Development (OECD) Screening Information Data Sets (SIDS) program) to ensure that testing can be contributed to the international effort and, conversely, that international SIDS testing and assessments can be used to fulfill the Challenge Program's requirements.

V. What Will be Discussed at the Public Meetings?

The purpose of these meetings is to provide an opportunity for periodic updates on the HPV Chemical Challenge program, as well as to provide a forum where interested parties can contribute information, discuss and give individual perspectives on various topics related to the HPV Chemical Challenge program. Each meeting will be open to the public, but participation in discussions may be limited to those who pre-register with the Agency as described in Unit III. of this notice.

An agenda for each meeting, which EPA will develop in consultation with the stakeholders, will be made publicly available as early as possible prior to the meeting. The agenda for each meeting will be distributed to the participants who registered for that meeting and will be posted on the EPA website at http://www.epa.gov/chemrtk.

VI. Where Will Subsequent Public Meetings be Held?

EPA intends to hold additional meetings in July, September, and November, the dates, times, and locations for which still need to be determined. When scheduled, EPA will announce and provide relevant information about the meeting on the EPA website at http://www.epa.gov/chemrtk. The dates, times, and locations for these subsequent meetings will not be published in the **Federal Register**. Interested parties should consult the EPA Internet site indicated above for up-to-date information about these subsequent meetings.

List of Subjects

Environmental protection.

Dated: April 30, 1999.

Joseph A. Carra,

Acting Director, Environmental Assistance Division, Office of Pollution Prevention and Toxics.

[FR Doc. 99–11276 Filed 5–4–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–870; FRL–6072–7]

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–870, must be received on or before June 4, 1999.

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 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 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
Dani Daniel	Rm. 211, CM #2, 703–305–5409, e-mail:daniel.dani@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Cynthia Giles-Parker (PM 22).	Rm. 249, 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–870] (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 (insert docket number) 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: April 23, 1999.

Peter Caulkins, 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. Novartis Crop Protection, Inc.

PP 9F5045

EPA has received a pesticide petition (9F5045) 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 (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of difenoconazole ((2S,4R)/(2R,4S)/(2R,4R)/(2S,4S) 1-(2-(4-(4-chlorophenoxy)-2-chlorophenyl)-4-methyl-1,3-dioxolan-2-yl)methyl-1H-1,2,4-triazole) in or on the raw agricultural commodity (RAC) rapeseed at 0.01 parts per million (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 support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residues in plants is understood for the purpose of the proposed tolerance. The metabolism of ^{14}C-difenoconazole has been studied using both phenyl and triazole labels in wheat, tomatoes, potatoes, grapes, and spring rape The metabolic pathway was the same in these four separate and distinct crops.

2. *Analytical method*—i. *Food*. Novartis Crop Protection, Inc. has submitted a practical analytical method (AG-575B, master record identification (MRID) No. 428065-04) for detecting and measuring levels of difenoconazole in or on food with a limit of quantitation (LOQ) that allows monitoring of food with residues at or above the levels set in the proposed tolerances. EPA has validated this method and copies have been provided to FDA for insertion into pesticide analytical manual (PAM) II. The method is available to anyone who is interested, and may be obtained from the Field Operations Division, Office of Pesticide Programs.

ii. *Livestock*. Novartis Crop Protection, Inc. has submitted a practical analytical method (AG-544A, MRID-43292401) for detecting and measuring levels of difenoconazole in or on cattle tissues and milk and poultry tissues and eggs, with a LOQ that allows monitoring of food with residues at or above the levels set in the proposed tolerances. EPA has validated this method and copies have been provided to FDA for insertion into PAM II. The method is available to anyone who is interested, and may be obtained from the Field Operations Division, Office of Pesticide Programs.

3. *Magnitude of residues*—i. *Food*. Six field trials were analyzed in concordance with the OPPTS guidelines based on expected reduced residues and environmental benefits of seed applications. The six trials, held in areas representing approximately 84% of commercial United States canola production (Agricultural Statistics, 1991), were conducted in Georgia (2%), Minnesota (16%), North Dakota (53%), South Dakota (2%), Idaho (6%), and Washington (5%). No residues were detected in rape seed at either a 1x or 3x treatment rate.

ii. *Livestock*. No tolerances are necessary for grain commodities. Tolerances in meat, milk, poultry or eggs were established for enforcement purposes.

B. Toxicological Profile

The following mammalian toxicity studies were conducted and submitted in support of the establishment of tolerances for difenoconazole.

1. *Acute toxicity*. Difenoconazole has a low order of acute toxicity. The oral rat LD_{50} is 1,453 milligram/kilogram (mg/kg). The rabbit acute dermal LD_{50} is > 2,010 mg/kg and the rat inhalation LC_{50} is > 3.285 milligrams per liter (mg/L). Difenoconazole is not a skin sensitizer in guinea pig and shows slight eye and dermal irritation in the rabbit.

2. *Genotoxicity*. There was no evidence of the induction of point mutations in an Ames test, no evidence of mutagenic effects in a mouse lymphoma test or in a nucleus anomaly test with Chinese hamsters, and no evidence of induction of DNA damage in a rat hepatocyte DNA repair test or in a human fibroblast DNA repair test.

3. *Reproductive and developmental toxicity*. An oral teratology study in rats had a maternal no-observed adverse effect level (NOAEL) of 16 mg/kg/day based on excess salivation and decreased body weight gain and food consumption. The developmental NOAEL of 85 mg/kg/day was based on effects seen secondary to maternal toxicity including slightly reduced fetal body weight and minor changes in skeletal ossification. An oral teratology study in rabbits had a maternal NOAEL of 25 mg/kg/day based on decreased body weight gain, death, and abortion. The developmental NOAEL of 25 mg/kg/day was based on effects seen secondary to maternal toxicity including a slight increase in post-implantation loss and resorptions, and decreased fetal weight. A 2-generation reproduction study in rats had a parental and reproductive NOAEL of 25 part per million (ppm) based on significantly reduced female body weight gain, and reductions in male pup weights at 21-days.

