Ms. Sandy Farmer, U.S. Environmental Protection Agency, OPPE, Regulatory Information Division (2137), 401 M Street, S.W., Washington, DC 20460

And to:

Office of Information and Regulatory Affairs, Office of Management and Budget (OMB), Attention: Desk Officer for EPA, 725 17th Street, N.W., Washington, DC 20503

SUPPLEMENTARY INFORMATION:

Review Requested: This is a request for extension of a currently approved information collection pursuant to 5 CFR 1320.12.

ICR Numbers: EPA ICR No. 0613.06; OMB Control No. 2070–0053.

Current Expiration Date: December 31, 1996.

Title: Trade Secret Clearance Justification.

Abstract: This information collection activity will affect registrants of pesticide products subject to Freedom of Information Act (FOIA) requests. The purpose of the collection is to determine the confidentiality of information submitted to the Agency under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). The collection is usually prompted by a request under the Freedom of Information Act (FOIA) for a record which may be entitled to confidential treatment. The collection instrument consists of nine questions codified under 40 CFR Part 2, Subpart B. A final determination on the releasability of the requested record is issued by EPA upon evaluation of the business's response.

EPA may not disclose information which is described by FIFRA section 10(d)(1) (A), (B), or (C). Under 40 CFR 2.204(a), EPA may take action to determine whether business information is entitled to confidential treatment when a request for disclosure is received under FOIA, when the Agency anticipates receiving a request under FOIA, or when the Agency wishes to determine if information in its possession is confidential. When determining whether information is entitled to confidential treatment, EPA is required by 40 CFR 2.204(e) to notify the affected business and provide an opportunity for comment. The requirements are mandatory to obtain a benefit.

Burden Statement: The annual public reporting and recordkeeping burden for this collection of information is estimated to average 21 hours per response. This estimate includes the time needed for: reading collection request; conferring with EPA; gathering resources and coordinating actions; reviewing information to identify potential confidential portions; processing, compiling, and reviewing claims of confidentiality for accuracy and appropriateness; reporting and substantiating findings; and storing, filing, or maintaining the information. No person is required to respond to a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations are displayed in 40 CFR Part 9.

Respondents/Affected Entities: Entities potentially affected by this action are registrants of pesticide products subject to Freedom of Information Act (FOIA) requests.

Estimated No. of Respondents: 90.

Estimated Total Annual Burden on Respondents: 1890 hours.

Frequency of Collection: once per event.

Changes in Burden Estimates: The administering office is fully automated and no longer utilizes contractor support for this ICR. Overall Agency and respondent costs have increased based on rates provided by the Bureau of Labor Statistics.

According to the procedures prescribed in 5 CFR 1320.12, EPA has submitted this ICR to OMB for review and approval. Any comments related to the renewal of this ICR should be submitted within 30 days, as described above.

Dated: December 3, 1996.

Joseph Retzer,

Director, Regulatory Information Division.

[FR Doc. 96–31273 Filed 12–9–96; 8:45 am]

BILLING CODE 6560–50–P

[FRL–5661–3]

Science Advisory Board; Emergency Notification of a Public Advisory Committee Meeting

Pursuant to the Federal Advisory Committee Act, Public Law 92–463, emergency notice is hereby given that the location of the December 19–20, 1996 meeting of the Integrated Human Exposure Committee (IHEC) of the Science Advisory Board (SAB) has been changed in order to accommodate the expected attendance by the public. This meeting will review the EPA's draft Exposure Factors Handbook, and was announced in the Federal Register for November 18, 1996 (Volume 61, Number 223, [Page 58683–58684).

The new location for the meeting is the Hyatt Arlington Hotel, 1325 Wilson Boulevard, Arlington, VA 22209. The hotel telephone number is 703–525–1234. Anyone desiring additional information should contact Ms. Dorothy Clark, Staff Secretary, Science Advisory Board (1400), US EPA, 401 M Street, SW, Washington, DC 20460, telephone (202) 260–8414, fax (202) 260–7118, or Internet at: clark.dorothy@epamail.epa.gov.

Dated: December 3, 1996.

Donald G. Barnes,

Staff Director, Science Advisory Board.

