after an oral dose of 1 mg/kg. The mentioned metabolite is a minor component in the serum of all 3 species with 5, 4, and 7% of the total radioactivity in serum in the guinea pig, rat and mouse respectively.

Measurement of the concentration of PP796 in the serum of rats and dogs after prolonged dosing showed:

i. No difference in the levels between sexes.

ii. A linear dose - peak serum level response and a linear dose - area under the curve response in dogs throughout the range of doses tested (i.e. 0.15-1.5 mg/kg/day) with slopes of 0.26 ug/ml per 1 mg/kg dose and 1.18 ug.hr/ml per 1 mg/kg dose, respectively. Similar effects were noted in rats in the dose range up to 1.25 mg/kg with slopes of 0.11 ug/ml and 0.52 ug.hr/ml per 1 mg/kg dose, i.e. about half the response seen in dogs.

iii. A biological half-life of < 3 hours in the dog.

There was no evidence to suggest that serum concentration significantly increased or decreased after prolonged administration, hence PP796 is unlikely to be cumulative.

7. *Metabolite toxicology*. The toxicity of metabolites of PP796 has not been studied. Given the level of anticipated exposure and the available animal metabolism data, it is unlikely metabolites of this inert will be of concern.

8. *Endocrine disruption*. There is no evidence that PP796 has hormone disrupting activity.

C. Aggregate Exposure

1. *Dietary exposure*. The residues of PP796 on raw agricultural commodities, due to application in paraquat dichloride formulations, are expected to be negligible. This is due to the low concentration in end use formulations (< 0.2% w/w) and the use pattern for paraquat dichloride, a nonselective herbicide. In the 1997 RED for paraquat dichloride the Theoretical Maximum Residue Concentrations (TMRC) were calculated for the then existing tolerances for paraquat dichloride. Based on the conservative approach (Tier 1), the chronic exposure of the U.S. population, and of the most highly exposed population subgroup (non-nursing infants less than 1-year old), to paraquat was calculated to be 0.000442 and 0.001398 mg/kg body weight/day, respectively (pg. 55 of RED Paraquat Dichloride).

A formulation that contained the maximum proposed amount of PP796 (0.3% w/w) would contain 110 times more paraquat ion than PP796 (assuming a technical containing 33.0% w/w paraquat ion). Therefore, the theoretical chronic exposure can be estimated by dividing the paraquat exposure numbers by 110, resulting in 0.00000402 mg/kg body weight/day for the U.S. population and 0.0000127 mg/kg body weight/day for the most exposed population (non-nursing infants (<1 years old).

i. *Food*. Exposures to PP796 from food are expected to be negligible.

ii. *Drinking water*. Exposures to PP796 from drinking water are expected to be negligible due to the low concentration in the end-use products. There are no aquatic uses of products containing paraquat dichloride.

2. *Non-dietary exposure*. End use products containing paraquat dichloride are restricted use pesticides. There are no residential or homeowner uses. Non-dietary exposure is expected to be negligible.

D. Cumulative Effects

PP796 is only approved for use in paraquat dichloride formulations. There is no evidence for a common mechanism of toxicity with other substances. Therefore, there is no expectation that the use of PP796 as an inert ingredient in paraquat formulations (up to 0.3 % w/w) would contribute to any cumulative toxicity arising from exposure to other substances having a common mechanism of toxicity.

