vigor is reduced by the toxin introduced by the feeding of pear psyllas which ultimately reduces overall yield. The Applicants state that the need for a method of reducing the overwintering adult population before they lay appreciable numbers of eggs in the spring is critical to pear psylla control. The only effective pre-bloom materials for some years were the synthetic pyrethroids, permethrin and fenvalerate. When widespread resistance to these materials became evident in the psylla population by 1987–88, the Applicants state that cyfluthrin was used under section 18 exemptions in 1988 – 1992, and was found to be efficacious.

In 1993, this use of fenoxycarb was first requested by Washington state, who claimed that resistance to cyfluthrin was being observed. However, the toxicology data available at that time for fenoxycarb did not support this use, and cyfluthrin was again used under section 18 during the 1993 season. In the 1994, 1995 and 1996 seasons, both Washington and Oregon requested exemptions for this use. Adequate toxicology data were available to support the use under section 18, and the exemptions were subsequently granted. The Applicants claim that most of the pear psylla populations are now resistant to cyfluthrin, and are therefore again requesting this use of fenoxycarb for control of pear psylla in pears.

The Applicants wish to treat up to 18,900 acres of pear trees in Oregon, and up to 26,000 acres in Washington. This would translate to a possible total of 4,725 pounds of active ingredient [(a.i.)] (18,900 lbs. product) in Oregon, and up to 6,500 lbs. a.i. (26,000 lbs. product) in Washington. Up to two applications would be made per growing season, at a maximum rate of 2 oz. a.i. (8 oz. product) per acre, diluted in water to make a minimum spray volume of 50–400 gallons per acre. Application of fenoxycarb would not be allowed by air or through chemigation equipment. Fenoxycarb would be used pre-bloom and would not be allowed to be applied during or after pear bloom, nor to open blossoms of weeds or cover crops. Negligible residues are expected because this is a pre-bloom only use and available residue chemistry data indicate non-detectable residues will occur.

The regulations governing section 18 require publication of a notice of receipt in the **Federal Register** and solicitation of public comment on an application for a specific exemption proposing the first food use of an active ingredient. Normally, a notice of receipt shall give the public 15 days in which to file comments on the application. The Administrator may shorten or eliminate the comment period if she determines that the time available for a decision on the application requires it (40 CFR §166.24). The comment period has been eliminated for these specific exemption requests because implementation of the Food Quality Protection Act delayed application processing, the use season had started and available data indicate that there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposure and all other exposures for which there is reliable information.

A record has been established for this notice under docket number [OPP–181038] (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 public record is located in Room 1132 of the Public Response and Program Resource 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, 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. Accordingly, interested persons may submit written views on this subject to the Filed Operations Division at the address above.

List of Subjects

Environmental protection, Pesticides and pests, Emergency exemptions.

Dated: March 24, 1997.

Stephen L. Johnson,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 97–8399 Filed 4–1–97; 8:45 am]

BILLING CODE 6560–50–F

[PF–726; FRL–5594–9]

ISK Biosciences Corporation; Pesticide Tolerance Petition Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of filing.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of time-limited tolerances for residues of the fungicide, chlorothalonil and its metabolite, 4-hydroxy-2,5,6-trichloroisophthalonitrile in or on non-bell peppers. This notice includes a summary of the petition that was prepared by the petitioner, ISK Biosciences Corporation.

DATES: Comments, identified by the docket control number [PF-726], must be received on or before, May 2, 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 Highway, Arlington, VA.