4. *Subchronic toxicity*. A 13-week rat feeding study identified liver as a target organ and had a NOAEL of 20 ppm. A 13-week mouse feeding study also identified liver as a target organ and had a NOAEL of 20 ppm. A 26-week dog feeding study further identified liver, and also the eyes, as target organs and had a NOAEL of 100 ppm. A 21-day dermal study in rabbits had a NOAEL of 10 mg/kg/day based on decreased body weight gain at 100 and 1,000 mg/kg/day.

5. *Chronic toxicity*. A 24-month feeding study in rats had a NOAEL of 20 ppm based on liver toxicity at 500 and 2,500 ppm. An 18-month mouse feeding study had an overall NOAEL of 30 ppm based on decreased body weight gain and liver toxicity at 300 ppm. A 12-month feeding study in dogs had a NOAEL of 100 ppm based on decreased food consumption and increased alkaline phosphatase levels at 500 ppm.

6. *Carcinogenicity*. A 24-month feeding study in rats had a NOAEL of 20 ppm based on liver toxicity at 500 and 2,500 ppm. There was no evidence of an oncogenic response. An 18-month mouse feeding study had an overall NOAEL of 30 ppm based on decreased body weight gain and liver toxicity at 300 ppm. There was an increase in liver tumors only at dose levels that exceeded the maximum tolerated dose (MTD). The oncogenic NOAEL was 300 ppm.

7. *Animal metabolism*. The metabolism of difenoconazole is well understood. Studies with ^{14}C-difenoconazole in the rat, goat, and hen demonstrate that the majority of the

administered dose (76 to > 98%) is eliminated via the excreta as parent and metabolites. Very low concentrations of radioactivity, accounting for < 1 to 4% of the applied dose, remain in tissues. The liver and kidney typically show the highest radioactivity, but in the rat, the highest concentration in any tissue was found in the fat. Concentrations in goat milk reached a plateau on day 6 of the study at 0.043 ppm for the triazole label and 0.007 ppm for the phenyl label when goats were fed approximately 5 ppm for 10 days. Similarly, very little radioactivity was deposited in eggs; radioactivity reached a plateau of 0.248 to 0.299 ppm in yolks after 7 to 8-days, and 0.007 to 0.153 ppm in whites after 5 days, in hens fed at a rate equivalent to 5 ppm in the diet for 14 consecutive days. CGA-205375, an alcohol resulting from the deketalization of the dioxolane ring of difenoconazole, is a major metabolite found in animal tissues, excreta, milk, and eggs. The presence of CGA-71019, containing only the triazole ring, and CGA-189138, containing only the phenyl ring, indicates that bridge cleavage can occur in animals as well as plants. The metabolite patterns in the excreta of hens, goats, and rats were similar.

8. *Metabolite toxicology*. The residue of concern for tolerance setting purposes is the parent compound. Metabolites of difenoconazole are considered to be of equal or lesser toxicity than the parent.

9. *Endocrine disruption*. Developmental toxicity studies in rats and rabbits and a 2-generation reproduction study in rats gave no specific indication that difenoconazole may have effects on the endocrine system with regard to development or reproduction. Furthermore, histologic investigations were conducted on endocrine organs (thyroid, adrenal, and pituitary, as well as endocrine sex organs) from long-term studies in dogs, rats, and mice. There was no indication that the endocrine system was targeted by difenoconazole, even when animals were treated with maximally tolerated doses over the majority of their lifetime. Difenoconazole has not been found in RAC at the LOQ. Based on the available toxicity information and the lack of detected residues, it is concluded that difenoconazole has no potential for interfering with the endocrine system, and there is no risk of endocrine disruption in humans.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. When the potential dietary exposure to difenoconazole from established and pending tolerances (assuming 100% treated) is calculated, the theoretical maximum residue concentration (TMRC) of 0.000583 mg/kg/day utilizes 5.83% of the reference dose (RfD) for the overall U. S. population. For the most exposed population subgroups, non-nursing infants, the TMRC is 0.001656 mg/kg/day, utilizing 16.56% of the RfD, followed by children (1-6 years old), who are exposed to 14.58% of the RfD. In this analysis, canola does not contribute to exposure.

ii. *Drinking water*. Other potential sources of exposure of the general population to residues of pesticides are in drinking water and from non-occupational activities. Difenoconazole is currently used as a seed treatment and residues are, therefore, incorporated into the soil. The likelihood of contamination of surface water from run-off is essentially negligible. In addition, parent and aged leaching, soil adsorption/desorption, and radiolabeled pipe studies indicated that difenoconazole has a low potential to leach in the soil and it would not be expected to reach aquatic environments. For these reasons, and because of the low use rate, exposures to residues in ground and surface water are not anticipated to contribute significantly to the aggregate exposure profile for difenoconazole.

2. *Non-dietary exposure*. Non-occupational exposure to difenoconazole has not been estimated since the current registration is limited to seed treatment. Therefore, the potential for non-occupational exposure to the general population is insignificant.

D. Cumulative Effects

Novartis has considered the potential for cumulative effects of difenoconazole and other substances of common mechanism of toxicity. Novartis has concluded that consideration of a common mechanism of toxicity in aggregate exposure assessment is not appropriate at this time. Novartis has no reliable information to indicate that the toxic effects (generalized liver toxicity) seen at high doses of difenoconazole would be cumulative with those of any other compound. Thus, Novartis is considering only the potential risk of difenoconazole from dietary exposure in its aggregate and cumulative exposure assessment.

