continuation of stakeholder meetings that started in 1995 to obtain input on the Agency's Drinking Water Program. These meetings were initiated as part of the Drinking Water Program Redirection efforts to help refocus EPA's drinking water priorities and to support strong, flexible partnerships among EPA, States, Tribes, local governments, and the public. At the upcoming meeting, EPA is specifically seeking input from stakeholders focused on issues related to environmental justice. EPA encourages the full participation of all stakeholders throughout this process.

DATES: This stakeholder meeting will be held on Thursday, March 12, 1998 from 10:00 a.m. to 5:00 p.m. EST. It will be held simultaneously in eleven cities across the United States via videoconference call.

Registration: To register for the meeting, please contact the name next to the city in which you plan to attend the meeting. Those registered for the meeting by Wednesday, March 4, 1998 will receive an agenda, logistics sheet, and background materials for the different regulations prior to the meeting. The following information contains the meeting location and contact name and phone number for registration in each city.

EPA Region 1, One Congress St., 10th Floor, Boston, MA 02203–0001: Rhona Julien, 617/565–9454.
EPA Region 2, 290 Broadway, 26th Floor, New York, NY, 10007: Wanda Ayala, 212/637–3660.
OSWERNJ, Edison Division of Science and Assessment, 2890 Woodbridge Ave., Edison, NJ 08837: Wanda Ayala, 212/637–3660.
EPA Region 3, 841 Chestnut Building, Philadelphia, PA 19107: Reggie Harris, 215/566–2988. (Philadelphia will be on conference call only)
EPA Region 4, 100 Alabama St., SW, Atlanta, GA 30303: Natalie Ellington, 404/562–9453.
EPA Region 5, 77 West Jackson, Blvd., Chicago, IL 60604–3507: Karla Johnson, 312/886–5993.
EPA Region 6, First Interstate Bank at Fountain Place, 1445 Ross Ave., 12th Floor, Suite 1200, Dallas, TX 75202–2733: Shirley Augurson, 214/665–7401.
EPA Region 7, 726 Minnesota Ave., Kansas City, KS 66101: Althea Moses, 913/551–7649.
EPA Region 8, 999 18th St., Suite 500, Denver, CO 80202–2405: Nancy Reish, 303/312–6040.
EPA Region 9, 75 Hawthorne St., San Francisco, CA 94105: Loretta Vanegas, 415/744–1946.
EPA Headquarters, Auditorium, 401 M St., SW, Washington, DC 20460: Safe Drinking Water Hotline, 1–800–426–4791.

SUPPLEMENTARY INFORMATION:

A. Background

Under the Safe Drinking Water Act (SDWA) Amendments of 1996, EPA must develop regulations for several contaminants and develop regulatory tools for more thorough analyses. The 1996 SDWA amendments require that new regulations be developed so as to ensure that they represent a meaningful opportunity for health risk reduction. Also required is a detailed analysis of the relationship to: health impacts, including those to sensitive subgroups; impacts of other contaminants; treatment objectives; incremental impacts above a baseline that considers current regulations; uncertainty; and affordability. EPA must also consider the impact on the technical, financial, and managerial capacity of water systems. In so doing, EPA must also use the best available, peer reviewed science and methods. After first defining a maximum contaminant level (MCL), or treatment technique standard based on affordable technology, EPA must determine whether the costs of that standard would be justified by the benefits. If not, EPA may adjust an MCL to a level that maximizes health risk reduction benefits at a cost that is justified by the benefits. The authority to adjust the MCL has limits that also require evaluation. The SDWA also requires that comprehensive, informative, and understandable information be provided to the public.