Comments and data may also be submitted electronically be 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 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by docket control number [PF-726]. Electronic comments on this notice 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 document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment

that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Cynthia Giles-Parker, Product Manager (PM) 22, Registration Division (7505W), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 229, CM #2, 1921 Jefferson Davis Highway, Arlington, VA, (703) 305–6226; e-mail: gilesparker.cynthia@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition (PP 6F4676) from ISK Biosciences Corporation, 5966 Heisley Road, P.O. Box 8000, Mentor, Ohio 44061-8000 proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. section 346a(d), to amend 40 CFR 180.275 by establishing a time-limited tolerance for a period of 2 years for residues of the fungicide chlorothalonil and its metabolite, 4-hydroxy-2,5,6-trichloroisophthalonitrile in or on the raw agricultural commodity non-bell peppers at 5.0 parts per million (ppm). ISK Biosciences Corporation has committed to providing additional residue data during this 2–year period from trials conducted in Mexico in support of a permanent tolerance. The proposed analytical method is by electron capture gas chromatography.

EPA has determined that the petition contains 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.

As required by section 408(d) of the FFDCA, as recently amended by the Food Quality Protection Act, ISK Biosciences Corporation 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 ISK Biosciences Corporation. 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. ISK Biosciences' Petition Summary

A. Residue Chemistry Data

1. *Plant/animal metabolism*. The nature of the residue of chlorothalonil in plants and animals, including ruminants, is well understood. Chlorothalonil is not systemic in plants. Any chlorothalonil residue found on non-bell peppers occurs as a surface residue. Chlorothalonil is rapidly metabolized in the ruminant and is not transferred to meat and milk from the dietary consumption by animals. Furthermore, chlorothalonil is not stable in meat or milk.

2. *Analytical method*. An adequate analytical method (gas chromatography) is available for enforcement purposes. The method is listed in the Pesticide Analytical Manual, Vol. II (PAM II).

3. *Magnitude of the residues*. Residue data from studies conducted with non-bell peppers support a tolerance of 5.0 ppm for combined residues of chlorothalonil and its metabolite, 4-hydroxy-2,5,6-trichloroisophthalonitrile in or on the raw agricultural commodity.

B. Toxicological Profile

The following studies on file with the Agency support this petition:

1. *Acute toxicity*. Acute toxicity studies include an acute oral rat study on technical chlorothalonil with an LD_{50} >10,000 milligrams/kilograms (mg/kg), an acute dermal toxicity study in the rabbit with an LD_{50} >20,000 mg/kg, a 4–hour inhalation study with finely ground technical chlorothalonil resulting in a LC_{50} of 0.092 mg/L (actual airborne concentration), a primary eye irritation study with irreversible eye effects in the rabbit at 21 days, a primary dermal irritation study showing technical chlorothalonil is not a dermal irritant, and a dermal sensitization study showing technical chlorothalonil is not a skin sensitizer.

2. *Genotoxicity*. The mutagenic potential of chlorothalonil has been evaluated in a large number of studies covering a variety of endpoints. The overall conclusion is that chlorothalonil is not mutagenic.

Mutagenicity studies with chlorothalonil include gene mutation assays in bacterial and mammalian cells; *in vitro* and *in vivo* chromosomal aberration assays; DNA repair assays in bacterial systems; and cell transformation assays. All were negative with the following two exceptions:

Chlorothalonil was positive in an *in vitro* chromosomal aberration assay in CHO cells without metabolic activation but was negative with metabolic activation.

In vivo chromosomal aberration studies in rats and mice were negative and one study in the Chinese hamster was equivocal. The results of this study could not be confirmed in a subsequent study at higher doses. The conclusion was that chlorothalonil does not cause chromosome aberrations in bone marrow cells of the Chinese hamster. It can be concluded that chlorothalonil does not have clastogenic potential in intact mammalian systems.

In bacterial DNA repair tests, chlorothalonil was negative in *Bacillus subtilis*, but was positive in Salmonella typhimurium. In an *in vivo* DNA binding study in rats with ^{14}C-chlorothalonil, there was no covalent binding of the radiolabel to the DNA of the kidney, the target organ for chlorothalonil toxicity in rodents.

3. *Developmental and reproductive toxicity*. A developmental toxicity study with rats given gavage doses of 0, 25, 100, and 400 mg/kg body weight/day from days 6 through 15 of gestation resulted in a no observed effect level (NOEL) for maternal toxicity of 100 mg/kg/day based on increased mortality, reduced body weight, and a slight increase in early resorptions at the highest dose. There were no developmental effects observed at any dose in this study.