E. Safety Determination

1. *U.S. population*. Using very conservative exposure assumptions (tolerance levels for 100% of the United States market) described and based on the completeness of the toxicity data base for difenoconazole, Novartis calculates that aggregate exposure to difenoconazole utilizes < 6% of the RfD for the U.S. population based on chronic toxicity endpoints (NOAEL = 1 mg/kg/day). If more realistic assumptions were used to estimate anticipated residues and appropriate market share, this percentage would be considerably lower, and would be significantly lower than 100%, even for the most highly exposed population subgroup. EPA generally has no concern for exposures below 100% of the RfD. Therefore, Novartis concludes that there is a reasonable certainty that no harm will result from daily aggregate exposure to residues of difenoconazole over a lifetime of exposure.

2. *Infants and children*. Developmental toxicity and 2-generation toxicity studies were evaluated to determine if there is a special concern for the safety of infants and children from exposure to residues of difenoconazole. There was no evidence of embryotoxicity or teratogenicity, and no effects on reproductive parameters, including number of live births, birth weights, and post-natal development, at dose levels that did not cause significant maternal toxicity. In addition, there were no effects in young post-weaning animals that were not seen in adult animals in the 2-generation reproduction study. Therefore, Novartis concludes that it is inappropriate to assume that infants and children are more sensitive than the general population to effects from exposure to residues of difenoconazole, and also concludes that the use of an additional safety factor to protect infants and children is unnecessary.

F. International Tolerances

There are pending Codex maximum residue levels (MRLs) for this compound in Mexico for oats, wheat, and barley. There are also MRLs for this compound in Australia for carrots at 0.02 ppm, and bananas at 0.05 ppm.

2. Novartis Crop Protection, Inc.

PP 9F5046

EPA has received a pesticide petition (9F5046) from Novartis Crop Protection, Inc., PO Box 18300, Greensboro, North Carolina 27419 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of Thiamethoxam in or on the raw agricultural commodity (RAC) rape seed at 0.02 parts per million (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 primary metabolic pathways of thiamethoxam in plants (corn, rice, pears, and cucumbers) were similar to those described for animals, with certain extensions of the pathway in plants. Parent compound and CGA-322704 were major metabolites in all crops. The metabolism of thiamethoxam in plants and animals is understood for the purposes of the proposed tolerances. Parent thiamethoxam and the metabolite, CGA-322704, are the residues of concern for tolerance setting purposes.

2. *Analytical method*. Novartis Crop Protection Inc. has submitted practical analytical methodology for detecting and measuring levels of thiamethoxam in or on RAC. The method is based on crop specific cleanup procedures and determination by liquid chromatography with either ultraviolet (UV) or mass spectrometry (MS) detection. The limit of detection (LOD) for each analyte of this method is 1.25 nanogram (ng) injected for samples analyzed by UV and 0.25 ng injected for samples analyzed by MS, and the limit of quantitation (LOQ) is 0.005 ppm for milk and juices and 0.01 parts per million (ppm) for all other substrates.

3. *Magnitude of residues*. A residue program was performed for thiamethoxam on a full geography of canola, using a maximum application rate of 400 g.a.i./100 kilogram (kg) seed (0.024 lbs. a.i./acre, at the typical seeding rate). Two field trials also included seed treated at 3 times the normal rate for thiamethoxam. No residues were detected above the method LOD for thiamethoxam. The proposed tolerance on canola is 0.02 ppm for thiamethoxam.

B. Toxicological Profile

1. *Acute toxicity*. Thiamethoxam has low acute toxicity. The oral LD_{50} in rats is 1,563 millogram kilogram (mg/kg) for males and females, combined. The rat dermal LD_{50} is > 2,000 mg/kg and the rat inhalation LC_{50} is > 3.72 milligrams per liter (mg/L) air. Thiamethoxam is not a skin sensitizer in guinea pigs and does not produce dermal or eye irritation in rabbits. End-use formulations of thiamethoxam have similar low acute toxicity profiles.

2. *Genotoxicty*. Thiamethoxam did not induce point mutations in bacteria (Ames assay in *Salmonella* typhimurium and *Escherichia coli*) or in cultured mammalian cells (Chinese hamster V79) and was not genotoxic in an *in vitro* unscheduled DNA synthesis assay in rat hepatocytes. Chromosome aberrations were not observed in an *in vitro* test using Chinese hamster ovary cells and there were no clastogenic or aneugenic effects on mouse bone marrow cells in an *in vivo* mouse micronucleus test. These studies show that thiamethoxam is not genotoxic.

3. *Reproductive and developmental toxicity*. In rat and rabbit teratology studies with thiamethoxam there was no evidence of teratogenicity. In rabbits, thiamethoxam caused decreased body weights (bwt), decreased food consumption and premature death of two females administered 150 mg/kg/day during gestation. This maternal toxicity was accompanied by reduced fetal bwts and an increase in the incidence of minor skeletal anomalies or variations. Reduced maternal bwts and food consumption were also noted in females administered 50 mg/kg/day thiamethoxam during gestation. There was no indication of developmental toxicity at 50 mg/kg/day. The no-observable adverse effect level (NOAEL) in rabbits for maternal toxicity was 15 mg/kg/day. The NOAEL for developmental toxicity was 50 mg/kg/day. In rats, thiamethoxam caused decreased bwts, decreased food consumption and hypoactivity at 200 and 750 mg/kg/day. Reduced fetal bwts and an increase in the incidence of minor skeletal anomalies and variations were observed only at 750 mg/kg/day. There was no indication of developmental toxicity at 200 mg/kg/day. The NOAEL in rats for maternal toxicity was 30 mg/kg/day and for developmental toxicity was 200 mg/kg/day. In a 2-generation reproduction study in rats, parental bwts and food consumption were decreased at 2,500 ppm highest dose tested (HDT). Hyaline changes in the kidneys of adult males were observed at 2,500 and 1,000 ppm. Reproductive parameters were not affected by treatment with thiamethoxam. Effects on offspring were secondary to parental toxicity and consisted of slightly reduced offspring bwts at 1,000 ppm and 2,500 ppm. The NOAEL for systemic toxicity in parental animals and for offspring toxicity was 30 ppm (equivalent to 1.3 - 6.4 mg/kg/day).

