mail) and the organization (if any) you will represent. Written comments should be submitted to Ms. Pozun at the above address prior to the meeting date.

Providing Oral or Written Comments at SAB Meetings

The Science Advisory Board expects that public statements presented at its meetings will not be repetitive of previously submitted oral or written statements. In general, each individual or group making an oral presentation will be limited to a total time of ten minutes. For teleconference meetings, opportunities for oral comment will usually be limited to no more than three minutes per speaker and no more than fifteen minutes total. Written comments (at least 35 copies) received in the SAB Staff Office sufficiently prior to a meeting date (usually one week before the meeting), may be mailed to the relevant SAB committee or subcommittee; comments received too close to the meeting date will normally be provided to the committee at its meeting.

Additional information concerning the Science Advisory Board, its structure, function, and composition, may be found on the SAB Website (http://www.epa.gov/sab) and in *The Annual Report of the Staff Director* which is available from the SAB Publications Staff at (202) 564–4533 or via fax at (202) 501–0323.

Meeting Access: Individuals requiring special accommodation at this teleconference meeting, including wheelchair access to the conference room, should contact the appropriate DFO at least five business days prior to the meeting so that appropriate arrangements can be made.

Dated: August 18, 1999.

Donald G. Barnes,

Staff Director, Science Advisory Board.

[FR Doc. 99–21942 Filed 8–23–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–868; FRL–6069–6]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–868, must be received on or before September 23, 1999.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

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

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 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: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Kathryn Boyle	Rm. Q616, CM #2, 703–305–6304, e-mail:boyle.kathryn@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Cynthia Giles-Parker (PM 22).	Rm. 229, CM #2, 703–305–7740, e-mail: giles-parker.cynthia@epamail.epa.gov.	Do.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–868] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number (insert docket number) and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

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

Dated: August 13, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required

by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. Centre Internationale d'Etudes du Lindane (C.I.E.L.) and its member company Inquinosa S.A.

PP 9F5057

EPA has received a pesticide petition (9F5057) from Centre Internationale d'Etudes du indane (C.I.E.L.) and its member company Inquinosa S.A., c/o Charles A. O'Conner III, Esq., McKenna & Cuneo, L.L.P., 1900 K Street, NW., Washington, DC 20006-1108, proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of lindane in or on the raw agricultural commodities (RAC) broccoli, brussels sprouts, cabbage, cauliflower, celery, collards, kale, kohlrabi, lettuce, mustard greens, spinach, and Swiss chard at 0.05 parts per million (ppm), corn (grain) at 0.01 ppm, and corn (forage and fodder) and radish at 0.1 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Use of lindane as a seed treatment results in uptake of lindane and extensive metabolism within the plant. The primary residue in RAC was parent lindane. Lindane metabolizes in plants through the same processes found in mammalian animals, i.e. dehydrogenation, dehydrochlorination, hydroxylation, and conjugation. Thus, the terminal metabolites for plants and animals are the same chorophenols, chlorobenzenes, etc. and conjugates of these classes of compounds.

2. *Analytical method*. A multi-residue method is currently being used by the United States food and drug administration (FDA) to determine lindane residues in raw and processed agricultural commodities, in order to monitor for tolerance compliance. The pesticide analytical manual (PAM) method for non-fatty foods by GLC, PAM 212.1, has also been validated for determination of lindane residues.

3. *Magnitude of residues*. Lindane residues were determined in mustard, radish, field corn, sweet corn, and spinach, using radio-labeled lindane applied as a seed treatment. Residues of parent lindane were: Radish roots; 0.030 ppm; mustard leaves, 0.017 ppm; field corn foliage, 0.008 ppm; sweet corn foliage 0.012 ppm; field corn grain, < 0.01 ppm; sweet corn grain, < 0.01 ppm; and spinach, < 0.02 ppm. Residues in animal tissues, milk, and eggs will be negligible, based on transfer factors determined in animal feeding studies, the low residue levels in animal feed items, and the limited market share.