A developmental toxicity study in rabbits given gavage doses of 0, 5, 10, or 20 mg/kg/day on days 7 through 19 of gestation resulted in a maternal NOEL of 10 mg/kg/day. Effects observed in the dams in the high-dose group were decreased body weight gain and reduced food consumption. There were no developmental effects observed in this study.

A two-generation reproduction study in rats fed diets containing 0, 500, 1,500 and 3,000 ppm resulted in a reproductive NOEL of 1,500 ppm (equivalent to 115 mg/kg/day) based on lower neonatal body weights by day 21. There were no effects seen on any other reproductive parameter at any dose level in this study.

4. *Subchronic toxicity*. i. A subchronic toxicity study (90 days) was conducted in rats at doses of 0, 1.5, 3.0, 10, and 40 mg/kg bwt. Treatment related hyperplasia and hyperkeratosis of the forestomach was observed at the two highest dose levels. Although the initial histopathological evaluation did not demonstrate any nephrotoxicity, a subsequent evaluation observed a treatment-related increase in hyperplasia of the proximal tubule epithelium at 40 mg/kg bwt in the male rats but not in the females. The no effect level for renal histopathology was 10

mg/kg bwt in males and 40 mg/kg bwt in females.

ii. A 90–day oral toxicity study was conducted in dogs with dose levels of technical chlorothalonil of 15, 150, and 750, mg/kg bwt/day. The two highest dosages resulted in lower body weight gain in male dogs. The no observed adverse effect level (NOAEL) was 15 mg/kg/day. There were no macroscopic or microscopic tissue alterations related to chlorothalonil and there were no signs of renal toxicity.

iii. Two 21–day dermal toxicity studies have been conducted with technical chlorothalonil. In the initial study doses of 50, 2.5, and 0.1 mg/kg bwt/day were administered to rabbits. The NOEL for systemic effects was greater than 50 mg/kg bwt/day and the NOEL for dermal irritation was 0.1 mg/kg bwt/day.

A subsequent 21–day dermal study was conducted in male rats, to specifically evaluate the potential for nephrotoxicity in this laboratory species following dermal dosing. In this study the doses were 60, 100, 250, and 600 mg/kg bwt/day. The NOEL for nephrotoxicity was greater than 600 mg/kg bwt/day.

5. *Estrogenic effects.* Based upon all of the chronic toxicity, teratogenicity, mutagenicity, and reproductive studies conducted with chlorothalonil and its metabolites, there were no results which indicate any potential to cause estrogenic effects or endocrine disruption. These effects would have manifested themselves in these studies as reproductive or teratogenic effects, or by producing histopathological changes in estrogen sensitive tissues such as the uterus, mammary glands, or the testes. Thus, it can be concluded based upon the *in-vivo* studies, that chlorothalonil does not cause estrogenic effects.

6. *Chronic toxicity.*—i. A 12–month chronic oral toxicity study in Beagle dogs was conducted with technical chlorothalonil at dose levels of 15, 150, and 500 mg/kg/day. The NOAEL was 150 mg/kg/day based on lower blood albumin levels at the highest dose. There was no nephrotoxicity observed at any dose in this study. This study replaced an old outdated study that was not conducted under current guidelines and did not use the current technical material.

ii. A chronic feeding/carcinogenicity study with Fischer 344 rats fed diets containing 0, 800, 1,600 or 3,500 ppm (equivalent to 0, 40, 80, or 175 mg/kg bwt/day) for 116 weeks in males or 129 weeks in females, resulted in a statistically higher incidence of combined renal adenomas and carcinomas. At the high dose, which was above the MTD, there was also a statistically significant higher incidence of tumors of the forestomach in female rats.