[FR Doc. 96–31271 Filed 12–9–96; 8:45 am]

BILLING CODE 6560–50–P

[PF–676; FRL–5575–8]

Merck Co., Inc.; Notice of Pesticide Petition Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of Filing.

SUMMARY: This notice announces the filing of a pesticide petition proposing the renewal/reissuance of regulations establishing tolerances for residues of the pesticide chemical abamectin (avermectin B_1) in or on various agricultural commodities. This notice includes a summary of the petition that was prepared by the petitioner, Merck Co., Inc.

DATES: Comments, identified by the docket number PF–676, must be received on or before January 9, 1997.

ADDRESSES: By mail, submit written comments to: Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202.

Comments and data may also be submitted electronically by sending electronic mail (e-mail) to: 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 in 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number PF–676. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic comments on this proposed rule may be filed online at many Federal Depository Libraries. Additional information on electronic submissions can be found in Unit II of this document.

Information submitted as a comment concerning this notice may be claimed

confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. No CBI should be submitted through e-mail. 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.

FOR FURTHER INFORMATION CONTACT: George LaRocca (PM–13), Rm. 204, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202. Mail address: Registration Division (7505C), Environmental Protection Agency, Washington, DC 20460. Telephone (703) 305–6100; e-mail larocca.george@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition from Merck Co., Inc., Agricultural Research and Development, Hillsborough Rd., Three Bridges, NJ 08487. The petition proposes amending 40 CFR 180.449 to renew/reissue the regulations that established tolerances for the insecticide abamectin (avermectin B_1) and its delta-8,9-isomer in or on cottonseed at 0.005 parts per million (ppm); citrus, whole fruit, at 0.02 ppm; citrus oil, at 0.1 ppm; citrus dried pulp, at 0.1 ppm; cattle, meat, at 0.02 ppm; cattle, meat byproducts, at 0.02 ppm; cattle, fat, at 0.015 ppm; milk, at 0.005 ppm; and hops, dried, at 0.5 ppm. These tolerances were originally established in response to pesticide petitions 7F3500, 8F3592, 4E04419, and food additive petition 8H5550. The petition also proposes to establish a tolerance in or on the raw agricultural commodity potatoes at 0.005 ppm. As required by section 408(d) of FFDCA, as recently amended by the Food Quality Protection Act, Merck included in the petition a summary of the petition and authorization for the summary to be published in the Federal Register in a notice of receipt of the petition. The summary represents the views of Merck; EPA is in the process of evaluating the petition. As required by section 408 (d)(3) EPA is including the summary as a part of this notice of filing. EPA has made minor edits to the summary for the purpose of clarity.

I. Merck Co., Inc.'s Petition Summary

This is a petition by Merck Co., Inc. (Merck), under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), as most recently amended by the Food Quality Protection Act (FQPA), asking that the Environmental Protection Agency (EPA) issue permanent tolerances without time limits for pesticide chemical residues consisting of the insecticide abamectin (avermectin B_1) and or its delta 8,9-isomer in or on the following food items: cottonseed; citrus, whole fruit; citrus, oil; citrus, dried citrus pulp; hops, dried; milk; cattle, meat; cattle, meat byproducts; cattle, fat; and potatoes. These tolerances were originally requested in petitions 7F3500, 8F3592, 4E04419, and 5F04508, and food additive petition 8H5550.

On April 30, 1996, the time-limited tolerances for abamectin use on cottonseed, citrus food and feed items, milk, and cattle food items expired. A proposal by EPA to extend the tolerances was published in the Federal Register; no public comments were received in response. However, the Agency did not publish a final rule prior to the enactment of FQPA. On October 21, 1996, in response to procedural guidance from EPA, Merck submitted to EPA a request for reissuance of the tolerances. With one exception (potatoes), the requested tolerances would replace the time-limited abamectin tolerances that have been issued in the past by EPA and that recently expired or will soon expire. Merck requested that EPA issue permanent tolerances for these commodities, saying that the time limitations associated with these earlier tolerances were a result of aquatic exposure issues, and that it understood that the Agency no longer imposes time limitations on tolerances because of non-dietary issues.