4. *Subchronic toxicity* Thiamethoxam was evaluated in 13-week subchronic oral toxicity studies in rats, dogs and mice. Liver, kidneys and spleen were identified as target organs. The NOAEL was 25 ppm (1.74 mg/kg/day) in male rats based on the finding of a hyaline change in the kidney at 250 ppm (17.6 mg/kg/day). This kidney effect represents an accumulation of alpha-2-microglobulin, which is unique to the male rat and not relevant for human risk assessment. The NOAEL was 1,250 ppm (92.5 mg/kg/day) for female rats. The NOAEL in dogs was 250 ppm (8.23 mg/kg/day). The NOAEL in mice was 10 ppm (1.41 mg/kg/day) for males and 100 ppm (19.2 mg/kg/day) for females. No dermal irritation was observed in a 28-day repeated dose dermal toxicity study with thiamethoxam in rats given 1,000 mg/kg/day. The dermal NOAEL for systemic toxicity in rats was 250 mg/kg/day for males and 60 mg/kg/day for females.

5. *Neurotoxicity*. Thiamethoxam did not cause neurotoxicity in an acute neurotoxicity study in rats or in a subchronic 13-week neurotoxicity study in rats. The NOAEL for systemic toxicity in the acute neurotoxicity study was 100 mg/kg. The NOAEL for systemic toxicity in the subchronic neurotoxicity study was 95.4 mg/kg/day for males and 216.4 mg/kg/day for females.

6. *Chronic toxicity*. The carcinogenic potential of thiamethoxam has been evaluated in rats and mice. The proposed carcinogenic classification for thiamethoxam is as a Group C carcinogen. This classification is based on a liver tumor response in male and female mice at dose levels exceeding the maximum tolerance dose (MTD) and/or causing organ toxicity and induction of liver metabolizing enzymes. A NOAEL for liver tumors in mice was established at 20 ppm (2.63 mg/kg/day). No evidence of carcinogenicity was observed in rats. In the absence of a mutagenic activity, it is concluded that the mechanism of action leading to liver tumors in mice is not via genotoxic effects. Therefore, mouse liver tumors associated with thiamethoxam treatment have a threshold level.

7. *Animal metabolism*. Metabolism of thiamethoxam has been well characterized in animals. Metabolism in rats proceeds primarily via hydrolysis of the oxadiazine ring, followed by N-demethylation. Several minor pathways of metabolism of thiamethoxam were identified in animals. In rats, the majority of the radioactive dose was absorbed and then excreted in the urine. Parent compound was the major residue in urine. In hens and goats, the metabolite profile was the same as in rats, with certain extensions of the pathway.

8. *Metabolite toxicology*. The metabolism profile for thiamethoxam supports the use of an analytical enforcement method that accounts for parent thiamethoxam and CGA-322704. Other metabolites are considered of

equal or lesser toxicity than parent compound.

9. *Endocrine disruption*. Thiamethoxam does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There is no evidence that thiamethoxam has any effect on endocrine function in developmental or reproduction studies. Furthermore, histological investigation of endocrine organs in chronic dog, rat and mouse studies did not indicate that the endocrine system is targeted by thiamethoxam.

C. Aggregate Exposure

1. *Dietary exposure—Food and drinking water*. Chronic and acute dietary exposure to thiamethoxam was based on the occurrence of no detectable residues of thiamethoxam or its major metabolite resulting from the use of Helix on canola. There is no adverse exposure to thiamethoxam in the diet when chronic and acute assessments are made using tolerance level residues for canola oil (analytical method limit of quantitation (LOQ)), and 100% market share. The inclusion of the maximum concentration of thiamethoxam in water, taken from the highest estimated residue observed from the generic expected environmental concentration (GENEEC) and screening concentration In GROund (SCI-GROW) models, led to a maximum chronic exposure of 0.000019 mg/kg bwt/day in the most sensitive population subgroup, non-nursing infants (< 1-year old). This is only 0.1% of the proposed reference dose (RfD) of 0.013 mg/kg bwt/day. The inclusion of the water concentration estimate in the acute exposure assessment led to a margin of exposure (MOE) (NOAEL/exposure) of 264,491 at the 99.9th percentile of the most sensitive population subgroup, all infants (< 1-year old). The results of these analyses show that there is reasonable certainty that no harm will result from the exposure to dietary residues of thiamethoxam (including drinking water) from the use of Helix on canola.

2. *Non-dietary exposure*. There are no other uses currently registered for thiamethoxam that would lead to exposure from non-dietary sources. The proposed uses involve application of thiamethoxam to canola seed as part of the Helix product in an agricultural environment. A discussion of exposure from non-dietary sources will be made when future uses of thiamethoxam are proposed.