B. Toxicological Profile

1. *Acute toxicity*—i. A rat acute oral study with an LD_{50}s of 88 to 150 milligram kilogram (mgkg) (Toxicity Category II).

ii. A rabbit acute dermal study with an LD_{50} of 500 to 1,000 mg/kg (Toxicity Category II).

iii. A primary eye irritation study in the rabbit showing moderate eye irritation, (EPA Category II).

iv. A primary dermal irritation study in the rabbit showing moderate irritation, (EPA Category II).

2. *Genotoxicty*. Lindane has been subject to a large number of gentoxicity assays using many different test systems. *In vitro* Ames/*Salmonella* mutagenicity assays were negative for mutagenic potential. Other point mutations assays in bacteria were also negative. The induction of chromosomal aberrations was not observed *in vivo* assays and *in vitro* assays were either negative or equivocal. Sister chromatid exchange and micronucleus assays were negative. Dominant lethal assays gave variable results.

3. *Reproductive and developmental toxicity*. Lindane is not considered to be a reproductive or a developmental toxin. In a 2-generation reproduction study, the no-observed adverse effect level (NOAEL) for reproductive and developmental toxicity was 20 ppm. In a developmental toxicity study, the rat maternal NOAEL and the developmental NOAEL were 5 mg/kg/day. The developmental NOAEL for the rabbit was 10 mg/kg/day while the maternal NOAEL was less than 5 mg/kg/day based on reduced food consumption, reduced weight gain, slight tachypnea and lethargy.

4. *Subchronic toxicity*. Ninety–day feeding studies were conducted in mice and rats with lindane. The NOAEL for the mouse study was greater than 10 ppm highest dose tested (HDT), for the rat study, the NOAEL was 10 ppm (0.75 mg/kg/day). Renal effects observed were related to a_{2u}-globulin and are not relevant to human safety. Hepatocellular hypertrophy and neurotoxicity were observed at the higher dose levels. A 14 week inhalation study in mice had a NOAEL 0.3 mg/cubic meter. In a 90-day inhalation study in rats, the NOAEL was 0.6 mg/cubic meter. Ninety-day dermal toxicity studies have been conducted in rats and rabbits. In both species, the NOAELs were 10 milligrams per kilograms bodyweight per day (mg/kg bw/day).

5. *Chronic toxicity*. A 2-year feeding study was conducted in dogs with lindane. The NOAEL was for this study 50 ppm. In a 2-year feeding study in rats, hepatocellular hypertrophy and renal effects related to a_{2u}-globulin were observed above the NOAEL of 0.7 (males) and 0.8 (females) mg/kg bw/day. Carcinogenicity - Lindane is not carcinogenic to rats. A 2-year combined chronic toxicity/oncogenicity study in the rat was negative for carcinogenicity and had a chronic toxicity NOAEL of 10 ppm (0.47 mg/kg bw/day) based on a slight increase in mortality and effects on the liver. A total of 8 mouse oncogenicity studies have been conducted in several strains of mice. The results of these studies have been variable and none of the studies are considered by the Agency to be adequate for a cancer risk assessment. A ninth study is in progress.

6. *Animal metabolism*. The metabolism of lindane has been thoroughly investigated. Lindane does not appear to bioaccumulate in tissues. Lindane is rapidly absorbed and metabolized. The metabolism of lindane occurs via several different pathways. Major routes of metabolism include stepwise elimination of chlorines and conjuations with sulfates and glucuronides. Another pathway is via the formation of mercapurates.

7. *Metabolite toxicology*. Dietary residues are comprised of lindane and a variety of metabolites. The dietary residues are qualitatively the same as those formed in the rat and have thus been bioassayed in the available toxicity studies. These metabolites are not considered to present a significant toxicological risk.