A. Residue Data

Abamectin (also known as avermectin B_1) is an effective miticide/insecticide that is used on various crops at the maximum use rate of 0.025 pounds active ingredient per acre. Residue data covering all the uses associated with the tolerances requested by this petition have been previously submitted to EPA for review and have been found by EPA to support the requested tolerances and preharvest intervals. See 54 FR 23209, May 31, 1989 (cottonseed); 54 FR 31836, Aug. 2, 1989 (citrus food and feed items, cattle food items, and milk); and 60 FR 47529, Sept. 13, 1995 (hops). Merck has submitted practical analytical methods (high density liquid chromatography—fluorescence, with crop-specific cleanup methods) for detecting and measuring levels of abamectin and its delta 8,9-isomer in or on food with residues at or above the proposed tolerance levels. EPA has provided information on these methods to EPA, and the methods are available to anyone interested in pesticide residue enforcement at the address listed under ''FOR FURTHER INFORMATION CONTACT'' above.

B. Abamectin Safety Data

To date Merck has submitted approximately 78 toxicology studies, including the following principal studies, to support the tolerances for abamectin (studies conducted with the delta 8,9-isomer of abamectin are noted):

1. *Acute studies.* A rat acute oral study with a LD_{50} of 4.4 to 11.8 mg/kg (males) and 10.9 to 14.9 mg/kg (females).

A rabbit acute dermal study with a LD_{50} >2,000 mg/kg. A rat acute inhalation study with a LC_{50} >5.73 mg/L.

A primary eye irritation study in rabbits which showed irritation.

A primary dermal irritation study in rabbits which showed no irritation.

A primary dermal sensitization study in guinea pigs which showed no skin sensitization potential.

An acute oral toxicity study in monkeys with a no observed adverse effects level (NOAEL) of 1.0 mg/kg based upon emesis at 2.0 mg/kg.

2. *Subchronic studies.* A rat 8–week feeding study with a NOAEL of 1.4 mg/kg/day based upon tremors.

A rat 14–week oral toxicity study with a NOAEL of 0.4 mg/kg/day, the highest dose tested.

A dog 12–week feeding study with a NOAEL of 0.5 mg/kg/day based upon mydriasis.

A dog 18–week oral study with a NOAEL of 0.25 mg/kg/day based upon mortality.

A CD-1 mouse 84–day feeding study with a NOAEL of 4 mg/kg/day based upon decreased body weights.

3. *Chronic studies.* A rat 53–week oncogenicity feeding study, negative for oncogenicity, with a NOAEL of 1.5 mg/kg/day based upon tremors.

A CD-1 mouse 94–week oncogenicity feeding study, negative for oncogenicity, with a NOAEL of 4 mg/kg/day based upon decreased body weights.

A dog 53–week chronic feeding study, with a NOAEL of 0.25 mg/kg/day based upon mydriasis.

4. *Developmental toxicity studies.* An oral teratology study in the CF–1 mouse with a maternal NOAEL of 0.05 mg/kg/day based upon decreased body weights and tremors. The fetal NOAEL was 0.20 mg/kg/day based upon cleft palates.

An oral teratology study with the delta 8,9-isomer in CF–1 mice with a maternal NOAEL of 0.10 mg/kg/day based upon decreased body weights. The fetal NOAEL was 0.06 mg/kg/day based upon cleft palate.

An oral teratology study in rabbits with a maternal NOAEL of 1.0 mg/kg/

day based upon decreased body weights and tremors. The fetal NOAEL was 1.0 mg/kg/day based upon clubbed feet.

An oral teratology study in rats with a maternal and fetal NOAEL at 1.6 mg/kg/day, the highest dose tested.

An oral teratology study with the delta 8,9-isomer with a maternal NOAEL in CF–1 mice that expressed P-glycoprotein greater than 1.5 mg/kg/day, the highest and only dose tested. No cleft palates were observed in fetuses that expressed normal levels of P-glycoprotein, but fetuses with low or no levels of P-glycoprotein had increased incidence of cleft palates.

5. *Reproductive effects study.* A two-generation study in rats with a NOAEL of 0.12 mg/kg/day in pups based upon retinal folds, decreased body weight, and mortality. The NOAELs for systemic and reproductive toxicity were 0.4 mg/kg/day.

6. *Mutagenicity studies.* The Ames assays conducted with and without metabolic activation were both negative.