E. Safety Determination

1. *U.S. population*. Based on the toxicity data presented and the very low level of exposure, Syngenta Crop Protection, Inc. believes that there is reasonable certainty that no harm will result to the general U.S. population by increasing the emetic level in paraquat dichloride formulations. PP796 is included in paraquat dichloride formulations as an added safety factor as required by USEPA. The 1987 *Guidance for the Reregistration of Pesticide Products Containing Paraquat Dichloride as the Active Ingredient* states on page 27 that ''The Agency is continuing to require that an emetic cleared under 40 CFR 180.1001(b) and (c) be incorporated into all manufacturing use and end use products containing paraquat. Rationale: Based on the history of poisoning by accidental ingestion of paraquat and partial effectiveness of therapeutic treatment after exposure, the Agency determined that an emetic is needed in formulations to induce rapid vomiting thereby reducing absorption of paraquat.'' Syngenta Crop Protection, Inc. has developed a novel formulation which significantly improves acute oral toxicity of paraquat dichloride formulations in vomiting species. This novel formulation improvement is largely accomplished by adding a gelling agent which slows the movement of paraquat into the intestine where most absorption occurs. Improving human safety is the primary reason for this request, as the emetic level is being increased to ensure adequate absorption from the gel in the stomach.

2. *Infants and children*. Based on the toxicity data presented and the very low level of exposure, Syngenta Crop Protection, Inc. believes that there is reasonable certainty that no harm will result to infants and children by increasing the emetic level in paraquat formulations. PP796 is included in paraquat dichloride formulations as an added safety factor as required by U.S. EPA.

F. International Tolerances

Import tolerances are not required for this inert ingredient. It is listed as a requirement in FAO Specification 56.302/TK (2003). The FAO specification requires that ''An effective emetic, having the following characteristics, be incorporated into the technical. It must be rapidly absorbed (more rapidly than paraquat) and be quick acting. Emesis must occur in about half an hour in at least 50% of cases. It must be an effective (strong) stimulant of the emetic center of the brain, to produce effective emesis. The emetic effect should have a limited 'action period', of about two to three hours, to allow effective treatment of poisoning. It must act centrally on the emetic center in the brain. It must not be a gastric irritant because, as paraquat itself is an irritant, this could potentiate the toxicity of paraquat. It must be toxicologically acceptable. It must have a short half-life in the body (to comply with the need for a limited action period). It must be compatible with, and stable in, the paraquat formulation and not affect the herbicidal efficacy or occupational use of the product. To date, the only compound found to meet these requirements is 2-amino-4,5-dihydro-6-methyl-4-propyl-s-triazole-(1,5a)pyrimidin-5-one (PP796). PP796 must be present in the technical at not less than 0.8 g/l. The method for determination of PP796 content is available from the Plant Protection Officer, FAO Plant Production and Protection Division.''

[FR Doc. 05–12922 Filed 6–29–05; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2005–0149; FRL–7718–9]

Indoxacarb; Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

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 a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket identification (ID) number OPP–2005–0149, must be received on or before August 1, 2005.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Shaja R. Brothers, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–3194; e-mail address: *brothers.shaja@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected entities may include, but are not limited to:

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Copies of this Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket ID number OPP–2005–0149. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access*. You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*.

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and To Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically*. If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also, include this contact information on the outside of any disk or CD ROM you submit, and in any

cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket/*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2005–0149. The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail*. Comments may be sent by e-mail to opp-docket@epa.gov, Attention: Docket ID Number OPP–2005–0149. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

iii. *Disk or CD ROM*. You may submit comments on a disk or CD ROM that you mail to the mailing address identified in Unit I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By mail*. Send your comments to: Public Information and Records Integrity Branch (PIRIB) (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID Number OPP–2005–0149.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA, Attention: Docket ID Number OPP–2005–0149. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

D. How Should I Submit CBI to the Agency?

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket ID number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in FFDCA section 408(d)(2); 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.

List of Subjects

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

Dated: June 21, 2005.

Lois Rossi,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petitions

The petitioner summary of the pesticide petitions is printed below as required by FFDCA section 408(d)(3). The summary of the petitions was prepared by the petitioner and represents the view of the petitioner. 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.