The upcoming meeting deals specifically with EPA's efforts to develop new regulations for specific drinking water contaminants and the processes involved in developing them. EPA is to propose a Maximum Contaminant Level Goal (MCLG) and National Primary Drinking Water Standards (NPDWSs) for radon by August 1999, and propose a NPDWS for arsenic by January 2000. EPA will revise and strengthen the 1989 Surface Water Treatment Rule and is required to have the Interim Enhanced Surface Water Treatment and Stage 1 Disinfection Byproducts Rules (DBPR) finalized by November 1998, and the Ground Water Disinfection Rule (GWDR) proposed by March 1999. EPA must also issue regulations to address filter backwash recycling and a Long Term Enhanced Surface Water Treatment Rule. These rules are to control microbial pathogens, disinfectants and disinfection byproducts (DBPs) in drinking water. Regulatory impact analysis (cost-benefit analysis) is also addressed in SDWA and will be discussed at the meeting.

B. Request for Stakeholder Involvement

EPA has announced this public meeting to hear the views of stakeholders on EPA's plans for proposed regulations for radon, ground water disinfection, surface water treatment, arsenic, and approaches for enacting regulatory cost and benefit analysis.

Dated: February 20, 1998.

Elizabeth R. Fellows,

Acting Director, Office of Ground Water and Drinking Water, Environmental Protection Agency.

[FR Doc. 98–5557 Filed 3–3–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–794; FRL–5774–1]

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–794, must be received on or before April 3, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, 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. 119 at the Virginia 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
Beth Edwards	Rm. 206, CM #2, 703–305–5400, e-mail: edwards.beth@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Sidney Jackson	Rm. 233, CM #2, 703–305–7610, e-mail: jackson.sidney@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–794] (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 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [PF–794] and appropriate petition number. Electronic comments on this 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: February 24, 1998.

James Jones,

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. DowElanco

PP 8F4942

EPA has received a pesticide petition (PP 8F4942) from DowElanco, 9330 Zionsville Road, Indianapolis, IN 46254 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 the insecticide spinosad in or on the raw agricultural commodity cotton gin byproducts at 1.5 parts per million (ppm). Because of the amount of spinosad residue found in cotton gin byproducts as well as wet apple pomace (pending tolerance under PP 6F4761) and almond hulls and citrus dried pulp (pending tolerances under PP 7F4871) and the amount of cotton gin byproducts, almond hulls, citrus dried pulp, and apple pomace potentially included in livestock rations, a livestock, fat residue tolerance of 0.8 ppm, a milk residue tolerance of 0.05 ppm, and a milk fat residue tolerance of 0.7 ppm are also being proposed. The following meat and milk tolerances for residues of spinosad are presently pending under PP 6F4761 and PP 7F4871: meat at 0.04 ppm, kidney and liver at 0.2 ppm, fat at 0.7 ppm, milk at 0.04 ppm, and milk fat at 0.5 ppm. An adequate analytical method is available for enforcement purposes. 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 spinosad in plants (apples, cabbage, cotton, tomato, and turnip) and animals (goats and poultry) is adequately understood for the purposes of these tolerances. A rotational crop study showed no carryover of measurable spinosad related residues in representative test crops.

2. *Analytical method*. There is a practical method (HPLC with UV detection) for detecting (0.004 ppm) and measuring (0.01 ppm) levels of spinosad in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set for these tolerances. The method has had a successful method tryout in the EPA's laboratories.

3. *Magnitude of residues*. Magnitude of residue studies were conducted for cotton gin byproducts at seven sites. Residues found in these studies ranged from less than the limit of quantitation of the analytical method to 0.9 ppm on cotton gin byproducts.

B. Toxicological Profile

1. *Acute toxicity*. Spinosad has low acute toxicity. The rat oral LD_{50} is 3,738 mg/kg for males and >5,000 milligrams/kilograms (mg/kg) for females, whereas the mouse oral LD_{50} is >5,000 mg/kg. The rabbit dermal LD_{50} is >2,000 mg/kg and the rat inhalation LC_{50} is >5.18 mg/l air. In addition, spinosad is not a skin sensitizer in guinea pigs and does not produce significant dermal or ocular irritation in rabbits. End use formulations of spinosad that are water based suspension concentrates have similar low acute toxicity profiles.