The V-79 mammalian cell mutagenesis assays conducted with and without metabolic activation did not produce mutations. In an alkaline elution/rat hepatocyte assay, abamectin was found to induce single strand DNA breaks without significant toxicity in rat hepatocytes treated *in vitro* at doses greater than 0.2 mM. This *in vitro* dose of 0.2 mM is biologically unobtainable *in vivo,* due to the toxicity of the compound. However, at these potentially lethal doses, *in vivo* treatment did not induce DNA single strand breaks in hepatocytes. In the mouse bone marrow assay, abamectin was not found to induce chromosomal damage. Merck has also conducted many studies and accumulated a great deal of clinical and follow-up experience with regard to ivermectin, a closely similar human and animal drug.

C. Toxicity Issues

1. *Acute toxicity.* Typical symptoms of classical CNS abamectin/ivermectin acute toxicity include mydriasis (dilated pupils, a marker effect occurring at relatively low exposure levels); fatigue or lethargy; and tremors. At sufficiently high exposure levels, coma and sometimes death may result. Once exposure ceases, recovery in affected living animals is rapid (typically within a few days).

Some species of animals are more sensitive generally to this classical pattern of abamectin toxicity than other species. In particular, a subpopulation of CF–1 mice and the neonatal rat have been observed to be sensitive to abamectin/ivermectin toxicity. Merck research has attributed the sensitivity of the subpopulation of CF–1 mice to the absence of P-glycoprotein, a major component of the blood-brain barrier. Neonatal rat sensitivity has been attributed in part to the lack of a fully developed blood-brain barrier. The neonatal blood-brain barrier is not complete until after 2 weeks following birth, while the blood-brain barrier in humans is completed pre-natally. The extensive human use of ivermectin has not identified a subpopulation of humans with deficient P-glycoprotein. Furthermore, the animal and human data bases do not indicate increased concerns for infants and children.

2. *Developmental effects.* Tests of abamectin and ivermectin have been conducted in a variety of species, and ivermectin is widely used as a human and animal drug. In livestock species there is no suggestion that ivermectin is a developmental toxicant. In mice and rabbits there is evidence that dosing with either abamectin or ivermectin may produce malformations, but only at doses that are clearly maternally toxic as well. However, the delta 8,9-isomer of abamectin has been shown to produce cleft palate malformations in the CF–1 mouse at dose levels that are not maternally toxic and that are much lower than the dose levels that show any indication of developmental toxicity in other species or in other mouse strains. Merck research has shown that the subpopulation of CF–1 mice with these malformations have inherited a genetic deficiency that prevents or severely limits their production of a P-glycoprotein which is a principal factor of the blood-brain barrier and which Merck hypothesizes may perform a protective function in fetal development as well, perhaps by playing a role in the blood-placenta barrier. Based upon extensive use of ivermectin in humans without observed adverse effects, this deficiency is not expected to occur in humans.

3. *Other toxicity issues.* There are no nonthreshold effects and no other toxic endpoints of concern. The oncogenicity assays and chronic feeding studies revealed no indication of carcinogenic potential. Abamectin was found to be non-mutagenic.

D. Exposure Analysis for Threshold Effects

1. *Chronic exposure assessments.* EPA's chronic dietary exposure assessments for abamectin currently use a reference dose (RfD) of 0.0004 mg/kg/day based upon a NOAEL of 0.12 mg/kg/day from effects on neonatal pups in the rat multigeneration reproduction study and an uncertainty factor of 300 (including an additional modifying factor of 3 to account for the severity of the effects).

As noted above, this acute toxicity in rat pups results solely from their exposure to abamectin in the milk they ingest. It is well understood that abamectin concentrates in fat and that rat milk has considerably more fat content than that of most other species (including humans), so that the exposure level for the rat dams considerably understates the exposure level of the affected rat pups. As discussed earlier, the blood-brain barrier of the neonatal rat pup is not fully formed until a week or more after birth, while in humans the barrier is complete well before birth. Due to these differences between rats and humans, using the neonatal rat to model risks to infants arguably is inappropriate; certainly use of the 0.0004 mg/kg/day RfD derived from the abamectin level in the rat dams' diet introduces additional conservative safety factors. Additional assurance comes from the absence of adverse effects in studies using neonatal and juvenile monkeys and from the absence of adverse effects in nursing human infants whose mothers have been treated with ivermectin.

