile duct lesions were observed in male rats at the LOEL of 34 mg/kg/day. This NOEL was divided by an Uncertainty Factor of 100, to allow for interspecies sensitivity and intraspecies variability.

1.As part of the hazard assessment process, the available toxicological database was reviewed to determine if there are toxicological endpoints of concern. For azoxystrobin, the Agency does not have a concern for acute dietary exposure since the available data do not indicate any evidence of significant toxicity from a 1–day or single event exposure by the oral route. Therefore, an acute dietary risk assessment is not required for azoxystrobin at this time.

2. *U.S. population*. The chronic dietary exposure analysis showed that exposure from the proposed new tolerances in or on tree nuts, pistachios, cucurbits, rice, and wheat for the general U.S. population would be 1.1% of the RfD. This analysis used a value of 0.05 ppm for banana pulp rather than the value of 0.5 that has been established for banana (whole fruit including peel) because adequate data were submitted to support use of the lower value in the dietary risk analyses.

3. *Infants and children*. The chronic dietary exposure analysis, using the same tolerances and commodities that were used for the same analysis for the general U.S. population showed that the exposure of Non-nursing Infants (the subgroup with the highest exposure) would be 4.1% of the RfD.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a margin of exposure analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. In either case, EPA generally defines the level of appreciable risk as exposure that is greater than 1/100th of the no observed effect level in the animal study appropriate to the particular risk assessment. This hundredfold uncertainty (safety) factor/margin of exposure (safety) is designed to account for combined inter- and intraspecies variability. EPA believes that reliable data support using the standard hundredfold margin/factor not the additional tenfold margin/factor when EPA has a complete database under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard margin/factor. The database for azoxystrobin is complete except that the acute and subchronic neurotoxicity studies require upgrading. The upgrade data are confirmatory only, have been submitted by the company, and await review by the Agency.

There was no evidence of increased susceptibility of infants or children to azoxystrobin. Therefore, no additional uncertainty factors are considered necessary at this time.

F. Endocrine Effects

EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect...''. The Agency is currently working with interested shareholders, including other government agencies, public interest groups, industry, and research scientists, to develop a screening and testing program and a priority setting scheme to implement this program. Congress has allowed three (3) years from the passage pf FQPA (August 3, 1999) to implement this program. When this program is implemented, EPA may require further testing of azoxystrobin and end-use product formulations for endocrine disrupter effects. There are currently no data or information suggesting that azoxystrobin has any endocrine effects.

G. International Tolerances

There are no Codex Maximum Residue Levels established for azoxystrobin. (Cynthia Giles-Parker)

[FR Doc. 97–26537 Filed 10–8–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–765; FRL–5745–9]

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–765, must be received on or before November 7, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), 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
Joe Tavano	Rm. 214, CM #2, 703–305–6411, e-mail: tavano.joe@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Bipin Gandhi,	Rm. 4W53, CS #1, 703–308–8380, e-mail: gandhi.bipin@epamail.epa.gov.	2800 Crystal Drive, Arlington, VA
Eugene Wilson	Rm. 245, CM #2, 703–305–6103, e-mail: wilson.eugene@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA

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–765] (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. Comment 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 PF–765 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: September 25, 1997.

James Jones,

Actinig 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. B2E Corporation

PP 7E4907

EPA has received a pesticide petition (PP 7E4907) from B2E Corporation, 16 School Street, Rye, NY 10580 proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act,(FFDCA) 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish an exemption from the requirement of a tolerance for 2-Hydroxyacetophenone (2-HAP) in or on the raw agricultural commodity. The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using UV detection. 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. Toxicological Profile

1. *Acute toxicity*. A rat acute oral study with an LD_{50} > 500 milligrams/kilogram (mg)/(kg), a rabbit acute dermal toxicity study with an LD_{50} > 2,000 mg/kg, a primary eye irritation study in the rabbit showing no irritation, a rabbit primary dermal irritation study showing 2-HAP is not an irritant, a skin sensitization study in guinea pigs showing 2-HAP is a slight skin sensitizer, and a 28 day rat inhalation study with a no observed-effect-level (NOEL) of 160 milligrams/cubic meter (mg)/(m^3).