8. *Endocrine disruption*. There was no evidence that exposure to lindane had any effect on reproduction, fertility or mating indices, development or maturation of embryos, or development, growth and survival of offspring in the battery of short-term, chronic,

reproductive and, developmental mammalian, avian and aquatic studies conducted. There were no gross or microscopic pathologic effects in endocrine organs or endocrine-sensitive tissues, or in any reproductive organs, tissues or endpoints that were considered related to exposure to lindane. There is negligible risk of endocrine disruption in humans or wildlife as a result of these proposed uses.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Estimates of dietary exposure to residues of lindane from the proposed uses are extremely low. A reference dose (RfD) of 0.0047 was established by EPA based upon a 100-fold uncertainty factor and the NOAEL in the chronic rat study. Maximum dietary residues from the requested uses result in an exposure that is less than 2% of the reference dose (RfD) for children 1-6 years, the most sensitive sub-population.

ii. *Drinking water*. Given the use pattern (seed treatment) strong soil binding characteristics and low soil mobility of lindane, the risk of significant ground and surface water contamination and exposure via drinking water is considered to be negligible.

2. *Non-dietary exposure*. There are no currently registered pesticidal uses of lindane that would result in non-dietary exposure.

D. Cumulative Effects

Lindane falls into the common category of chlorinated hydrocarbon insecticides however, there is no information to suggest that lindane has a common mechanism of mammalian toxicity with any other pesticide. It is not appropriate to combine exposures in this case.

E. Safety Determination

1. *U.S. population*. As presented above, the exposure of the U.S. general population to lindane is low, and the risks, based on comparisons to the reference dose, are negligible. Margins of safety are very large.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of lindane, CIEL considered data from developmental toxicity studies in the rat, and rabbit and a 2-generation reproduction study in rats. No developmental or reproductive effects were observed up in the absence of parental toxicity in any of the three studies. Using the same conservative assumptions that were made previously for the dietary exposure analysis for the U.S. general population, the percent of the RfD utilized by pre-adult sub-populations is less than 2%. CIEL concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to lindane residues.

F. International Tolerances

International maximum residue levels (MRLs) have been established for aproximately 30 commodities. The MRL values for commodities represented in this petition include; 2 ppm on head lettuce and spinach, 1 ppm on kohlrabi and radish, and 0.5 ppm on brussels sprouts, cabbage, cauliflower, and cereal grain.

2. K-I Chemical U.S.A. Inc.

PP 8F4941

EPA has received a pesticide petition (8F4941) from K-I Chemical U.S.A. Inc., Westchester Financial Center, 11 Martine Avenue, 9th Floor, White Plains, NY, 10606 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of prohexadione calcium (cyclohexanecarboxylic acid, 3, 5-dioxo-4-(1-oxopropyl)-, ion(1-), calcium, calcium salt) in or on the raw agricultural commodity peanut nutmeat at 1.0, peanut hay at 0.6, pome fruit at 3.0, and cattle meat byproduct (kidney) at 0.1 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

In the **Federal Register** of August 5, 1998 (63 FR 41828) (FRL-5799-6) EPA issued a notice of filing of pesticide petition 8F4941 from K-I Chemical U.S.A. Inc. at the above address proposing to amend 40 CFR part 180 by establishing tolerances for residues of prohexadione calcium in or on the raw agricultural commodities (RAC) peanut nutmeat and hay at 0.8 and 0.4 ppm respectively. EPA has received an amendment to PP 8F4941 from K-I Chemical U.S.A. Inc., proposing to amend the earlier petition by increasing the tolerances for residues on peanut nutmeat and hay, and by proposing to amend 40 CFR part 180 by establishing tolerances on pome fruit and cattle meat byproducts. This notice contains information submitted in addition to that contained in the August 5, 1998 notice.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism in plants (peanuts and apples) and animals (goats and poultry) is adequately understood.

2. *Analytical method*. The proposed analytical method involves homogenization, extraction, filtration, partition and cleanup, methylation and analysis by a gas chromatography system with a mass selective detector. The limit of quantitation (LOQ) is 0.05 ppm.