Notwithstanding these issues, Merck has calculated chronic exposure estimates and compared them to this RfD. Using mean anticipated residues, adjusted for percent crop treated with abamectin, the chronic exposure for the overall U.S. population was estimated to be 0.000005 mg/kg/day, which is approximately 1.4% of the RfD. For infants, exposure was similarly estimated to be 0.000005 mg/kg/day (1.4% of the RfD). The exposure estimates for the two most highly exposed population subgroups, children 1 to 6 years old and children 7 to 12 years old, were 0.000013 mg/kg/day (3.2% of the RfD) and 0.000008 mg/kg/day (2.1) of the RfD), respectively.

2. *Acute exposure assessments.* In evaluating the potential hazard of abamectin acute exposure for women of childbearing age, EPA currently uses a NOAEL of 0.05 mg/kg/day for maternotoxic effects of abamectin in CF–1 mice and a NOAEL of 0.06 mg/kg/day for developmental effects of the delta 8,9-isomer in CF–1 mice. To assess the potential hazard of acute exposure of infants and children, EPA uses the rat 2-generation reproduction study NOAEL of 0.12 mg/kg/day based upon the toxicity observed in the nursing pups.

The relevance of the neonatal rat model has already been discussed. As to the relevance of the CF–1 mouse studies, Merck research has shown that both the induction of cleft palate in fetuses and the induction of maternal

toxicity at low dose levels result from a heritable genetic deficiency that precludes some animals of that strain from producing P-glycoprotein. In a recent study, where dams that expressed P-glycoprotein were treated with the delta 8,9-isomer of abamectin and mated to males with and without P-glycoprotein, every fetus that did not inherit the ability to express P-glycoprotein developed cleft palate while every fetus that inherited the ability to express P-glycoprotein fully was free of the malformation. Additionally, in the dams (all of whom were chosen because they possessed the ability to express the P-glycoprotein) no effects were seen at the 1.5 mg/kg/day dose (the only dose tested), in contrast to the much lower maternotoxicity NOAELs (as low as 0.05 mg/kg/day) seen in comparable studies using abamectin or its delta 8,9-isomer in CF–1 mice that had not been tested for ability to express P-glycoprotein. Epidemiological studies of humans treated with ivermectin, as well as breeding-animal studies on ivermectin conducted to obtain its approval as animal drug and surveys of adverse reaction reports (billions of treatments have been administered to animals) all indicate a lack of a human population susceptible to the induction of birth defects by ivermectin or abamectin. Accordingly, the CF–1 mouse is not an appropriate model to assess the toxicity of the avermectins.

Despite these issues, Merck has incorporated these toxicity endpoints from the CF–1 mouse into acute exposure assessments. (For purposes of simplification, Merck has used the NOAEL of 0.05 mg/kg/day for acute exposure assessments for the overall U.S. population and also for women of childbearing age.) These assessments show that the margins of exposure (MOEs) at the 95th percentile of exposure (using a Monte Carlo analysis conducted in accordance with Tier 3 of EPA' June 1996 ''Acute Dietary Exposure Assessment''guidance document) are significantly greater than the EPA standard of 100 for all subpopulations. The 95th percentile of exposure for the overall U.S. population was estimated to be 0.000023 mg/kg/day (MOE of 2,146), while that for women greater than 13 years of age was 0.000017 mg/kg/day (MOE of 2,970). For children 1 to 6 years old, the 95th percentile of exposure was estimated to be 0.000042 mg/kg/day (MOE of 2,863), while that for children 7 to 12 years old was 0.000030 mg/kg/day (MOE of 3,965). For infants, the 95th percentile of exposure was estimated to be 0.0028 mg/kg/day (MOE of 4,244).

E. Aggregate Exposure

The dietary assessments (both acute and chronic) accounts for all anticipated dietary exposure for tolerances that are subject to this request (citrus and derivatives, cottonseed, meat, meat byproducts, milk, and hops), and all other active and pending tolerances for abamectin. The other active tolerances are for tomatoes, strawberries, celery, lettuce, cucurbits, peppers, apples, pears, almonds, and walnuts. The tolerance petition for potatoes is pending. The assessments also take into account the use on grapes under an emergency exemption.