Interregional Research Project No. 4

PP 5E6911 and PP 5E6926

EPA has received pesticide petitions (PP) 5E6911 and 5E6926 from Interregional Research Project No. 4 (IR-4), 681 U.S. Highway #1 South, North Brunswick, NJ 08902-3390 proposing, pursuant to section 408(d) of the (FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of indoxacarb, (*S*)-methyl 7-chloro-2,5-dihydro-2-[[(methoxycarbonyl)[4-(trifluoromethoxy)phenyl] amino]carbonyl]indeno[1,2e] [1,3,4]oxadiazine-4a(3*H*)-carboxylate] and its *R*-enantiomer [(*R*)-methyl 7-chloro-2,5-dihydro-2-[[(methoxycarbonyl)[4-(trifluoromethoxy) phenyl]

amino]carbonyl]indeno [1,2-e] [1,3,4] oxadiazine-4a(3*H*)-carboxylate] in or on the following raw agricultural commodities:

1. PP 5E6911 proposes the establishment of tolerances for leafy greens, except spinach, subgroup 4A at 10 parts per million (ppm), spinach at 3.0 ppm, leafy petioles subgroup 4B at 1.5 ppm, fruit, pome, except pear, group 11 at 1.0 ppm, vegetable, tuberous and corm, subgroup 1C at 0.01 ppm, and okra at 0.5 ppm.

2. PP 5E6929 proposes the establishment of tolerances for pea, southern at 0.1 ppm; peppermint, tops at 10 ppm; and spearmint, tops at 10 ppm.

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.

A. Residue Chemistry

The active ingredient in the end-use formulation, DuPont™ Avaunt® insecticide, is a 75:25 mixture of two isomers, indoxacarb (DPX-KN128) and IN-KN127. Only one of the isomers, indoxacarb (DPX-KN128), has insecticidal activity. Since the insecticidal efficacy is based on the concentration of indoxacarb (DPX-KN128), the application rates have been normalized on an indoxacarb (DPX-KN128) basis. The proposed tolerance expression includes both indoxacarb (DPX-KN128) and IN-KN127, and the residue method does not distinguish between the enantiomers. Therefore, residues are reported as the sum of indoxacarb (DPX-KN128) combined with IN-KN127. Residues of indoxacarb (DPX-KN128) combined with IN-KN127 will be referred to as KN128/KN127.

1. *Plant metabolism*. The metabolism of indoxacarb in plants is adequately understood to support these tolerances. The only significant residue is the parent compound.

2. *Analytical method*. The plant residue enforcement method detects and quanitates indoxacarb in various matrices including sweet corn, lettuce, tomato, broccoli, apple, grape, cottonseed, tomato, peanut and soybean commodity samples by high performance liquid chromotography using ultra-violet detection (HPLC-UV). The limit of quanitation in the method allows monitoring of crops with indoxacarb residues at or above the levels proposed in these tolerances.

3. *Magnitude of residues*. The magnitude of residues for the proposed tolerances is adequately understood.

B. Toxicological Profile

Guideline	Title	Results	Category
870.1100	Acute oral toxicity	Lethal Dose LD_{50}:1,730 mg/kg (male rat) LD_{50}: 268 mg/kg/ (female rat)	Category II
870.1200	Acute dermal toxicity	LD_{50}: >5,000 mg/kg (rat)	Category IV
870.1300	Acute inhalation toxicity	Lethal Concentration LC_{50}: >5.5 mg/L (male rat) (70% MUP)	Category IV
870.2400	Primary eye irritation	Effects reversed within 72 hours (rabbit)	Category III
870.2500	Primary dermal irritation	No irritation (rabbit)	Category IV
870.2600	Skin sensitization	Sensitizer (guinea pig)	

Formulated products are slightly less acutely toxic than indoxacarb.

1. *Acute neurotoxicity study*. In an acute neurotoxicity study, indoxacarb exhibited decreased forelimb grip strength, decreased foot splay, and some evidence of slightly reduced motor activity, but only at the highest doses tested. The no observed adverse effect level (NOAEL) was 100 milligrams/kilogram (mg/kg) for males, and 12.5 mg/kg for females, based on body weight effects in females 50 mg/kg.