D. Cumulative Effects

The potential for cumulative effects of thiamethoxam and other substances that have a common mechanism of toxicity has also been considered. Thiamethoxam belongs to a new pesticide chemical class known as the neonicotinoids. There is no reliable information to indicate that toxic effects produced by thiamethoxam would be cumulative with those of any other chemical including another pesticide. Therefore, Novartis believes it is appropriate to consider only the potential risks of thiamethoxam in an aggregate risk assessment.

E. Safety Determination

1. *U.S. population*. Using the exposure assumptions and the proposed RfD described above, the aggregate exposure (including drinking water) to thiamethoxam from the application of helix to canola will utilize < 0.1% of the RfD for the U.S. population. Therefore, Novartis concludes that there is reasonable certainty that no harm will result from aggregate exposure to thiamethoxam residues from the use of helix on canola.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of thiamethoxam, data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat have been considered.

In teratology studies, delayed fetal development was apparent only at maternally toxic doses of thiamethoxam in rats and rabbits. In rabbits, 150 mg/kg/day was clearly toxic to does, causing death, weight loss, reduced food consumption and perineal or vaginal discharge. Developmental toxicity occurred secondary to maternal toxicity and consisted of reduced fetal bwts and an increase in minor skeletal anomalies or variations. Maternal toxicity was also noted at 50 mg/kg/day, consisting of reduced bwts and food consumption and total resorptions in one female. There was no indication of developmental toxicity at 50 mg/kg/day. The NOAEL for maternal toxicity was 15 mg/kg/day and for developmental toxicity was 50 mg/kg/day in rabbits. In rats, 200 and 750 mg/kg/day caused maternal toxicity, but developmental toxicity secondary to maternal toxicity was observed only at 750 mg/kg/day. The NOAEL for maternal toxicity was 30 mg/kg/day and for developmental toxicity was 200 mg/kg/day.

In a rat multigeneration study, parental toxic effects were noted at 2,500 ppm (250 mg/kg/day). and 1,000 ppm (100 mg/kg/day). Offspring bwts were reduced in males and females at 2,500 ppm (250 mg/kg/day) and in females (F1 only) at 1,000 ppm (100 mg/kg/day). The NOAEL for systemic toxicity in adult males was 30 ppm (approximately 3 mg/kg/day, range = 1.3 - 4.3 mg/kg/day) and in adult females was 1,000 ppm (approximately 100 mg/kg/day, range = 59.3 - 219.6 mg/kg/day). The NOAEL for toxicity to offspring was 30 ppm (approximately 3 mg/kg/day, range = 1.3 - 4.3 mg/kg/day). These studies show no evidence that developing offspring are more sensitive to than adults to the effects of thiamethoxam.

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 requirements, the database for thiamethoxam relative to pre- and post-natal effects for children is complete. Further, for thiamethoxam, the developmental studies showed no increased sensitivity in fetuses as compared to maternal animals following in utero exposures in rats and rabbits, and no increased sensitivity in pups as compared to the adults in the multi-generation reproductive toxicity study. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children and that an RfD of 0.013 mg/kg/day is appropriate for assessing aggregate risk to infants and children of thiamethoxam.

Assuming tolerance level residues and 100% of crops treated, only 0.1% of the thiamethoxam chronic RfD is utilized in the population subgroup all infant (< 1-year old) when helix is used as a seed treatment on canola. Therefore, based on the completeness and reliability of the toxicity database, Novartis concludes that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to thiamethoxam residues.

F. International Tolerances

There are no Codex maximum residue level (MRLs) established for residues of thiamethoxam on canola.

3. Norvartis Crop Protection, Inc.

PP 9F5051

EPA has received a pesticide petition (PP 9F5051) from Novartis Crop Protection, Inc. Greensboro, North Carolina, 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 Thiamethoxam in or on the raw agricultural commodity (RAC) fruiting vegetables at 0.25 parts per million (ppm), tomato paste at 0.80 ppm, head and stem brassica vegetables at 1.0 ppm, leafy brassica greens at 2.0 ppm, cucurbit vegetables at 0.2 ppm, leafy vegetables, tuberous and corm vegetables at 0.02 pm, barley hay at 0.05 ppm, barley straw at 0.03 ppm, cottonseed at 0.05 ppm, cotton gin by-products at 1.0 ppm, pome fruit at 0.2 ppm, wheat forage at 0.5 ppm, wheat grain, wheat straw, wheat hay, barley grain, sorghum grain, sorghum forage and sorghum fodder at 0.02 ppm and milk at 0.02 ppm. EPA has 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 primary metabolic pathways of thiamethoxam in plants (corn, rice, pears, and cucumbers) were similar to those described for animals, with certain extensions of the pathway in plants. Parent compound and CGA-322704 were major metabolites in all crops. The metabolism of thiamethoxam in plants and animals is understood for the purposes of the proposed tolerances. Parent thiamethoxam and the metabolite, CGA-322704, are the residues of concern for tolerance setting purposes.

2. *Analytical method*. Novartis Crop Protection Inc. has submitted practical analytical methodology for detecting and measuring levels of thiamethoxam in or on RAC. The method is based on crop specific cleanup procedures and determination by liquid chromatography with either ultraviolet (UV) or mass spectrometry (MS) detection. The limit of detection (LOD) for each analyte of this method is 1.25 nanogram (ng) injected for samples analyzed by UV and 0.25 ng injected for samples analyzed by MS, and the limit of quantitation (LOQ) is 0.005 ppm for milk and juices and 0.01 ppm for all other substrates.