2. *Genotoxicty*. 2-HAP was tested in the Ames Salmonella/microsome plate incorporation assay both in the presence and the absence of a metabolic activation system. Under the conditions of the assay, 2-HAP did not exhibit genetic activity according to the assay criteria. It can therefore be considered non-mutagenic.

3. *Ecotoxicity*. A study of acute toxicity to Bluegill Sunfish was conducted at five nominal concentrations, selected on the basis of preliminary toxicity screening, as well as a control and the solvent (acetone). The fish (10 in each replicate) were observed at 24, 48, 72 and 96 hour intervals for signs of toxic effects and mortality. 2-HAP was determined to have an LC_{50} (96 hours) of 115 milligrams/liter (mg)/(L) and a no observed effect-concentration (NOEC) of 31.3 mg/L.

A study of acute toxicity to Daphnids was conducted at five nominal concentrations as well as a control and solvent (acetone) over 48 hours (hrs). They were observed at 24 and 48 hours for signs of toxic effects and mortality. 2-HAP was calculated to have an EC_{50} (48 hr) of 57 mg/L under these conditions. The NOEC was found to be 25 mg/L.

B. Environmental Fate

Aerobic soil metabolism was evaluated by a Ready Biodegradation by CO_2 Production study. The test liquid was added to test medium at 10 and 20 mg/L. Unacclimated diluted inoculum (20 ml, 1.3 million CFU. ml) was added to 2 liters of diluted test material, positive control material (glucose at 20 milligrams/milliter (mg)/(ml) or control medium. Carbon dioxide free air was bubbled through the stirred 22.6–23.2 ° C. incubation mixtures and carbon dioxide collected for 28 days. Carbon dioxide was measured by titration of barium hydroxide traps at regular intervals of the study. Percent biodegradation was estimated by percent of theoretical carbon dioxide

(TCO_2) production achieved based on the empirical formula, assuming that all organic carbon in the test material is converted to carbon dioxide, and by measurement of total organic carbon (TOC) remaining after the 28 day incubation.

After a lag of about 1 day, test material carbon dioxide production achieved 93.2% (at 10 mg/L) and 86.7% (at 20 mg/l) TCO_2 28 days after study start. The soluble organic carbon content at study termination was < 0.5 mg/L and 0.7 mg/l initial concentrations of test material respectively. This corresponds to 100% (at 10 mg/L) and 98.6% (at 20 mg/L) removal of test material also indication effective mineralization.

The 2-HAP produced greater than 60% of the TCO_2 within 28 days of incubation and can be considered readily biodegradable.

Anaerobic degradation is not expected to be a factor given the application of the product.

C. Aggregate Exposure

1. *Dietary exposure*. Dietary exposure for 2-HAP is expected to be negligible for the application of 2-HAP in non-food use pesticides. If 2-HAP were to be incorporated in pesticides used for food crops, the level of 2-HAP would be at most, a small fraction of the acceptable tolerances of the pesticides. The use level within the pesticide is only a maximum of 0.1% by weight. The rapid biodegradability make significant uptake into plant tissue unlikely. Human exposure may be expected to be within acceptable (note: FDA classifies this as a GRAS material for use in meat products, poultry, condiments, soups and seasonings) limits.

2. *Drinking water.* Although 2-HAP is not considered to be hydrolyzable, it is readily biodegradable. Use levels at a maximum of 0.1% within pesticides also make it unlikely that there will be a presence in groundwater. Based on this data, exposure to residues in drinking water in not anticipated. The EPA has not established a Maximum Concentration Level for residues of 2-HAP in drinking water.

3. *Non-dietary exposure.* Evaluations by B2E Corporation of the estimated non-occupational exposure to 2-HAP have concluded that the potential exposure for the general population may be from residues in food crops discussed above. Another possible exposure is from the use on turf of pesticides containing 2-HAP as an inert. The route of exposure would be dermal (assuming that people would be walking barefoot on treated areas) and the material has been shown to have a low order of acute dermal toxicity (rabbit - LD_{50} 10,300 mg/kg).

D. Cumulative Effects

B2E Corporation considered the potential for cumulative effects of 2-HAP and similar substances that may have a common mechanism of toxicity. there is no information to indicate that toxic effects that might be found at high levels of exposure to 2-HAP would be cumulative with other chemical compounds. The potential risks of 2-HAP are judged solely in its aggregate exposure.