2. *Genotoxicty*. Indoxacarb, has shown no genotoxic activity in the following listed *in vitro* and *in vivo* tests: Ames-negative; *in vitro* mammalian gene mutation Chinese hampster ovary/hypoxanthine quanine phopphoribosyl transferase (CHO/HGPRT)-negative; *in vitro* unscheduled DNA synthesis-negative; *in vitro* chromosomal aberration-negative; and *in vivo* mouse micronucleus-negative.

3. *Reproductive and developmental toxicity*. The results of a series of studies indicated that there were no reproductive, developmental or reproductive hazards associated with the use of indoxacarb. In a 2–generation rat reproduction study, the parental NOAEL was 1.5 mg/kg/day. The parental NOAEL was based on observations of reduced weight gain and food consumption for the higher concentration groups of the F_0generation and potential treatment-related changes in spleen weights for the higher groups of the F_1 generation. There was no effect on mating or fertility. The NOAEL for fertility and reproduction was 6.4 mg/kg/day. The offspring NOAEL was 1.5 mg/kg/day, and was based on the reduced mean pup weights noted for the F_1 litters of the higher concentration groups. The effects on pup weights occurred only at a maternal effect level and may have been due to altered growth and nutrition in the dams. In studies conducted to evaluate developmental toxicity potential, indoxacarb was neither reproductive nor uniquely toxic to the conceptus (i.e., not considered a developmental toxin). Developmental studies conducted in rats and rabbits demonstrated that the rat was more susceptible than the rabbit to the maternal and fetal effects of DPX-MP062. Developmental toxicity was observed only in the presence of maternal toxicity. The NOAEL for maternal and fetal effects in rats was 2 mg/kg/day based on body weight effects and decreased food consumption at 4 mg/kg/day. The NOAEL for developmental effects in fetuses was >4 mg/kg/day. In rabbits, the maternal and fetal NOAELS were 500 mg/kg/day based on body weight effects, decreased food consumption in dams and decreased weight and delayed ossification in fetuses at 1,000 mg/kg/day.

4. *Subchronic toxicity*. Subchronic 90–day feeding studies were conducted with rats, mice, and dogs. In a 90–day feeding study in rats, the NOAEL was

3.1 and 2.1 mg/kg/day for males and females, respectively. In male rats, the NOAEL was based on decreased body weight and nutritional parameters, mild hemolytic anemia and decreased total protein and globulin concentration. In female rats, the NOAEL was based on decreased body weight and food efficiency. In a subchronic neurotoxicity study in rats, there was no evidence of neurotoxicity at 11.9 and 6.09 mg/kg/day, the highest dose tested for males and females, respectively. The subchronic NOAEL in dogs (5.0 mg/kg/day, M/F) was based on hemolytic anemia. Erythrocyte values for most dogs were within a range that would be considered normal for dogs in a clinical setting. Mice were less sensitive to indoxacarb than the rats or dogs. NOAELs (23 mg/kg/day, males, 16 mg/kg/day, females) were based on mortality (males only); increased reticulocytes and Heinz bodies and decreased body weight, weight gain, food consumption, food efficiency; and increased clinical signs (leaning to one side and/or with abnormal gait or mobility) (females only). In a 28–day repeated dose dermal study, the NOAEL was 50 mg/kg/day based on decreased body weights, body weight gains, food consumption, and food efficiency in females, and changes in hematology parameters, the spleen and clinical signs of toxicity in both sexes in rats.