3. *Magnitude of residues*. A residue program was performed for thiamethoxam on a full geography of cucumbers, cantaloupes and squash as representative cucurbit crops, tomatoes and peppers as representative fruiting vegetable crops, head lettuce, leaf lettuce, celery and spinach as representative leafy vegetable crops, broccoli and cabbage as representative head and stem brassica vegetable crops, mustard greens as a representative leafy brassica green vegetable crop, potatoes as a representative crop of tuberous and corm vegetables, and apples and pears as representative pome fruit crops. A seed treatment residue program was performed for thiamethoxam on sorghum, wheat, barley and cotton where seed was treated using specific seed treatment formulations. Cotton was also treated via foliar application. Field residue trials were performed for thiamethoxam on tobacco using both an in-furrow transplant drench and a post-foliar spray. Novartis also completed a three-level dairy study and calculated the rate of transfer of residues of thiamethoxam from residues in the animal feed to beef and dairy commodities.

B. Toxicological Profile

1. *Acute toxicity*. Thiamethoxam has low acute toxicity. The oral LD_{50} in rats is 1,563 milligram kilogram (mg/kg) for males and females, combined. The rat dermal LD_{50} is > 2,000 mg/kg and the rat inhalation LC_{50} is > 3.72 milligrams per liter (mg/L) air. Thiamethoxam is not a skin sensitizer in guinea pigs and does not produce dermal or eye irritation in rabbits. End-use formulations of thiamethoxam have similar low acute toxicity profiles.

2. *Genotoxicty*. Thiamethoxam did not induce point mutations in bacteria (Ames assay in *Salmonella typhimurium* and *Escherichia coli*) or in cultured mammalian cells (Chinese hamster V79) and was not genotoxic in an *in vitro* unscheduled DNA synthesis assay in rat hepatocytes. Chromosome aberrations were not observed in an *in vitro* test using Chinese hamster ovary cells and there were no clastogenic or aneugenic effects on mouse bone marrow cells in an *in vivo* mouse micronucleus test. These studies show that thiamethoxam is not genotoxic.

3. *Reproductive and developmental toxicity*. In rat and rabbit teratology studies with thiamethoxam there was no evidence of teratogenicity. In rabbits, thiamethoxam caused decreased body weights (bwts), decreased food consumption and premature death of two females administered 150 mg/kg/day during gestation. This maternal toxicity was accompanied by reduced fetal bwts and an increase in the incidence of minor skeletal anomalies or variations. Reduced maternal body weights (bwts) and food consumption were also noted in females administered 50 mg/kg/day thiamethoxam during gestation. There was no indication of developmental toxicity at 50 mg/kg/day. The no-observable adverse effect level (NOAEL) in rabbits for maternal toxicity was 15 mg/kg/day. The NOAEL for developmental toxicity was 50 mg/kg/day. In rats, thiamethoxam caused decreased bwts, decreased food consumption and hypoactivity at 200 and 750 mg/kg/day. Reduced fetal bwts and an increase in the incidence of minor skeletal anomalies and variations were observed only at 750 mg/kg/day. There was no indication of developmental toxicity at 200 mg/kg/day. The NOAEL in rats for maternal toxicity was 30 mg/kg/day and for developmental toxicity was 200 mg/kg/day. In a 2-generation reproduction study in rats, parental bwts and food consumption were decreased at 2,500 ppm highest dose tested (HDT). Hyaline changes in the kidneys of adult males were observed at 2,500 and 1,000 ppm. Reproductive parameters were not affected by treatment with thiamethoxam. Effects on offspring were secondary to parental toxicity and consisted of slightly reduced offspring bwts at 1,000 ppm and 2,500 ppm. The NOAEL for systemic toxicity in parental animals and for offspring toxicity was 30 ppm (equivalent to 1.3 - 6.4 mg/kg/day).

4. *Subchronic toxicity*. Thiamethoxam was evaluated in 13-week subchronic oral toxicity studies in rats, dogs and mice. Liver, kidneys and spleen were identified as target organs. The NOAEL was 25 ppm (1.74 mg/kg/day) in male rats based on the finding of a hyaline change in the kidney at 250 ppm (17.6 mg/kg/day). This kidney effect represents an accumulation of alpha-2-microglobulin, which is unique to the male rat and not relevant for human risk assessment. The NOAEL was 1,250 ppm (92.5 mg/kg/day) for female rats. The NOAEL in dogs was 250 ppm (8.23 mg/kg/day). The NOAEL in mice was 10 ppm (1.41 mg/kg/day) for males and 100 ppm (19.2 mg/kg/day) for females. No dermal irritation was observed in a 28-day repeated dose dermal toxicity study with thiamethoxam in rats given 1,000 mg/kg/day. The dermal NOAEL for systemic toxicity in rats was 250 mg/kg/day for males and 60 mg/kg/day for females.

5. *Neurotoxicity*. Thiamethoxam did not cause neurotoxicity in an acute neurotoxicity study in rats or in a subchronic 13-week neurotoxicity study in rats. The NOAEL for systemic toxicity in the acute neurotoxicity study was 100 mg/kg. The NOAEL for systemic toxicity in the subchronic neurotoxicity study was 95.4 mg/kg/day for males and 216.4 mg/kg/day for females.

6. *Chronic toxicity*. Chronic toxicity studies with thiamethoxam have been conducted in rats and dogs. In the dog,

minor changes in blood chemistry parameters, including increased plasma creatinine and plasma urea levels, and decreased alanine aminotransferase activities, occurred at the lowest-observable adverse effect level (LOAEL) of 750 ppm (21.0 mg/kg/day). The NOAEL in the dog was 150 ppm (4.05 mg/kg/day). The NOAEL established in the rat chronic toxicity study was 30 ppm (1.29 mg/kg/day) for males, based on kidney changes, (hyaline change, chronic tubular lesions, basophilic proliferation and lymphocytic infiltration) at the LOAEL of 500 ppm (21.0 mg/kg/day). These kidney changes are attributed to an accumulation of alpha-2-microglobulin, which is specific to the male rat, and not relevant to humans. In the female rat, the NOAEL was 1,000 ppm (50.3 mg/kg/day) based on decreased bwts and hemosiderosis of the spleen at the LOAEL of 3,000 ppm (155 mg/kg/day).