iii. In a second chronic feeding/carcinogenicity study with Fischer 344 rats, designed to define the NOEL for tumors and the preneoplastic hyperplasia, animals were fed diets containing 0, 2, 4, 15, or 175 mg/kg/day. The NOEL in this study, based on renal tubular hyperplasia, was a nominal dose of 2 mg/kg bwt/day. Because of the potential for chlorothalonil to bind to diet, the 2 mg/kg bwt/day dose, expressed as unbound chlorothalonil is 1.8 mg/kg bwt/day. The NOEL for hyperplasia and hyperkeratosis of the forestomach was 4 mg/kg bwt/day or a dose of 3.8 mg/kg bwt/day based on unbound chlorothalonil.

iv. A 2–year carcinogenicity study, conducted in CD-1 mice at dietary levels of 0, 750, and 1,500 or 3,000 ppm (equivalent to 0, 107, 214, or 428 mg/kg/day), resulted in a statistically higher incidence of squamous cell carcinomas of the forestomach in both sexes, and a statistically higher incidence of combined renal adenomas/carcinomas in only the male mice receiving the low dose. There were no renal tumors in any female mouse in this study.

v. A 2-year carcinogenicity study in male CD-1 mice for the purpose of establishing the no effect level for renal and forestomach effects, was conducted at dietary levels of 0, 10/15, 40, 175, or 750 ppm (equivalent to 0, 1.4/2.1, 5.7, 25, or 107 mg/kg/day). The NOEL level for renal effects was 40 ppm and the NOEL for forestomach effects was 15 ppm. This study did not duplicate the results from the previous study where a statistically higher incidence of renal tumors, when compared to controls, was observed at 750 ppm.

In 1987, EPA's Office of Pesticide Programs' Toxicology Branch Peer Review Committee classified chlorothalonil as a B2 (probable human carcinogen), based on evidence of carcinogenicity in the forestomach and kidneys of rats and mice. The Agency currently regulates chlorothalonil as a B2 carcinogen although ISK Biosciences Corporation has provided a significant amount of mechanistic data indicating that the tumors result from a threshold mechanism. A potency factor, Q1*, of 0.00766 $(mg/kg/day)^{-1}$ has been used by the Agency when conducting mathematical modeling to estimate carcinogenic risk to man. ISK Biosciences Corporation believes that because the nephrotoxicity seen in the rat is due to a threshold mechanism, any risk associated with chlorothalonil can be managed using the margin of safety (exposure) approach.

Numerous metabolism and toxicology studies indicate that chlorothalonil is non-genotoxic and produces a species-specific renal toxicity in the rat that eventually may lead to tumor formation through an epigenetic mechanism.[1] Studies comparing metabolism and toxicological effects in dogs with those in rats demonstrate that the renal effects observed in the rat are due to the exposure of the kidney of the rat to significant levels of nephrotoxic thiol metabolites of chlorothalonil. In the dog, no thiol metabolites are found and there are no toxic effects seen in kidneys of dogs dosed with high levels of chlorothalonil.

7. *Reference Dose (RfD).* The no effect level for chlorothalonil in the rat is 1.8 mg/kg bwt based on the nephrotoxicity observed in the chronic rat study. The no effect level in the dog was 15 mg/kg bwt in the 90–day study and 150 mg/kg bwt based on the one-year study. No effect levels for maternal toxicity from developmental studies are 10 mg/kg bwt in rabbits and 100 mg/kg bwt in the rat. The no effect level for pup growth in the reproduction study was 1,500 mg/kg bwt which would be most conservatively estimated as equating to approximately 75 mg/kg bwt. Data indicate that the nephrotoxicity in the rat is produced through a mechanism for which there is a clear threshold. In a study which measured cell turnover in the rat kidney with bromodeoxyuridine (BRDU) immunohistochemical staining, a NOEL was established at 1.5 mg/kg bwt. Other chronic studies have established the NOEL for hyperplasia in the kidney to be 1.8 mg/kg bwt. If all the available toxicity data in laboratory animals is considered without regards to its applicability to humans, the lowest NOEL for any adverse effect would be 1.5 mg/kg bwt/day. Because the mechanism of toxicity which is related to the tumor formation in the kidney has been shown to have a threshold, the use of the normal 100 fold safety factor in conjunction with the 1.5 mg/kg no effect level would produce a RfD which would provide more than adequate safety for all of the possible effects seen in any laboratory animal.