5.*Chronic toxicity*. Chronic studies with indoxacarb were conducted on rats, mice, and dogs to determine carcinogenic potential and/or chronic toxicity of the compound. Effects generally similar to those observed in the 90–day studies were seen in the chronic studies. Indoxacarb, was not carcinogenic in rats or mice. The chronic NOAEL in male rats was 5 mg/kg/day based on body weight and nutritional effects. In females, the NOAEL of 2.1 mg/kg/day was based on body weight and nutritional changes, as well as biologically significant hematologic changes at 3.6 mg/kg/day and above. Hemolytic effects were present only through the 6–month evaluation and only in females. The regenerative nature of indoxacarb-induced hemolytic anemia was demonstrated by the absence of significant changes in indicators of circulating erythrocyte mass at later evaluations. In mice, the chronic NOAEL of 2.6 mg/kg/day for males was based on deceased body weight and weight gain effects and food efficiency at 13.8 mg/kg/day and above. The NOAEL for females was 4.0 mg/kg/day based on body weight nutritional effects, neurotoxicity, and clinical signs at 20 mg/kg/day. In dogs, the chronic NOAEL was about 2.3 and 2.4 mg/kg/day in males and females, respectively based on hemolytic effects similar to those seen in the subchronic dog study.

6. *Animal metabolism*. Animal metabolism has been studied in the rat, hen, and cow and is well understood. In contrast to crops, indoxacarb is extensively metabolized in animals.

i. *Poultry*. In poultry, hens were fed at 10 ppm/day for 5 days, 87-88% of the total administered dose was excreted; parent comprised 51-54% of the total dose in excreta. Concentrations of residues in eggs were low, 0.3-0.4 of the total dose, as were the concentrations of residues in muscle, 0.2% of the total dose. Parent and metabolite IN-JT333 were not detected in egg whites; only insecticidally inactive metabolites were identified. Parent and IN-JT333 were found in egg yolks; however, their concentrations were very low-0.01-0.02 ppm. Concentrations of parent and IN-JT333 in muscle were at or below the limit of quantitation, (LOQ) 0.01 ppm.

ii. *Poultry feeding study*. A poultry feeding study was not conducted for the initial section 3 registration because finite concentrations of residues would not be expected based on the low concentration of residues in the metabolism study. However, the Agency has required a poultry feeding study as a condition of registration for indoxacarb. The study was submitted on October 31, 2003. Once the Agency has determined the components of the tolerance expression, poultry meat, fat, by-products and egg tolerances will be proposed.

iii. *Cattle*. For the cow study, the cattle were fed at 10 ppm/day for 5-days; approximately 20% of the total administered dose was excreted in urine and 53-60% was excreted in feces in 5–days. Four- tenths to 1.2% of the total dose in urine was parent indicating extensive metabolism; parent represented 46-68% of the fecal activity. Thus, most residues were not absorbed; those residues that were absorbed were extensively metabolized. Less than 1% of the total administered dose was in milk, most of which was parent compound. The insecticidally active metabolite IN-JT333 was not found in milk. Residues in muscle represented less than 0.01% of the total administered dose most of which was parent. IN-JT333 was not detected in muscle. No other metabolites were seen above 10% of the dose, thus only parent and IN-JT333 were monitored in the cattle feeding study.

iv. *Cattle feeding study*. A cattle feeding study was conducted with indoxacarb at doses of 7.5 ppm, 22.5 and 75 ppm. The mean KN128/KN127 concentrations were proportional to the dosing level in whole milk, skim milk, cream, muscle, fat, liver and kidney. Based on final residue values for the respective commodities contributing to the cattle diet, the anticipated dietary burden in dairy cattle is 51.7 ppm and the anticipated dietary burden in beef cattle is 49.1 ppm. The proposed grape use will not increase the animal dietary burden. Based on standard curves constructed from data in the cattle feeding study, KN128/KN127 concentrations at the 51.7 ppm feeding level are 0.123 ppm for whole milk, 0.033 ppm for skim milk, and 1.46 ppm for cream. The KN128/KN127 concentrations at the 49.1 ppm feeding level are 0.046 ppm for muscle, 1.37 ppm for fat, 0.012 ppm for liver, and 0.026 ppm for kidney. Tolerances have been established at 1.5 ppm in fat (cattle, goat, horse, sheep and hog), 0.05 ppm in meat, 0.03 ppm in meat by-products, 0.15 ppm in milk, and 4.0 ppm in milk fat.