7. *Carcinogenicity*. The carcinogenic potential of thiamethoxam has been evaluated in rats and mice. The proposed carcinogenic classification for thiamethoxam is as a Group C carcinogen. This classification is based on a liver tumor response in male and female mice at dose levels exceeding the maximum tolerance dose (MTD) and/or causing organ toxicity and induction of liver metabolizing enzymes. A NOAEL for liver tumors in mice was established at 20 ppm (2.63 mg/kg/day). No evidence of carcinogenicity was observed in rats. In the absence of a mutagenic activity, it is concluded that the mechanism of action leading to liver tumors in mice is not via genotoxic effects. Therefore, mouse liver tumors associated with thiamethoxam treatment have a threshold level.

8. *Animal metabolism*. Metabolism of thiamethoxam has been well characterized in animals. Metabolism in rats proceeds primarily via hydrolysis of the oxadiazine ring, followed by N-demethylation. Several minor pathways of metabolism of thiamethoxam were identified in animals. In rats, the majority of the radioactive dose was absorbed and then excreted in the urine. Parent compound was the major residue in urine. In hens and goats, the metabolite profile was the same as in rats, with certain extensions of the pathway.

9. *Metabolite toxicology*. The metabolism profile for thiamethoxam supports the use of an analytical enforcement method that accounts for parent thiamethoxam and CGA-322704. Other metabolites are considered of equal or lesser toxicity than parent compound.

10. *Endocrine disruption*. Thiamethoxam does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There is no evidence that thiamethoxam has any effect on endocrine function in developmental or reproduction studies. Furthermore, histological investigation of endocrine organs in chronic dog, rat and mouse studies did not indicate that the endocrine system is targeted by thiamethoxam.

C. Aggregate Exposure

1. *Dietary exposure*. Chronic dietary exposure was estimated using a Tier I approach by inputting tolerance level residues into the dietary exposure evaluation model (DEEM™) software. The Tier I assessment was partially refined by adjusting for projected percent crop-treated information, and was made using the department of agriculture (USDA) National Food consumption Survey, Continuing Survey of Food Intakes by Individuals (CSFII) 1994-96. The maximum total exposure to the U. S. population (48 States, all seasons) was calculated to be 4.1% of the reference dose of 0.013 mg/kg bwt/day. The maximum exposure to the most sensitive population sub-group, children (1-6 years) was 9.5% of the reference dose (RfD). The inclusion of the maximum concentration of thiamethoxam in water, taken from the highest estimated concentration observed from the generic expected environmental concentration (GENEEC) and screening concentration In GROund water (SCI GROW) models, led to a maximum chronic dietary exposure of 4.5% in the United States population and 10.0% in children (1-6 years old).

Acute dietary exposure was calculated using a Tier III, probabilistic assessment. A distribution of residue data points was included for the typically non-blended commodities of vegetables (tuberous, fruiting, cucurbit, brassica and leafy), pome fruits, meat and milk, while the average field trial value was used for the typically blended commodities of grains (wheat, sorghum, and barley), seed oil (cotton and canola), apple juice and tomato paste and puree. The acute assessment used adjustment for percent of crop treated, and was made using the DEEM software with the Monte Carlo analysis and the CSFII 1994-96 food consumption survey. The margin of exposure (MOE) (NOAEL/exposure) for the United States population (all seasons) at the 99.9th percentile of the exposure distribution was 4,995 using the NOAEL value of 15 mg/kg bwt/day. At the 99.9th percentile, the MOE for the most sensitive population sub-group (non-nursing infants < 1-year old) was 1,012. Inclusion of the drinking water value to the acute assessment led to an MOE of 4,904 at the 99.9th percentile of the United States population, and 1,008 for the population sub-group non-nursing infants < 1-year old. The results of these analyses show that there is reasonable certainty that no harm will result from exposure to dietary residues (including drinking water) of thiamethoxam.

2. *Non-dietary exposure*. Novartis also requests registrations for the use of thiamethoxam on dogs, turf and ornamentals. Novartis has identified potential non-dietary exposures to toddlers for these uses. These exposures include the following scenarios:

i. Incidental non-dietary ingestion of residues on lawns from hand-to-mouth transfer.

ii. Ingestion of thiamethoxam treated grass.

iii. Incidental ingestion of pesticide residues on pets from hand-to-mouth transfer.

According to current EPA policy, these exposures are considered to be short-term oral exposures. EPA does not expect incidental ingestion of pesticide residues on pets from hand-to-mouth transfer to occur during the same period as the exposures from the turf uses. Thus, Novartis considered these exposures in separate estimates of risk. According to current EPA policy, if an oral endpoint is needed for short-term risk assessment (for incorporation of food, water, or oral hand-to-mouth type exposures into an aggregate risk assessment), the acute oral endpoint (acute RfD = 15 mg/kg bwt/day) will be used to incorporate the oral component into aggregate risk. Short-term aggregate exposure is defined by EPA to be average food and water exposure (chronic exposure) plus residential exposure. The short-term risk estimates for the population subgroup children, 1 to 6-years old, is summarized below. This population subgroup was chosen because it has the highest chronic food exposure and because toddlers have the highest exposure from the residential uses. From the results below, Novartis concludes there is no concern associated with the aggregate exposure to thiamethoxam.