In two recent reviews of chlorothalonil by the Joint Meeting of Pesticide Residue Experts (1990 and 1992), and the review by the World Heath Organization's International Program for Chemical Safety, these

[1] ''Mechanistic Interpretation of the Oncogenicity of Chlorothalonil in Rodents and an Assessment of Human Relevance,'' by Drs. C. F. Wilkinson and J. C. Killeen, Regulatory Toxicology and Pharmacology 24: 69-84 (1996), Article No. 006.

esteemed groups concluded that the rat was not the appropriate species to use in consideration of the risk assessment for man. They concluded that the dog was the more appropriate species for determination of subchronic and chronic effects. If the toxicological data for the dog were used, the NOEL would be at least 15 mg/kg bwt, based on the most recent 90-day study in the dog.

Therefore, under the most conservative scenario (using the toxicological data in the rat), the RfD would be 1.8 mg/kg bwt/day divided by a 100 fold safety factor or 0.018 mg/kg bwt/day with a threshold model being used for carcinogenic risk assessment. In the scenario that uses the toxicological data in the dog, the reference dose would be 15 mg/kg bwt/day, divided by a safety factor of 100 or 0.15 mg/kg bwt/day.

C. Aggregate Exposure

The following is a description of the likelihood of exposure to chlorothalonil from various routes.

1. *Dietary exposure—i. food*. ISK Biosciences Corporation has conducted a dietary exposure analysis for chlorothalonil and its metabolite, 4-hydroxy-2,5,6-trichloroisophthalonitrile (SDS-3701) in or on non-bell peppers utilizing EPA's Dietary Risk Evaluation System (DRES) based on the 1977-78 Food Consumption Survey. The results demonstrate that the dietary exposure from anticipated residues of 0.5 ppm contributed from non-bell peppers is 0.00000218 mg/kg bwt/day for the U.S. population or 0.0121% of the RfD.

The Agency had calculated that the exposure of the general population from existing published tolerances for chlorothalonil is 0.000134 mg/kg bwt/day or 0.744 percent of the RfD.

ii. *Drinking water*. Chlorothalonil was included for monitoring in the National Survey of Pesticides in Drinking Water Wells conducted by EPA. No chlorothalonil residues were detected in any of the 1,300 community water systems and domestic wells (using methodology for chlorothalonil having a limit of detection [LOD] of 0.06 mg/l and limit of quantitation of 0.12 mg/l). The absence of chlorothalonil detections in the National Survey provides adequate information to conclude that chlorothalonil is not a contaminant in drinking water wells and that the population is not exposed to chlorothalonil in these water sources. These findings are consistent with the known physical/chemical properties of chlorothalonil including low water solubility (0.9 ppm) and high affinity for organic matter including soil. It has also been demonstrated that chlorothalonil does not leach into groundwater from applications made to growing crops.

Aerobic aquatic metabolism studies with chlorothalonil establish a half-life in natural aquatic habitats of less than 10 hours, depending on environmental conditions. Considering the short half-life of chlorothalonil in natural water/sediment systems and that surface water is filtered and treated prior to consumption, chlorothalonil is not likely to be present in drinking water obtained from natural surface water systems.

An exposure estimate, based on surface water concentration recently cited by EPA, would conclude that the average concentration in surface water would be less than 0.002 ppb. Assuming that everyone in the U.S. consumed untreated surface water, the exposure to chlorothalonil of the general population would be less than 5.8×10^{-7} mg/kg bwt/day. This would be a worst case scenario, which would greatly overestimate exposure.