2. *Genotoxicity*. Short term assays for genotoxicity consisting of a bacterial reverse mutation assay (Ames test), an

in vitro assay for cytogenetic damage using the Chinese hamster ovary cells, an in vitro mammalian gene mutation assay using mouse lymphoma cells, an in vitro assay for DNA damage and repair in rat hepatocytes, and an in vivo cytogenetic assay in the mouse bone marrow (micronucleus test) have been conducted with spinosad. These studies show a lack of genotoxicity.

3. *Reproductive and developmental toxicity*. Spinosad caused decreased body weights in maternal rats given 200 milligrams/kilograms/day (mg/kg/day) by gavage (highest dose tested). This was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The NOELs for maternal and fetal effects in rats were 50 and 200 mg/kg/day, respectively. A teratology study in rabbits showed that spinosad caused decreased body weight gain and a few abortions in maternal rabbits given 50 mg/kg/day (highest dose tested). Maternal toxicity was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The NOELs for maternal and fetal effects in rabbits were 10 and 50 mg/kg/day, respectively. The NOEL found for maternal and pup effects in a rat reproduction study was 10 mg/kg/day. Neonatal effects at 100 mg/kg/day (highest dose tested in the rat reproduction study) were attributed to maternal toxicity.

4. *Subchronic toxicity*. Spinosad was evaluated in 13-week dietary studies and showed NOELs of 4.9 mg/kg/day in dogs, 6 mg/kg/day in mice, and 8.6 mg/kg/day in rats. No dermal irritation or systemic toxicity occurred in a 21-day repeated dose dermal toxicity study in rabbits given 1,000 mg/kg/day.

5. *Chronic toxicity*. Based on chronic testing with spinosad in the dog and the rat, the EPA has set a reference dose (RfD) of 0.0268 mg/kg/day for spinosad. The RfD has incorporated a 100-fold safety factor to the NOELs found in the chronic dog study. The NOELs shown in the dog chronic study were 2.68 and 2.72 mg/kg/day, respectively for male and female dogs. The NOELs shown in the rat chronic study were 2.4 and 3.0 mg/kg/day, respectively for male and female rats. Using the Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), it is proposed that spinosad be classified as Group E for carcinogenicity (no evidence of carcinogenicity) based on the results of carcinogenicity studies in two species. There was no evidence of carcinogenicity in an 18-month mouse feeding study and a 24-month rat feeding study at all dosages tested. The NOELs shown in the mouse oncogenicity study were 11.4 and 13.8 mg/kg/day, respectively for male and female mice. The NOELs shown in the rat chronic/oncogenicity study were 2.4 and 3.0 mg/kg/day, respectively for male and female rats. A maximum tolerated dose was achieved at the top dosage level tested in both of these studies based on excessive mortality. Thus, the doses tested are adequate for identifying a cancer risk. Accordingly, a cancer risk assessment is not needed.

6. *Animal metabolism*. There were no major differences in the bioavailability, routes or rates of excretion, or metabolism of spinosyn A and spinosyn D following oral administration in rats. Urine and fecal excretions were almost completed in 48-hours post-dosing. In addition, the routes and rates of excretion were not affected by repeated administration.

7. *Metabolite toxicology*. The residue of concern for tolerance setting purposes is the parent material (spinosyn A and spinosyn D). Thus, there is no need to address metabolite toxicity.

8. *Neurotoxicity*. Spinosad did not cause neurotoxicity in rats in acute, subchronic, or chronic toxicity studies.

9. *Endocrine effects*. There is no evidence to suggest that spinosad has an effect on any endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure from use of spinosad on cotton gin byproducts as well as from other existing or pending uses, a conservative estimate of aggregate exposure is determined by basing the TMRC on the proposed tolerance levels for spinosad and assuming that 100% of the cotton gin byproducts and other existing and pending crop uses grown in the U.S. were treated with spinosad. The TMRC is obtained by multiplying the tolerance residue levels by the consumption data which estimates the amount of crops and related foodstuffs consumed by various population subgroups. The use of a tolerance level and 100% of crop treated clearly results in an overestimate of human exposure and a safety determination for the use of spinosad on crops cited in this summary that is based on a conservative exposure assessment.