E. Safety Determination

1. *U.S. population*. Based on the exposure assumptions and the toxicity data described above, there is no appreciable risk to human health. It can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to 2-HAP residues.

2. *Infants and children*. Based on the use patterns of the material and the levels of exposure, there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to 2-HAP residue.

F. International Tolerances

No international tolerances have been established.

2. Novartis Crop Protection, Inc.

PP 6F4616, 6F4617, 6F4618, & 6F4633

EPA has received a pesticide petition (PP 6F4616, 6F4617, 6F4618, & 6F4633) from Novartis Crop Protection, Inc., P.O. Box 18300, Greensboro, NC 27419–8300 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 Fenoxycarb, ethyl[2-(4-phenoxyphenoxy)ethyl]carbamate in or on the raw agricultural commodities: pome fruit at 0.02 parts per million (ppm); nutmeat at 0.05 ppm; almond hulls at 4.0 ppm; citrus fruit at 0.05 ppm; grass Forage (except Bluegrass) at 0.6 ppm; grass hay (except Bluegrass) at 0.5 ppm; milk, meat and meat byproducts of cattle, goats, hogs, horses and sheep at 0.01 ppm; and fat of cattle, goats, hogs, horses and sheep at 0.05 ppm. The proposed analytical method involves Column switching high performance liquid chromatography and UV detection. 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 petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of fenoxycarb in plants (apples, citrus and grass) is well understood. Identified metabolic pathways are similar in plants and animals. It has been determined that fenoxycarb, per se, is the residue of concern for tolerance setting purposes.The metabolism of fenoxycarb in plants (apples, citrus and grass) is well understood. Identified metabolic pathways are similar in plants and animals. It has been determined that fenoxycarb, per se, is 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 fenoxycarb in or on food. The limits of detection (2.5 ng) and quantitation (0.01 ppm) allow monitoring of food with residues at or above the levels in the proposed tolerances. All methods are based on crop specific cleanup procedures and determination nce liquid chromatography with column-switching and UV detection.

3. *Magnitude of residues*. Residue trials: 15 residue trials in 8 states on apples and pears; 16 field trials in 13 states on grasses; 13 residue trials in 4 states on citrus; 8 residue trials in 6 states on tree nuts. No residues of fenoxycarb (0.01 ppm) were found in apples or pears treated at the maximum labeled rate. The maximum residues found in grasses were 0.056 ppm in forage and 0.041 in hay. Only one detectable residue at 0.02 ppm was found on citrus. This grapefruit sample was aerially treated with the maximum labeled rate. The maximum residue found in nutmeats treated at the maximum labeled rate was 0.02 ppm.

B. Toxicological Profile

1. *Acute toxicity*. The following acute toxicity studies have been conducted to support the proposed tolerance for fenoxycarb. The studies indicate that fenoxycarb has a low order of acute toxicity with effects in catgegory III and IV.

- Rat acute oral study with an LD_{50} >10,000 mg/kg.
- Rabbit acute dermal study with an LD_{50} > 2,000 mg/kg.
- Rat inhalation study with an LC_{50} > 4.4 mg/L.
- Primary eye irritation study in the rabbit showing slight eye irritation.
- Primary dermal irritation study in the rabbit showing fenoxycarb is not a skin irritant.

- Skin sensitization study showing fenoxycarb is not a skin sensitizer in the Guinea pig.
- Dermal absorption study showing a maximum of 30.2% of fenoxycarb is absorbed by the rat following a 24 hour dermal exposure.

2. *Genotoxicty*. Results from the following assays indicate that fenoxycarb is not genotoxic: Ames Assay - Negative; Mouse Micronucleus Test - Negative; *Saccharomyces cerevisiae* D7 test - Negative.

3. *Reproductive and developmental toxicity*. Novartis conducted a teratogenicity study in the rat at doses of 0, 50, 150, or 500 mg/kg/day by gavage with maternal and developmental NOELs of ≥ 500 mg/kg/day.

Novartis also conducted a teratogenicity study in the rabbit at doses of 0, 30, 100, 200 or 300 mg/kg/day. The maternal NOEL based on reduced body weight gains was 100 mg/kg/day. The developmental NOEL was ≥ 300 mg/kg/day.