7. *Metabolite toxicology*. In rats, indoxacarb was readily absorbed at the low dose 5 mg/kg, but saturated at the high dose 150 mg/kg. Indoxacarb, was metabolized extensively, based on very low excretion of parent compound in bile and extensive excretion of metabolized dose in the urine and feces. Some parent compound remained unabsorbed and was excreted in the feces. No parent compound was excreted in the urine. The retention and elimination of the metabolite IN-JT333 from fat appeared to be the overall rate determining process for elimination of radioactive residues from the body. Metabolites in urine were cleaved products containing only one radiolabel, while the major metabolites in the feces retained both radiolabels. Major metabolic reactions included hydroxylation of the indanone ring, hydrolysis of the carboxylmethyl group from the amino nitrogen and the opening of the oxadiazine ring, which gave rise to cleaved products. Metabolites were identified by mass spectral analysis, NMR, UV and/or by comparison to standards chemically synthesized or produced by microsomal enzymes.

8. *Endocrine disruption*. Lifespan, and multi-generational bioassays in mammals, acute, and subchronic studies on aquatic organisms and wildlife did not reveal endocrine effects. Any endocrine related effects would have been detected in this definitive array of required tests. The probability of any such effect due to agricultural uses of indoxacarb is negligible.

C. Aggregate Exposure

1. *Dietary exposure*—i.*Food*. The chronic, and acute dietary exposure resulting from the currently approved use of indoxacarb on apples, crop group 5 brassica vegetables, cotton, pears, peppers, sweet corn, tomatoes, eggplant, alfalfa, head and leaf lettuce, peanuts, potatoes, soybeans, cranberries current section 18 use and the proposed uses on grapes, leafy brassica, leafy greens crop subgroup 4A except spinach, spinach, leaf petioles crop subgroup 4B, tuberous and corm vegetables crop subgroup 1C, pome fruits crop group 11 except pear, okra, pea southern and mint are well within acceptable limits for all sectors of the population.

Chronic dietary exposure. The Dietary Exposure Evaluation Model (DEEM), Exponent, Inc., formerly Novigen Sciences, Inc., Version 7.87, was used to conduct the chronic dietary exposure assessment for the U.S. general population with the RfD of 0.02 mg/kg/day based on a NOAEL of 2.0 mg/kg/day from the subchronic rat feeding study, the subchronic rat neurotoxicity study, and the chronic/carcinogenicity study and using an uncertainty factor of 100.

The analysis used overall mean field trial values, processing factors and projected peak percent crop treated values. Secondary residues in milk, meat and poultry products were also included in the analysis. The chronic dietary exposure to indoxacarb for the U.S. population is 0.000185 mg/kg/day. The exposure of the most highly exposed subgroup in the population, children age 1-2 years, is 0.000347 mg/kg/day. The exposure for all infants and females 20+ not pregnant and nursing is 0.000126 mg/kg/day and 0.000179 mg/kg/day respectively. The results of this analysis indicate large margins of safety for each population subgroup, and very low probability of effects resulting from chronic exposure to indoxacarb.

Acute dietary exposure. DEEM, Exponent, Inc., formerly Novigen Sciences, Inc., Version 7.87, was used to conduct the acute dietary exposure assessment for the U.S. general population with the RfD of 0.12 mg/kg/day based on the NOAEL of 12.5 mg/kg in the acute neurotoxicity study and an uncertainty factor of 100. The acute RfD for females 13–50 years of age is 0.02 mg/kg/day, based on the NOAEL of 2 mg/kg/day observed in the developmental rat toxicity study and using an uncertainty factor of 100.