3. *Short-term aggregate exposure and risk including turf for children 1 to 6-years old*—i. Dietary exposure estimate including water is 0.001296 mg/kg bwt/day.

ii. Residential exposure from turf is calculated to be 0.00497 mg/kg bwt/day.

iii. Total exposure equals 0.0063 mg/kg bwt/day.

iv. Percent Acute RfD consumed is 0.04%

4. *Short-term aggregate exposure and risk including pet use for children 1 to 6-years old*—i. Dietary exposure estimate including water is 0.001296 mg/kg bwt/day.

ii. Predicted hand to mouth transfer is 0.0341 mg/kg bwt/day.

iii. Total exposure equals 0.035 mg/kg bwt/day.

iv. Percent Acute RfD consumed is 0.23%.

D. Cumulative Effects

The potential for cumulative effects of thiamethoxam and other substances that have a common mechanism of toxicity has also been considered. Thiamethoxam belongs to a new pesticide chemical class known as the neonicotinoids. There is no reliable information to indicate that toxic effects produced by thiamethoxam would be cumulative with those of any other chemical including another pesticide. Therefore, Novartis believes it is appropriate to consider only the potential risks of thiamethoxam in an aggregate risk assessment.

E. Safety Determination

1. *U. S. population*. Using the chronic exposure assumptions and the proposed RfD described above, the aggregate exposure (including drinking water) to thiamethoxam to the U. S. population (48 States, all seasons) was calculated to be 4.5% of the RfD of 0.013 mg/kg bwt/day. Therefore, Novartis concludes that there is reasonable certainty that no harm will result from aggregate chronic exposure to thiamethoxam residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of thiamethoxam, data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat have been considered.

In teratology studies, delayed fetal development was apparent only at maternally toxic doses of thiamethoxam in rats and rabbits. In rabbits, 150 mg/kg/day was clearly toxic to does, causing death, weight loss, reduced food consumption and perineal or vaginal discharge. Developmental toxicity occurred secondary to maternal toxicity and consisted of reduced fetal bwts and an increase in minor skeletal anomalies or variations. Maternal toxicity was also noted at 50 mg/kg/day, consisting of reduced bwts and food consumption and total resorptions in one female. There was no indication of developmental toxicity at 50 mg/kg/day. The NOAEL for maternal toxicity was 15 mg/kg/day and for developmental toxicity was 50 mg/kg/day in rabbits. In rats, 200 and 750 mg/kg/day caused maternal toxicity, but developmental toxicity secondary to maternal toxicity was observed only at 750 mg/kg/day. The NOAEL for maternal toxicity was 30 mg/kg/day and for developmental toxicity was 200 mg/kg/day.

In a rat multigeneration study, parental toxic effects were noted at 2,500 ppm (250 mg/kg/day) and 1,000 ppm (100 mg/kg/day). Offspring bwts were reduced in males and females at 2,500 ppm (250 mg/kg/day) and in females (F1 only) at 1,000 ppm (100 mg/kg/day). The NOAEL for systemic toxicity in adult males was 30 ppm (approximately 3 mg/kg/day, range = 1.3 - 4.3 mg/kg/day) and in adult females was 1,000 ppm (approximately 100 mg/kg/day, range = 59.3 - 219.6 mg/kg/day). The NOAEL for toxicity to offspring was 30 ppm (approximately 3 mg/kg/day, range = 1.3 - 6.4 mg/kg/day). These studies show no evidence that developing offspring are more sensitive to than adults to the effects of thiamethoxam.

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 requirements, the database for thiamethoxam relative to pre- and post-natal effects for children is complete. Further, for thiamethoxam, the developmental studies showed no increased sensitivity in fetuses as compared to maternal animals following in utero exposures in rats and rabbits, and no increased sensitivity in pups as compared to the adults in the multi-generation reproductive toxicity study. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children and that an RfD of 0.013 mg/kg/day is appropriate for assessing aggregate risk to infants and children of thiamethoxam.

Assuming tolerance level residues and adjusting for the percent of crops treated, only 7.0% of the thiamethoxam chronic RfD is utilized in the population subgroup all infant (> 1-year old). Therefore, based on the completeness and reliability of the toxicity database, Novartis concludes that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to thiamethoxam residues.

F. International Tolerances

There are no Codex maximum residue levels (MRLs) established for residues of thiamethoxam on fruiting vegetables, tomato paste, head and stem brassica vegetables, leafy brassica greens, cucurbit vegetables, leafy vegetables, tuberous and corm vegetables, barley grain, barley hay, barley straw, cottonseed, cotton gin by-products, pome fruit, wheat grain, wheat forage, wheat straw, wheat hay, sorghum grain, sorghum forage, sorghum fodder, or milk. (Dani Daniel)

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–181069; FRL 6078–7]

Emamectin Benzoate, Receipt of Application for Emergency Exemptions; Solicitation of Public Comment

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has received a specific exemption request from the Oklahoma Department of Agriculture (hereafter referred to as the ''Applicant'') to use the insecticide emamectin benzoate (CAS 137512–74–4) to treat up to 150,000 acres of cotton to control the beet armyworm. Emamectin benzoate is an unregistered material, and its proposed use is thus use of a ''new'' chemical. Therefore, in accordance with 40 CFR 166.24, EPA is soliciting public comment before making the decision whether or not to grant the exemption.

DATES: Comments must be received on or before May 20, 1999.

ADDRESSES: Three copies of written comments, bearing the identification notation ''OPP–181069,'' should be submitted by mail to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring comments to: Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Follow the instructions under SUPPLEMENTARY INFORMATION. No Confidential Business Information (CBI) should be submitted through e-mail.

Information submitted in any comment concerning this notice may be claimed confidential by marking any part or all of that information as CBI.