In a 2-generation reproduction study, rats were dosed of 0, 200, 600 or 1,800 ppm. The systemic NOEL was 200 ppm based on decreased body weight gains and food consumption, increased gonad weights (without effects on reproductive performance or a morphological correlate), liver hypertrophy and focal necrosis and increased liver weights. There were no effects on fertility or reproductive performance. Based on decreased pup weights and slight delays in pinna unfolding and eye opening, there was no clear developmental NOEL. A derived NOEL (DNOEL), determined using analysis of variance and regression, was 40 ppm.

4. *Subchronic toxicity*. Novartis conducted a 21–day dermal study in which fenoxycarb was applied to the shaved skin of 5 male and 5 female New Zealand White rabbits at dose levels of 0, 20, 200, or 2,000 mg/kg for 21 consecutive days. The only effect observed was a slight increase in liver weights at the high dose. However, there was no histopathological correlate to this finding and the change was interpreted as representing an adaptive response. The NOEL was 200 mg/kg.

In a 6–month oral (capsule) study of dogs dosed at 0, 50, 150 or 500 mg/kg/day, the NOEL was 150 mg/kg/day based on reduced weight gain in females.

In a 90–day feeding study, Sprague Dawley rats were fed fenoxycarb at dietary concentrations to result in doses of 0, 80, 250 or 800 mg/kg/day. Based on slight liver weight increases at 80 mg/kg/day, the NOEL was < 80 mg/kg/day.

Novartis conducted a 90–day feeding study in mice in which mice were fed dietary concentrations of fenoxycarb to result in doses of 0, 100, 300 or 900 mg/kg/day. Based on increased liver weight accompanied by fatty changes, glycogen depletion and increased multinucleated hepatocytes, the NOEL was 100 mg/kg/day.

Rats in a 21–day inhalation study were exposed to 0, 0.01, 0.10 or 1.13 mg/L for 6 hrs/day/5 days/week. Based on decreased body weight gain in males and increased liver weight in females the NOEL was 0.10 mg/L.

5. *Chronic toxicity*. In a 52 week oral (capsule) study, dogs were dosed at levels of 0, 25, 80 or 260 mg/kg/day. Based on decreased body weight gain and food consumption and decreases in adrenal weights and inorganic phosphorous the NOEL was 25 mg/kg/day.

In a 24–month chronic feeding and oncogenicity study, rats were dosed at levels of 0, 200, 600 or 1,800 ppm. Based on liver toxicity (non-neoplastic histopathology and increased liver enzymes) the NOEL was 200 ppm. There was no evidence of carcinogenic potential.

In an 80–week chronic feeding and oncogenicity study, mice were dosed at 0, 30, 110 or 420 ppm for males and 0, 20, 80 or 320 ppm for females. Systemic toxicity was not observed at any level. The NOEL for chronic toxicity was ≥ 420 ppm and 320 ppm for males and females, respectively. There was evidence of carcinogenic potential. Lung adenomas and combined adenoma/carcinoma in addition to Harderian gland tumor incidences were increased in males at 420 ppm.

In an 18–month oncogenicity study, mice were dosed at 0, 10, 50, 500 or 2,000 ppm with a NOEL of 50 ppm (5 – 6 mg/kg/day). A carcinogenic response was noted in the lung in males and females at 500 and 2,000 ppm and in the liver of male mice at 500 and 2,000 ppm.

In a study investigating biochemical parameters in livers, mice were treated at doses of 0, 50, 500 or 2,000 ppm showing that fenoxycarb is a strong inducer of hepatic xenobiotic metabolizing enzymes in the mouse and can be classified as a peroxisome proliferator..

6. *Animal metabolism*. The metabolism of fenoxycarb in animals (goat and rat) is well understood. It has been determined that fenoxycarb, per se, is the residue of concern in animal commodities for tolerance setting purposes.

C. Aggregate Exposure

1. *Food*. For purposes of assessing the potential dietary exposure under the proposed tolerances, Novartis has estimated aggregate exposure based on exposure from anticipated residues on pome fruit, tree nuts, citrus, cattle meat and milk. Since there were no detections of fenoxycarb in pome fruit, tree nuts or citrus treated according to label directions, the anticipated residue of 0.005 ppm, one-half the limit of quantitation, was used. Exposure via meat and milk comes from the possible consumption by cattle of almond hulls, grass, citrus pulp and apple pomace. Theoretical residues in milk make up greater than 50% of the possible exposure to fenoxycarb. Almost all of the theoretical residue in milk comes from almond hulls in the theoretical diet for cattle. The anticipated residue in milk is greatly exaggerated since almond hulls, in general, are not a significant portion of cattle diet. Percent crop treated figures for food crops and cattle feed were also used in the analysis.