2. *Non-dietary exposure*. Potential non-dietary exposures to chlorothalonil may result from the following uses of chlorothalonil. In each case, the exposure would be from the dermal route and only for an intermittent duration. The two 21–day dermal studies that have been conducted in the rabbit and rat indicate that there is no nephrotoxicity associated with the dermal exposure to chlorothalonil at dose levels up to 600 mg/kg/day. Therefore, the exposures from the uses of chlorothalonil listed below, would not be expected to add to the carcinogenic risk associated with chlorothalonil.

i. *Golf course uses*. Chlorothalonil products are commonly applied to golf course tees and greens to control a broad complex of turf diseases. Application to golf course fairways is much less common.

Golf is not a game played by infants or small children, therefore no exposure to infants and children would be anticipated.

ii. *Residential owner uses*. Applications of chlorothalonil products to home lawns are rare. Thus, there is very little exposure to chlorothalonil related to use on residential turf. Applications to roses and other ornamentals in home gardens is also a minor use of chlorothalonil.

iii. *Paint*. Chlorothalonil is used in paints and stains for control of mildew and molds on exterior surfaces of buildings. Chlorothalonil is also occasionally used for interior paints, but this use represents only a small proportion of the chlorothalonil used in paints. About 2% of the chlorothalonil used in paint is used in interior paint; however, only 0.2% or less of interior paints in the United States contain chlorothalonil. In paints chlorothalonil is tightly bound within the paint matrices; thus, effective control of mildew may last for several years and the potential for exposure is very limited.

iv. *Grouts*. Chlorothalonil is used in cement tile grouts, also for control of mildew and molds. Chlorothalonil is bound within the grout matrices and very little is available for exposure. This is a minor use of chlorothalonil and non-occupational dermal exposure of humans to chlorothalonil from this source is extremely low.

v. *Wood treatment*. Chlorothalonil is not currently used for pressure-treating wood. It is used for control of sapstain as a surface treatment on rough-cut, newly-sawn lumber to protect it from molds and mildews while drying. Being a surface residue, it is removed during the finishing operations prior to sale of the wood. Chlorothalonil does not occur in structural wood used for residential or occupational scenarios.

D. Cumulative Effects

ISK Biosciences has considered the potential for cumulative effects of chlorothalonil and other substances that have a common mechanism of toxicity. Chlorothalonil is a halogenated benzonitrile which readily undergoes displacement of the 2, 4, and 6 chlorines by glutathione and other thiol containing amino acids and proteins. In the rat, the thiol metabolites are sufficiently absorbed to produce a nephrotoxic effect. In dogs where this absorption does not occur, nephrotoxicity does not occur. ISK Biosciences does not have any information to indicate that toxic effects observed in rats occur through a mechanism which is common to any other agricultural chemical. Thus, consideration of common mechanisms of toxicity is not appropriate at this time.

Chlorothalonil should not be confused with chlorinated hydrocarbon pesticides which have significantly different chemical and biological properties.

E. Safety Determination

1. *U.S. population*. ISK Biosciences Corporation has conducted a risk assessment for chlorothalonil in or on non-bell peppers using the 1977-78 Food Consumption Survey and a potency factor, Q1*, of 0.00766 $(mg/kg/day)^{-1}$ and has determined that oncogenic dietary risks associated with

potential exposure using an anticipated residue of 0.5 ppm, would 1.7×10^{-8}.

The Agency has used a linearized model to estimate the carcinogenic risk associated with chlorothalonil, whereas ISK Biosciences believes that a threshold based model is appropriate. Using the overestimated exposure estimates of EPA, with a threshold based model and using the conservative RfD of 0.018 mg/kg bwt/day, the margin of safety for the general population would exceed 10,000 and the margin of safety for infants and children would exceed 7,000. Using corrected exposure estimates would obviously yield larger margins of exposure. Using a conservative RfD of 0.018 mg/kg/day, as the Agency has done in recent DRES analyses, and incorporating corrections needed in exposure values for mushrooms and several other lesser corrections, ISK Biosciences Corporation calculated the overall dietary exposure to ''anticipated residues'' of chlorothalonil from all registered uses and pending uses of chlorothalonil to be 0.36% of the RfD for the general U.S. population.