2. *Drinking water*. Another potential source of dietary exposure are residues in drinking water. Based on the available environmental studies conducted with spinosad wherein it's properties show little or no mobility in soil, there is no anticipated exposure to residues of spinosad in drinking water. In addition, there is no established Maximum Concentration Level for residues of spinosad in drinking water.

3. *Non-dietary exposure*. Spinosad is currently registered for use on cotton with several crop registrations pending all of which involve applications of spinosad in the agriculture environment. Spinosad is also currently registered for use on turf and ornamentals at low rates of application (0.04 to 0.54 lb a.i. per acre). Thus, the potential for non-dietary exposure to the general population is not expected to be significant.

D. Cumulative Effects

The potential for cumulative effects of spinosad and other substances that have a common mechanism of toxicity is also considered. In terms of insect control, spinosad causes excitation of the insect nervous system, leading to involuntary muscle contractions, prostration with tremors, and finally paralysis. These effects are consistent with the activation of nicotinic acetylcholine receptors by a mechanism that is clearly novel and unique among known insecticidal compounds. Spinosad also has effects on the GABA receptor function that may contribute further to its insecticidal activity. Based on results found in tests with various mammalian species, spinosad appears to have a mechanism of toxicity like that of many amphiphilic cationic compounds. There is no reliable information to indicate that toxic effects produced by spinosad would be cumulative with those of any other pesticide chemical. Thus it is appropriate to consider only the potential risks of spinosad in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions and the proposed RfD described above, the aggregate exposure to spinosad use on cotton gin byproducts and other existing or pending crop uses will utilize 20.6% of the RfD for the U.S. population. A more realistic estimate of dietary exposure and risk relative to a chronic toxicity endpoint is obtained if average (anticipated) residue values from field trials are used. Inserting the average residue values in place of tolerance residue levels produces a more realistic, but still conservative risk assessment. Based on average or anticipated residues in a dietary risk analysis, the use of spinosad on cotton gin byproducts and other existing or pending crop uses will utilize 4.5% 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.

Thus, it is clear that there is reasonable certainty that no harm will result from aggregate exposure to spinosad residues on cotton gin products and other existing or pending crop uses.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of spinosad, data from developmental toxicity studies in rats and rabbits and a 2-generation reproduction study in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability and potential systemic toxicity of mating animals and on various parameters associated with the well-being of pups.

Section 408 of the FFDCA 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 for spinosad relative to pre- and post-natal effects for children is complete. Further, for spinosad, the NOELs in the dog chronic feeding study which was used to calculate the RfD (0.0268 mg/kg/day) are already lower than the NOELs from the developmental studies in rats and rabbits by a factor of more than 10-fold.

Concerning the reproduction study in rats, the pup effects shown at the highest dose tested were attributed to maternal toxicity. Therefore, it is concluded that an additional uncertainty factor is not needed and that the RfD at 0.0268 mg/kg/day is appropriate for assessing risk to infants and children.

Using the conservative exposure assumptions previously described (tolerance level residues), the percent RfD utilized by the aggregate exposure to residues of spinosad on cotton gin byproducts and other existing or pending crop uses is 38.1% for children 1 to 6 years old, the most sensitive population subgroup. If average or anticipated residues are used in the dietary risk analysis, the use of spinosad on these crops will utilize 11.1% of the RfD for children 1 to 6 years old. Thus, 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 spinosad residues on cotton gin byproducts and other existing or pending crop uses.