2. *Drinking water*. The product chemistry data for fenoxycarb indicate that movement of fenoxycarb into drinking water would be unlikely and that fenoxycarb would be expected to have a strong affinity for binding to the soil. Soil metabolism data further demonstrate that fenoxycarb and its residues have an affinity for binding to soil, and thus a low propensity to move from the soil surface. Field studies in Washington, Georgia and in California showed that fenoxycarb did not move below the top 6 inches of the soil. Based on the available data, Novartis does not anticipate exposure to residues of fenoxycarb in drinking water. There is no established Maximum Contaminant Level for residues of fenoxycarb in drinking water The product chemistry data for fenoxycarb indicate that movement of fenoxycarb into drinking water would be unlikely and that fenoxycarb would be expected to have a strong affinity for binding to the soil. Soil metabolism data further demonstrate that fenoxycarb and its residues have an affinity for binding to soil, and thus a low propensity to move from the soil surface. Field studies in Washington, Georgia and in California showed that fenoxycarb did not move below the top 6 inches of the soil. Based on the available data, Novartis does not anticipate exposure to residues of fenoxycarb in drinking water. There is no established Maximum Contaminant Level for residues of fenoxycarb in drinking water.

3. *Non-dietary exposure*. Other potential sources of exposure of the general population to residues of pesticides are exposure from non-occupational sources. Novartis has estimated non-occupational exposure to fenoxycarb and concludes that the potential for exposure is insignificant. The potential for non-occupational exposure to fenoxycarb resulting from use of pet sprays or carpet sprays containing fenoxycarb is not included in safety determinations for the U.S. population and infants (shown below) since the registrations for these uses have been canceled. Exposure through turf uses of fenoxycarb as a fire ant bait is also considered not significant. Used as a fire ant bait, fenoxycarb is only applied to turf with active fire ant infestations and has no efficacy as a preventive treatment. Turf infested with fire ants is not commonly used for recreational activities because of the danger presented by fire ants. In addition, studies demonstrate that > 95% of the bait applied to fire ant infestations is removed by the ants within 24 hours. Therefore opportunity for exposure to fenoxycarb as a fire ant bait through treated turf is extremely small.

D. Cumulative Effects

Novartis also considered the potential for cumulative effects of fenoxycarb and other substances that have a common mechanism of toxicity. Novartis concluded that consideration of a common mechanism of toxicity is not appropriate at this time. Novartis does not have reliable information to indicate that toxic effects produced by fenoxycarb would be cumulative with those of any other chemical compounds; thus Novartis is considering only the potential risks from dietary exposure of fenoxycarb in its aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the exposure assumptions described above and based on the completeness and reliability of the toxicity data base for fenoxycarb, Novartis has calculated that aggregate exposure to fenoxycarb will utilize 0.016% of the Reference Dose (RfD) for the U.S. population - 48 states - all seasons, based on chronic toxicity endpoints. Lifetime carcinogenic risk for dietary exposure based on quantitative risk assessment and a Q_1* of 5.6×10^{-2} $(mg/kg/day)^{-1}$, is 7.31×10^{-7}. EPA generally has no concern for exposures below 100% of the RfD or lifetime carcinogenic risks less than 1×10^{-6}. Since anticipated residues of fenoxycarb in food are extremely low and all short term NOELs are at least an order of magnitude higher than the chronic NOEL, no acute risk from exposure to residues of fenoxycarb is anticipated. Therefore, Novartis concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fenoxycarb residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of fenoxycarb, Novartis considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. No evidence of developmental toxicity was observed in rats or rabbits. Fenoxycarb did not impair any reproductive or postnatal development parameters and was neither embryotoxic nor teratogenic. The NOELs for maternal and developmental toxicity in the rat were determined to be ≥ 500 mg/kg/day. The NOEL for maternal toxicity in the rabbit, based on reduced body weight gains, was 100 mg/kg/day and the NOEL for developmental toxicity was ≥ 300 mg/kg/day. In a 2-generation reproduction study in rats, the systemic NOEL for parental animals was 200 ppm based on decreased body weight gains and food consumption, increased gonad weights (without effects on reproductive performance or a morphological correlate), liver hypertrophy and focal necrosis and increased liver weights. There were no effects on fertility or reproductive performance. Based on decreased pup weights and slight delays in pinna unfolding and eye opening, there was no clear developmental NOEL. A NOEL of 40 ppm was derived using analysis of variance and regression. The mild nature of the effects of fenoxycarb on rat pups and the lack of effects in the developmental toxicity studies suggest that there is no particular sensitivity to fenoxycarb for infants and children.