Because the worst case assumption for human exposure from drinking water indicate that exposure would be only 1% of the dietary exposure, the risk assessment is not significantly altered by considering the exposure from drinking water.

2. *Infants and children*. There is a complete data base for chlorothalonil which includes pre- and post-natal developmental toxicity data as well as mechanistic data related to the rodent specific nephrotoxicity observed in subchronic and chronic studies. The toxicological effects of chlorothalonil in rodents are well understood. Chlorothalonil has a low level of toxicity in dogs.

In a two-generation reproduction study in rats, all reproductive parameters investigated showed no treatment-related effects except pup weight gain. Specifically, the weights of pups exposed to chlorothalonil were comparable to controls at parturition through day 4 of lactation. It was only after day 4 of lactation, when the pups begin to consume the test diet, that body weight gain lags behind controls. This only occurred at the highest dose tested; 3,000 ppm. The dose of chlorothalonil the pups would receive would be far in excess of the estimated adult dose of 150 mg/kg bwt/day (3,000 ppm divided by 20). The doses for the pups could have easily exceeded 500 mg/kg bwt/day. Dose levels of 375 mg/kg bwt and above have been shown to significantly affect body weight in the rat. Therefore, the reduction of body weight gain observed in the reproduction study is considered to be comparable to the effects that have been observed in older rats. The NOEL for this effect was 1,500 ppm.

In developmental toxicity studies conducted in the rat and the rabbit, chlorothalonil did not cause any developmental effects even at dose levels that produced significant maternal toxicity. In the rabbit a dose level of 20 mg/kg bwt caused maternal toxicity, but there were no developmental effects and in the rat, a dose level of 400 mg/kg bwt caused maternal toxicity without developmental toxicity.

The extensive data base that is available for chlorothalonil is devoid of any indication that chlorothalonil would represent any unusual or disproportionate hazard to infants or children. Therefore, there is no need to impose an additional 10x safety factor for infants or children. The standard uncertainty factor of 100x should be used for all segments of the human population when calculating risks associated with chlorothalonil.

F. International Tolerances

There is currently no maximum residue level set for chlorothalonil on non-bell peppers by the Codex Alimentarius Commission.

II. Public Record

A record has been established for this notice under docket control number [PF–726] (including comments and data submitted electronically as described below). A public version of the 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 public record is located in Room 1132 of the Public Response and 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 rulemaking, 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 rulemaking record which will also include all comments submitted directly in writing. The official rulemaking record is the paper record maintained at the address in **ADDRESSES** at the beginning of this document.

List of Subjects

Environmental protection, Administrative practice and procedure, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: March 24, 1997.

Stephen L. Johnson,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 97–8388 Filed 4–1–97; 8:45 am]

BILLING CODE 6560–50–F

FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collections Being Reviewed by the Federal Communications Commission

March 25, 1997.

SUMMARY: The Federal Communications Commissions, as part of its continuing effort to reduce paperwork burden invites the general public and other Federal agencies to take this opportunity to comment on the following information collection, as required by the Paperwork Reduction Act of 1995, Public Law 104–13. An agency may not conduct or sponsor a collection of information unless it displays a currently valid control number. No person shall be subject to any penalty for failing to comply with a collection of information subject to the Paperwork Reduction Act (PRA) that does not display a valid control number. Comments are requested concerning (a) whether the proposed collection of information is necessary for the proper performance of the functions of the Commission, including whether the information shall have practical utility; (b) the accuracy of the Commission's burden estimate; (c) ways to enhance the quality, utility, and clarify of the information collected; and (d) ways to minimize the burden of the collection of information on the respondents, including the use of automated collection techniques or other forms of information technology.

DATES: Persons wishing to comment on this information collection should submit comments June 2, 1997.

ADDRESSES: Direct all comments to Dorothy Conway, Federal Communications Commissions, Room