Additional uses of abamectin include a bait for fire ants, an indoor crack and crevice treatment, and a roach bait; however, significant exposure from these products is not likely. The fire ant bait contains approximately 0.011% abamectin and is used primarily in the southern portion of the United States, where the fire ant is most prevalent. Post application exposure resulting from mound-directed treatment is considered unlikely, and significant exposure from the broadcast treatment is also unlikely since the treatment rate is very low (1.0 lb of bait, containing only 50 mg of abamectin, per acre). In a recent exposure study using the crack and crevice treatment, no measurable air or surface residues were detected. Significant exposure is not expected from the roach bait because of the child resistant safety packaging and the essentially non-existent vapor pressure of abamectin.

Based upon the available studies of abamectin's fate in the environment, there is no reason to expect human exposure to residues of abamectin in drinking water. It has been clearly demonstrated that abamectin does not leach.

The typical therapeutic dose of ivermectin as a human drug is 200 μg/kg (0.2 mg/kg). Merck is in the process of quantitatively assessing the total dietary exposure resulting from abamectin and ivermectin uses. Generally, use of ivermectin in food-producing animals is only once per year and the ivermectin residues in most treated animals are below the level of detection.

F. Endocrine Effects

There is no evidence that abamectin is an endocrine disrupter. Evaluation of the rat multigenerational study demonstrated no effect on the time to mating or on the mating and fertility indices, suggesting no effects on the estrous cycle, on mating behavior, or on male or female fertility at doses up to 0.4 mg/kg/day, the highest dose tested. Furthermore, the range finding study demonstrated no adverse effect on female fertility at doses up to 1.5 mg/kg/day, the highest dose tested. Similarly, chronic and subchronic toxicity studies in mice, rats, and dogs did not demonstrate any evidence of toxicity to the male or female reproductive tract, or to the thyroid or pituitary (based upon organ weights and gross and histopathologic examination). In the developmental studies, the pattern of toxicity observed does not seem suggestive of any endocrine effect. Finally, experience with ivermectin in breeding animals, including sperm evaluations in multiple species, shows no adverse effects suggestive of endocrine disruption.

G. International Tolerances

The U.S. tolerances for pears and citrus are greater than the Codex proposals, reflecting the differences in how the United States and Codex CCPR treat the highest residue values from field studies. The differences in tolerances for cottonseed and milk are the result of differences in the limits of detection of the analytical methods accepted by the two organizations. Assuming label directions are followed, actual anticipated residues in foods in commerce should not be affected by the different tolerances, since the same residue database has been used to set both the Codex and U.S. tolerances.

H. Safety to Infants and Children

Merck's petition notes that EPA has evaluated abamectin repeatedly since its introduction in 1985 and has found repeatedly that the level of dietary exposure is sufficiently low to provide ample margins of safety to guard against any potential adverse effects of abamectin. The FQPA authorizes the employment of an additional safety factor of up to 10X to guard against the possibility of prenatal or postnatal toxicity, or to account for an incomplete database on toxicity or exposure. Merck states that the database for abamectin is complete and argues that there is no need for an additional safety factor because of the conservatism in the end points selected for risk assessment. Additionally, there is much more information regarding human risk potential than is the case with most pesticides, because of the widespread animal-drug and human-drug uses of ivermectin, the closely related analog of abamectin.

It is the opinion of Merck that the use of an additional safety factor to address

risks to infants and children is not necessary. The established endpoints for abamectin in the CF–1 mouse and the neonatal rat have been shown by Merck to be overly conservative. Similar endpoints for ivermectin are not used by the Food and Drug Administration to support the allowable daily intake for ivermectin residues in food from treated animals.

No evidence of toxicity was observed in neonatal rhesus monkeys after 14 days of repeated administration of 0.1 mg/kg/day (highest dose tested) and in juvenile rhesus monkeys after repeated administration of 1.0 mg/kg/day (highest dose tested). The comparative data on abamectin and ivermectin in primates also clearly demonstrate the dose response for exposure to either compound is much less steep than that seen in the neonatal rat. Single doses as high as 24 mg/kg of either abamectin or ivermectin in rhesus monkeys did not result in mortality; however, this dose was more than two times the LD_{50} in the adult rat and more than 20 times the LD_{50} in the neonatal rat. The absence of a steep dose-response curve in primates provides a further margin of safety regarding the probability of toxicity occurring in infants or children exposed to avermectin compounds. The significant human clinical experience and widespread animal drug uses of ivermectin without systemically toxic, developmental, or postnatal effects supports the safety of abamectin to infants and children.''