Using the same exposure assumptions used for the determination in the general population, Novartis has concluded that the percent of the RfD that will be utilized by aggregate exposure to residues of fenoxycarb is 0.038% for nursing infants less than 1 year old, 0.098% for non-nursing infants, 0.048% for children 1–6 years old and 0.028% for children 7–12 years old. Therefore, based on the completeness and reliability of the toxicity data base, Novartis concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to fenoxycarb residues.

F. International Tolerances

No Codex MRLs have been established for residues of fenoxycarb.

3. Novartis Crop Protection, Inc.

PP 7F4897

EPA has received a pesticide petition (PP 7F4897) from Novartis Crop Protection, Inc., 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 180.368 by establishing a tolerance for residues of metolachlor in or on the raw agricultural commodities sunflower seed at 0.3 ppm and sunflower meal at 0.6 ppm. The proposed analytical method involves extraction by acid reflux, filtration, partition and cleanup with analysis by gas chromatography using Nitrogen/Phosphorous (N/P) detection. 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 metabolism of metolachlor in plants is well understood. Metabolism in plants involves conjugation of the chloroacetyl side chain with glutathione, with subsequent conversion to the cysteine and thiolactic acid conjugates. Oxidation to the corresponding sulfoxide derivatives occurs and cleavage of the side chain ether group, followed by conjugation with glucose.

2. *Analytical method*. Novartis Crop Protection has submitted a practical analytical method involving extraction by acid reflux, filtration, partition and cleanup with analysis by gas chromatography using Nitrogen/ Phosphorous (N/P) detection. The methodology converts residues of metolachlor into a mixture of CGA–37913 and CGA–49751. The limit of quantitation (LOQ) for the method is 0.03 ppm for CGA–37913 and 0.05 ppm for CGA–49751.

3. *Magnitude of residues*. Eight residue trials were conducted in major sunflower growing areas of the United States [CA, KS, TX (2), MN(2), ND, IL). Five tests were conducted with metolachlor alone and three were conducted as a tank mix of metolachlor and another product. Metolachlor residues were analyzed for in all trials. Applications were made at the

maximum labeled rate of 3.0 lbs. active ingredient/Acre (ai/A) and at 2 times the maximum labeled rate (6.0 lbs. ai/A). A processing study was also conducted with seeds processed into meal, hulls, crude oil, refined oil and soapstock. According to the Revised Table II of Subdivision O, only meal and refined oil are now required. Based on these studies and an earlier EPA review of these data, tolerances are proposed in sunflower seeds at 0.3 ppm and in sunflower meal at 0.6 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Metolachlor has a low order of acute toxicity. The combined rat oral LD_{50} is 2,877 mg/kg. The acute rabbit dermal LD_{50} is > 2,000 mg/kg and the rat inhalation LC_{50} is > 4.33 mg/L. Metolachlor is not irritating to the skin and eye. It has been shown to be positive in guinea pigs for skin sensitization. End use formulations of metolachlor also have a low order of acute toxicity and cause slight skin and eye irritation.

2. *Genotoxicty*. Assays for genotoxicity were comprised of tests evaluating metolachlor's potential to induce point mutations (Salmonella assay and an L5178/TK+/- mouse lymphoma assay), chromosome aberrations (mouse micronucleus and a dominant lethal assay) and the ability to induce either unscheduled or scheduled DNA synthesis in rat hepatocytes or DNA damage or repair in human fibroblasts. The results indicate that metolachlor is not mutagenic or clastogenic and does not provoke unscheduled DNA synthesis.