The Tier 3, analysis used distributions of field trial residue data adjusted for projected peak percent crop treated. Secondary residues in milk, meat and poultry products were also included in the analysis. The acute dietary exposure to indoxacarb for the U.S. population is 0.020267 mg/kg/day. The exposure of the most highly exposed subgroup in the population, children age 3–5 years, is 0.005358 mg/kg/day, and the exposure for all infants is 0.018458 mg/kg/day. The results of this analysis indicate large margins of safety for each population subgroup, and very low probability of effects resulting from acute exposure to indoxacarb.

ii. *Drinking water*. Indoxacarb, is highly unlikely to contaminate groundwater resources due to its immobility in soil, low water solubility, high soil sorption, and moderate soil half-life. Based on the PRZM/EXAMS and SCI-GROW models the estimated environmental concentrations (EECs) of indoxacarb and its *R*-enantiomer for acute exposures are estimated to be 6.84 parts per billion (ppb) for surface water and 0.0025 ppb for groundwater. The EECs for chronic exposures are estimated to be 0.316 ppb for surface water and 0.0025 ppb for groundwater. Drinking water levels of comparison (DWLOCs), theoretical upper allowable limits on the pesticides concentration in drinking water, were calculated to be much higher than the EECs. The chronic DWLOCs ranged from 198 to 697 ppb. The acute DWLOCs ranged from 440 to 3,890 ppb. Thus, exposure via drinking water is acceptable.

2. *Non-dietary exposure*. Indoxacarb, product registrations for residential non-food uses have been approved. Non-occupational, non-dietary exposure for DPX-MP062 has been estimated to be extremely small. Therefore, the potential for non-dietary exposure is insignificant.

D. Cumulative Effects

EPA's consideration of a common mechanism of toxicity is not necessary at this time because there is no indication that toxic effects of indoxacarb would be cumulative with those of any other chemical compounds. Oxadiazine chemistry is new, and indoxacarb has a novel mode of action compared to currently registered active ingredients.

E. Safety Determination

1. *U.S. population*. Dietary and occupational exposure will be the major routes of exposure to the U.S. population. The chronic dietary exposure to indoxacarb utilized 1% of the RfD for the U.S. general population. The acute dietary exposure to indoxacarb will utilize 17% of the aPAD acute population adjusted dose for the overall U.S. general population.

Using only Pesticide Handler Exposure Database levels A and B those with a high level of confidence, margin of exposures (MOEs) for occupational exposure are 650 for mixer/loaders, and 1,351 for airblast applicators worst-case. Based on the completeness and reliability of the toxicity data and the conservative exposure assessments, there is a reasonable certainty that no harm will result from the chronic and acute aggregate exposure of residues of indoxacarb, including all anticipated dietary exposure and all othernon-occupational exposures for the U.S. general population.

2. *Infants and children*. The chronic dietary exposure to indoxacarb for the most highly exposed population subgroup, children ages 1–2 and 3–5, utilized 2% of the RfD. For all infants, the chronic exposure accounts for 1% of the RfD. For acute exposure at the 99.9th percentile, children ages 3-5 utilized 30% aPAD, and all infants utilized and 15% aPAD.

Residential uses of indoxacarb/DPX-MP062 have been approved and exposure is calculated to be extremely minimal. The estimated levels of indoxacarb in drinking water are well below the DWLOC. Based on (a) the completeness and reliability of the toxicity data; (b) the lack of toxicological endpoints of special concern; (c) the lack of any indication that children are more sensitive than adults to indoxacarb; and (d) the conservative exposure assessment, there is a reasonable certainty that no harm will result to infants and children from the aggregate exposure of residues of indoxacarb, including all anticipated dietary exposure and all other non-occupational exposures. Accordingly, there is no need to apply an additional safety factor for infants and children.

F. International Tolerances

To date, numerous tolerances exist for indoxacarb residues in various food and feed crops, and foods of animal origin in at least 25 countries.

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ENVIRONMENTAL PROTECTION AGENCY

[IN–162–1; FRL–7930–8]

Adequacy Status of Evansville, Indiana, 8-Hour Ozone Redesignation and Maintenance Plan for Transportation Conformity Purposes

AGENCY: Environmental Protection Agency (EPA).