II. Administrative Matters

A record has been established for this notice of filing under docket number PF–677 (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 4:00 p.m., Monday through Friday, excluding legal holidays. The public record is located in Room 1132 of the Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

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.

The official record for this notice of filing, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer all comments received electronically into printed, paper form as they are received and will place the paper copies in the official record which will also include all comments submitted directly in writing. The official record is the paper record maintained at the address in ''ADDRESSES'' at the beginning of this document.

List of Subjects

Environmental protection, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping.

Authority: 7 U.S.C. 136a.

Dated: December 3, 1996.

Stephen L. Johnson,

Acting Director, Office of Pesticide Programs.

[FR Doc. 96–31303 Filed 12–09–96; 8:45 am]

BILLING CODE 6560–50–F

[FRL–5655–4]

State Program Requirements; Approval of Application by Oklahoma to Administer the National Pollutant Discharge Elimination System (NPDES) Program

AGENCY: Environmental Protection Agency.

ACTION: Final approval of the Oklahoma Pollutant Discharge Elimination System under the Clean Water Act.

SUMMARY: On November 19, 1996, the Regional Administrator for the Environmental Protection Agency (EPA), Region 6, approved the application by the State of Oklahoma to administer and enforce the National Pollutant Discharge Elimination System (NPDES) program for regulating discharges of pollutants into waters of the State. The authority to approve state programs is provided to EPA in Section 402(b) of the Clean Water Act (CWA). The approved state program i.e., the Oklahoma Pollutant Discharge Elimination System (OPDES) program is a partial program to the extent described in this Notice (see section titled ''Scope of the OPDES program), which will operate *in lieu* of the EPA administered NPDES program pursuant to Section 402 of the CWA. The OPDES program will be administered by the Oklahoma Department of Environmental Quality (ODEQ). In making its decision, EPA has considered all comments and issues raised during the publicly noticed comment period. Summaries of the comments and EPA responses are contained in this notice. The comments and public hearing record are contained in the administrative record supporting this notice.

EFFECTIVE DATE: November 19, 1996. Because CWA section 301(a) prohibits new discharges until they are authorized by an NPDES permit, this action is immediately effective to avoid further suspension of permitting actions in Oklahoma and the unnecessary burden such a suspension would impose on new dischargers.

FOR FURTHER INFORMATION CONTACT: Ms. Ellen Caldwell at U.S. EPA, Region 6, Water Quality Protection Division, 1445 Ross Avenue, Dallas, Texas 75202, or by calling (214) 665–7513, or electronically at

CALDWELL.ELLEN@EPAMAIL.EPA.GOV

or Norma Aldridge, Department of Environmental Quality, Water Quality Division, 1000 N.E. 10th Street, Oklahoma City, Oklahoma 73117–1212, or by calling (405) 271–5205.

SUPPLEMENTARY INFORMATION: Oklahoma's application for OPDES program approval was submitted on June 10, 1996, and final supplements were received on August 20, 1996. The documents were described in the Federal Register Notice of August 29, 1996, (61 FR 45420) in which EPA requested comments and gave notice of public hearing. Further notice was also provided by way of publication published on August 28, 1996, in The Lawton Constitution, the Daily Oklahoman, the Tulsa World, the McAlester News Capital & Democrat, the Guymon Daily Herald, and the Woodward News. Copies of the application were made available at the addresses below and could also be purchased from the State for the cost of $358.65 (the cost of the principal documents, i.e the Attorney General's Statement, Memorandum of Agreement, Program Description, and the Enforcement Management System all without their associated appendices is $163.35). An electronic copy of the documents stored on computer disk was provided at no cost to interested parties which supply disks to ODEQ for that purpose. (Citizens may still request a disc copy and should supply 3 new, 3.5'' high density/double sided microdisk. The documents will be in WordPerfect 6.0.) EPA provided copies of the public notice to permitted facilities, Indian tribes, and other federal and state agencies.

As a part of the public participation process, both a public meeting and hearing were held in MidWest City, Oklahoma, on September 30, 1996. The public meeting provided as an informal question and answer session, and began