3. *Reproductive and developmental toxicity*. The developmental and teratogenic potential of metolachlor was investigated in rats and rabbits. The results indicate that metolachlor is not embyrotoxic or teratogenic in either species at maternally toxic doses. The NOEL for developmental toxicity for metolachlor was 360 mg/kg/day for both the rat and rabbit while the NOEL for maternal toxicity was established at 120 mg/kg/day in the rabbit and 360 mg/kg/day in the rat. A 2-generation reproduction study was conducted with metolachlor in rats at feeding levels of 0, 30, 300 and 1,000 ppm. The reproductive NOEL of 300 ppm (equivalent to 23.5 to 26 mg/kg/day) was based upon reduced pup weights in the F1a and F2a litters at the 1,000 ppm dose level (equivalent to 75.8 to 85.7 mg/kg/day). The NOEL for parental toxicity was equal to or greater than the 1,000 ppm dose level.

4. *Subchronic toxicity*. Metolachlor was evaluated in a 21–day dermal toxicity study in the rabbit and a 6–month dietary study in dogs; NOELs of 100 mg/kg/day and 7.5 mg/kg/day were established in the rabbit and dog, respectively. The liver was identified as the main target organ.

5. *Chronic toxicity*. A 1–year dog study was conducted at dose levels of 0, 3.3, 9.7, or 32.7 mg/kg/day. The Agency-determined RfD for metolachlor is based on the 1–year dog study with a NOEL of 9.7 mg/kg/day. The RfD for metolachlor is established at 0.1 mg/kg/day using a 100-fold uncertainty factor. A combined chronic toxicity/oncogenicity study was also conducted in rats at dose levels of 0. 1.5, 15 or 150 mg/kg/day. The NOEL for systemic toxicity was 15 mg/kg/day. An evaluation of the carcinogenic potential of metolachlor was made from two sets of oncogenicity studies conducted with metolachlor in rats and mice. Using the Guidelines for Carcinogenic Risk Assessment published September 24, 1986 (51 FR 33992) and the results of the November, 1994 Carcinogenic Peer Review, EPA has classified metolachlor as a Group C carcinogen and recommended using a Margin of Exposure (MOE) approach to quantify risk. This classification is based upon the marginal tumor response observed in livers of female rats treated with a high (cytotoxic) dose of metolachlor (3,000 ppm). The two studies conducted in mice were negative for oncogenicity.

6. *Animal metabolism*. The qualitative nature of the metabolism of metolachlor in animals is well understood. Metolachlor is rapidly metabolized and almost totally eliminated in the excreta of rats, goats, and poultry. Metabolism in plants and animals proceeds through common Phase 1 intermediates and glutathione conjugation.

7. *Metabolite toxicology*. The metabolism of metolachlor has been well characterized in standard FIFRA rat metabolism studies. The metabolites found are considered to be toxicologically similar to parent. Metolachlor does not readily undergo dealkylation to form an aniline or quinone amine as has been reported for other members of the chloroacetanilide class of chemicals. Therefore, it is not appropriate to include metolachlor with the group of chloroacetanilides that readily undergo dealkylation, producing a common toxic metabolite (quinone imine).

C. Aggregate Exposure

1. *Dietary exposure*. Dietary exposure consists of exposures from food and drinking water.

2. *Food*. For purposes of assessing the potential dietary exposure to metolachlor, aggregate exposure has been estimated based on the TMRC from the use of metolachlor in or on raw agricultural commodities for which tolerances have been previously established (40 CFR 180.368). The incremental effect on dietary risk resulting from the addition of sunflowers to the label was assessed by conservatively assuming that exposure would occur at the proposed tolerance level of 0.3 ppm with 100% of the crop treated.

The TMRC is obtained by multiplying the tolerance level residue for all these raw agricultural commodities by the consumption data which estimates the amount of these products consumed by various population subgroups. Some of these raw agricultural commodities (e.g. corn forage and fodder, peanut hay, sunflower meal) are fed to animals; thus exposure of humans to residues in these fed commodities might result if such residues are transferred to meat, milk, poultry, or eggs. Therefore, tolerances of 0.02 ppm for milk, meat and eggs and 0.2 ppm for kidney and 0.05 ppm for liver have been established for metolachlor. In an EPA review of sunflower residue data previously submitted by Novartis, the EPA has indicated that any secondary residues in meat, milk, poultry and eggs will be covered by existing metolachlor tolerances.