F. International Tolerances

There are no Codex maximum residue levels established for residues of spinosad on cotton gin byproducts or any other food or feed crop. (Beth Edwards)

2. Interregional Research Project

PP 4E4420 and 6E4638

EPA has received pesticide petitions (PP 4E4420 and 6E4638) from the Interregional Research Project Number 4 (IR-4), 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 combined residues (free and bound) of the herbicide metolachlor and its metabolites, CGA- 37913 and CGA- 49751, expressed as the parent compound, in or on the raw agricultural commodities (RACs) peppers at 0.5 ppm, forage of the grass forage, fodder and hay group (excluding Bermudagrass), forage at 12 ppm and hay of the grass forage, fodder and hay group (excluding Bermudagrass) at 0.3 ppm. Time-limited tolerances are being proposed for peppers and grass grown for seed to allow time to developed magnitude of residue data from an additional three field trials for bell pepper and five additional field trials for grass forage and hay. 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 support granting of the petitions. Additional data may be needed before EPA rules on the petitions. This notice contains a summary of the petitions submitted by Novartis Crop Protection, Inc. (Novartis), the registrant.

A. Residue Chemistry

1. *Plant and animal metabolism*. The qualitative nature of the metabolism of metolachlor in plants and animals 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. In animals, 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.

2. *Analytical method*. IR-4 has submitted a practical analytical method involving extraction by acid reflux, filtration, partition and cleanup with analysis by gas chromatography using nitrogen specific detection. The methodology accounts for residues of CGA-37913 and CGA-49751 which are formed from metolachlor and its metabolites under acid hydrolysis. The limit of quantitation (LOQ) for the method is 0.03 ppm for CGA-37913 and 0.05 ppm for CGA-49751. Residues of CGA-37913 and CGA-49751 are reported as metolachlor equivalents.

3. *Magnitude of residues*. For peppers - This petition for the establishment of a 0.5 ppm tolerance for metolachlor on peppers is supported by the individual tolerances already established in a number of pepper varieties: bell (0.1 ppm), chili (0.5 ppm), Cubanelle (0.1 ppm), and tabasco (0.5 ppm).

In four field trials, 1.5 to 3.5 lbs. metolachlor per acre, was applied 48 hours after transplanting of bell peppers. Residues from these samples were less than 0.1 ppm. Metolachlor was also applied at 2.0 to 4.0 lbs active per acre to Cubanelle peppers shortly after transplanting. Residues recovered from these samples were also below the 0.1 ppm level. In tabasco peppers, 4 lbs metolachlor per acre was applied as a directed spray to the pepper plants and peppers were harvested either 7 or 14 days after treatment. Residues of nearly 0.5 ppm were recovered 7 days after treatment, however, the residue levels dropped to approximately 0.25 ppm when harvested 14 days after treatment. For chili peppers, metolachlor was applied post-emergence as a foliar application at 2.0 lbs active per acre. Samples harvested at approximately 40 days after treatment had residues of 0.36 ppm (as CGA-49751), however, samples taken later than this date had residues below 0.03 ppm. In one additional chili pepper trial, metolachlor was applied at rates of 1 to 4 lbs active ingredient per acre to direct seeded peppers. No residues were recovered from the peppers harvested 204 days after the application. The proposed label would allow one surface broadcast application of metolachlor at 1.25 to 2.0 pints (1.25 to 2.0 lbs. active) per acre within 48 hours after transplanting peppers and with a pre-harvest interval of 63 days.

For Grass Grown for Seed - This petition is supported by six field residue tests conducted on grasses grown for seed. Quantitative measurements of the metolachlor hydrolysates, CGA-37913 and CGA-49751, were made for all

samples and reported as metolachlor equivalents. In all residue tests, metolachlor (Dual 8E®) was applied post-emergence at a maximum of 2.0 lbs. a.i./A at the early regrowth stage prior to weed emergence. The maximum residue in forage was 27 ppm (60-day PHI). Residues in forage declined with increasing PHI. Maximum residues in straw, screenings, and seed were 0.11 ppm, 0.04 ppm, and <0.08 ppm, respectively.