In conducting this exposure assessment, it has been conservatively assumed that 100% of all raw agricultural commodities for which tolerances have been established for metolachlor will contain metolachlor residues and those residues would be at the level of the tolerance--which results in an overestimation of human exposure.

3. *Drinking water*. Another potential source of exposure of the general population to residues of pesticides are residues in drinking water. Based on the available studies used by EPA to assess environmental exposure, it is not anticipated that exposure to residues of metolachlor in drinking water will exceed 20% of the RfD (0.02 mg/kg/day), a value upon which the Health Advisory Level of 70 ppb for metolachlor is based. In fact, based on experience with metolachlor, it is believed that metolachlor will be infrequently found in groundwater (less than 5% of the samples analyzed), and when found, it will be in the low ppb range.

4. *Non-dietary exposure*. Although metolachlor may be used on turf and ornamentals in a residential setting, that use represents less than 0.1% of the total herbicide market for residential turf and landscape uses. Currently, there

are no acceptable, reliable exposure data available to assess any potential risks. However, given the small amount of material that is used, it is concluded that the potential for non-occupational exposure to the general population is unlikely.

D. Cumulative Effects

The potential for cumulative effects of metolachlor and other substances that have a common mechanism of toxicity has also been considered. It is concluded that consideration of a common mechanism of toxicity with other registered pesticides in this chemical class (chloroacetamides) is not appropriate. Since EPA has concluded that the carcinogenic potential of metolachlor is not the same as other registered chloroacetamide herbicides, based on differences in rodent metabolism (EPA Peer Review of metolachlor, 1994), it is believed that only metolachlor should be considered in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above, based on the completeness and reliability of the toxicity data, it is concluded that aggregate exposure to metolachlor will utilize 1.3% of the RfD for the U.S. population. 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. Therefore, it is concluded that there is a reasonable certainty that no harm will result from aggregate exposure to metolachlor or metolachlor residues.

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

Developmental toxicity (reduced mean fetal body weight, reduced number of implantations/dam with resulting decreased litter size, and a slight increase in resorptions/dam with a resulting increase in post-implantation loss) was observed in studies conducted with metolachlor in rats and rabbits. The NOEL's for developmental effects in both rats and rabbits were established at 360 mg/kg/day. The developmental effect observed in the metolachlor rat study is believed to be a secondary effect resulting from maternal stress (lacrimation, salivation, decreased body weight gain and food consumption and death) observed at the limit dose of 1,000 mg/kg/day.

A 2-generation reproduction study was conducted with metolachlor at feeding levels of 0, 30, 300 and 1,000 ppm. The reproductive NOEL of 300 ppm (equivalent to 23.5 to 26 mg/kg/day) was based upon reduced pup weights in the F1a and F2a litters at the 1,000 ppm dose level (equivalent to 75.8 to 85.7 mg/kg/day). The NOEL for parental toxicity was equal to or greater than the 1,000 ppm dose level.

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 relative to pre- and post-natal effects for children is complete. Further, for the chemical metolachlor, the NOEL of 9.7 mg/kg/day from the metolachlor chronic dog study, which was used to calculate the RfD (discussed above), is already lower than the developmental NOEL's of 360 mg/kg/day from the metolachlor teratogenicity studies in rats and rabbits. In the metolachlor reproduction study, the lack of severity of the pup effects observed (decreased body weight) at the systemic lowest observed-effect-level (equivalent to 75.8 to 85.7 mg/kg/day) and the fact that the effects were observed at a dose that is nearly 10 times greater than the NOEL in the chronic dog study (9.7 mg/kg/day) suggest there is no additional sensitivity for infants and children. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children and that the RfD at 0.1 mg/kg/day based on the chronic dog study is appropriate for assessing aggregate risk to infants and children from use of metolachlor.

Using the conservative exposure assumptions described above, the percent of the RfD that will be utilized by aggregate exposure to residues of metolachlor including the proposed use on sunflowers is 1.1% for nursing infants less than 1 year old, 3.3% for non-nursing infants, 2.7% for children 1 to 6 years old and 2.0% for children 7 to 12 years old. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to metolachlor residues.

F. International Tolerances

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRL's) established for residues of metolachlor in or on raw agricultural commodities.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–769; FRL 5748–6]

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–769, must be received on or before November 7, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), 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'' of this document. No Confidential Business Information (CBI) 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 CBI. Information so marked 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.,