B. Toxicological Profile

1. *Acute toxicity*. Metolachlor has a low order of acute toxicity. The combined rat oral lethal dose $(LD)_{50}$ is 2,877 milligrams(mg)/kilogram(kg). The acute rabbit dermal LD_{50} is >2,000 mg/kg and the rat inhalation lethal concentration $(LC)_{50}$ is >4.33 mg/liter (L). Metolachlor was not irritating to the skin and eye. It was 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. *Genotoxicity*. 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 deoxyribonucleic acid (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*. Adverse developmental and reproductive potential of metolachlor was investigated in rats and rabbits. The results indicate that metolachlor is not embyrotoxic or reproductive toxic in either species at maternally toxic doses. The no-observed-effect level (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 reference dose(RfD) for metolachlor is based on the one 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/carcinogenicity 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.

6. *Carcinogenicity*. An evaluation of the carcinogenic potential of metolachlor was made from two sets of carcinogenicity studies conducted with metolachlor in rats and mice. EPA has classified metolachlor as a Group C (possible human) carcinogen and uses 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 carcinogenicity.

A NOEL of 15 mg/kg/day from the 2 year rat feeding study was determined to be appropriate for use in the MOE carcinogenic risk assessment. However, because the chronic reference dose is lower (9.7 mg/kg/day) than the carcinogenic NOEL (15 mg/kg/day), the EPA is using the Reference Dose for quantification of human risk.

7. *Estrogenic potential/endocrine disruption*. Metolachlor does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There is no evidence that metolachlor has any effect on endocrine function in developmental or reproduction studies. Furthermore, histological investigation of endocrine organs in the chronic dog, rat and mouse studies conducted with metolachlor did not indicate that the endocrine system is targeted by metolachlor, even at maximally tolerated doses administered for a lifetime. Although residues of metolachlor have been found in raw agricultural commodities, there is no evidence that metolachlor bioaccumulates in the environment.

C. Aggregate Exposure

1. *Dietary (food) exposure*. For purposes of assessing the potential dietary exposure to metolachlor, aggregate exposure has been estimated based on the Theoretical Maximum Residue Contribution (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 peppers to the label was assessed by assuming that exposure would occur at the proposed tolerance level of 0.5 ppm with 100% of the crop treated. The potential human dietary exposure from grasses grown for seed comes from the consumption of grass forage and hay by animals. Based on the tolerances proposed in forage (12 ppm) and hay (0.3 ppm), it has been determined that tolerances previously established for metolachlor in animal commodities of milk and meat, fat, kidney, liver and meat byproducts are adequate to cover secondary residues resulting from animal consumption of grass forage and hay.

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) 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 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.

2. *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, Novartis anticipates that exposure to residues of metolachlor in drinking water will not exceed 20% of the RfD (0.02 mg/kg/day), a value upon which the Health Advisory Level of 70 parts per billion (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.

3. *Non-dietary exposure*. Although metolachlor may be used on turf and ornamentals in a residential setting, that use represents less than 0.1 percent 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 from non-dietary exposure. However, given the small amount of material that is used, Novartis believes 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. Novartis believes that consideration of a common mechanism of toxicity with other registered pesticides in this chemical class (chloroacetamides) is not appropriate. EPA 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). Novartis maintains that only metolachlor should be considered in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the exposure assumptions described above, based on the completeness and reliability of the toxicity data, Novartis has concluded that aggregate exposure to metolachlor including the proposed new uses on peppers and grasses grown for seed will utilize approximately 3.0% 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, Novartis believes 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) were observed in studies on 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.

Section 408 of the FFDCA 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 developmental toxicity 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 (LOEL) (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, Novartis concludes 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 exposure assumptions described above, Novartis concludes that the approximate percentages of the RfD that will be utilized by aggregate exposure to residues of metolachlor including published and pending tolerances is 1% for U. S. population, for nursing infants less than 1%, 3% for non-nursing infants, 3% for children 1 to 6 years old and 2% for children 7 to 12 years old.

Therefore, based on the completeness and reliability of the toxicity data 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 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. (Sidney Jackson)

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ENVIRONMENTAL PROTECTION AGENCY

[PF–792; FRL–5772–6]

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–792, must be received on or before April 3, 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. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.