3. *Magnitude of residues*. Twelve peanut trials were conducted with prohexadione calcium in the principle peanut growing regions of the country (NC, SC, GA, AL, FL, OK, TX). Prohexadione calcium was applied to peanuts three times at the rate of 0.125 lbs active ingredient acre (ai/A). Peanut hay and nutmeat were analyzed for residues of prohexadione (free acid). Prohexadione residues in the nutmeat ranged from < 0.05 to 0.30 ppm. Residues in hay ranged from < 0.05 to 0.26 ppm. The residue values in this study were reported as prohexadione free acid. The highest residue values for peanut nutmeat and hay were converted to prohexadione calcium equivalents for the tolerance expression. Therefore, the proposed tolerance for prohexadione calcium in/on peanut nutmeat is 1.0 ppm based on the conversion of the highest peanut nutmeat raw agricultural commodities (RAC) ppm for prohexadione (0.795 ppm) to prohexadione calcium equivalents (0.895 ppm). The proposed tolerance for prohexadione calcium in/on peanut hay is 0.6 ppm and it is based on the conversion the highest peanut hay RAC ppm for prohexadione (0.457 ppm) to prohexadione calcium equivalents (0.539 ppm).

A study was conducted to determine the level of prohexadione calcium derived residues in or on processed commodities. Peanut samples treated at an exaggerated rate were processed into peanut meal and refined oil. Peanut nutmeat and processed commodities were analyzed for prohexadione. Residues in the meal were less than in the nuts, and no residues were detected in the refined oil. Therefore, there was no concentration of prohexadione residues in processed commodities.

Twenty apple trials were conducted with prohexadione calcium in the principle apple growing regions of the country (NY, PA, NC, VA, MI, WI, CO, UT, CA, WA, ID, and OR) in order to determine the magnitude of prohexadione residues in/on apples.

Apple trees received two foliar applications of prohexadione calcium with a 21 day interval between each application and the second application was made 45 days prior to harvest (45 day PHI). The target rate for each application was 0.85 lbs a.i./A and a maximum seasonal application rate of 1.7 lbs a.i./A. Prohexadione residues in apples ranged from < 0.05 ppm to 2.23 ppm. The residue values in this study were reported as prohexadione free acid. The highest apple RAC ppm for prohexadione (2.23 ppm) when converted to prohexadione calcium equivalents is 2.63 ppm.

Apple samples treated with prohexadione calcium at an exaggerated rate were processed according to commercial practices into washed apples, wet pomace, and fresh juice. Samples of unwashed apples, washed apples, wet pomace, and juice were analyzed for residues of prohexadione. Residues of prohexadione in the washed apples, pomace and juice were less than in the unwashed whole fruit. Therefore, there is no concentration of prohexadione residues in the processed commodities and separate tolerances for processed fractions are not necessary.

Eight pear trials were conducted with prohexadione calcium in the principle pear growing regions of the United States (NY, CA, WA, ID, and OR) to determine the magnitude of prohexadione calcium residues in/on pear raw agricultural commodity. Pear trees received one foliar application of prohexadione calcium at an application rate of 1.7 lbs a.i./A. Pears were harvested 45 days after the application (45 day PHI). Prohexadione calcium residues in pears ranged from 0.220 ppm to 0.985 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Based on available acute toxicity data prohexadione calcium does not pose any acute toxicity risks. The acute toxicity studies place technical prohexadione calcium and its formulated end-use products in acute toxicity category III for acute dermal; and in acute toxicity category IV for acute oral, acute inhalation, eye irritation, and skin irritation and the technical material is not a skin sensitizer.

2. *Genotoxicty*. Ames Test (1 Study; point mutation): Negative; *In Vitro* V79 Cells CH/HGPRT Locus Mammalian Cell Mutation Assay (1 Study; point mutation): Negative; *In Vitro* CHO Cytogenetic Assay (1 Study; Chromosome Damage): Negative; *In Vivo* Mouse Micronucleus (1 Study; Chromosome Damage): Negative; *In Vivo* Rat Bone Marrow Cytogenetic Assay (1 Study; Chromosomal Damage): Negative; Rec Assay (1 Study; DNA damage and repair): Negative; *In Vitro Rat Hepatocyte (1 Study; DNA damage and repair): Negative.*

Prohexadione calcium has been tested in a total of 7 genetic toxicology assays consisting of *in vitro* and *in vivo* studies. Based on the results described above, it can be stated in summary that prohexadione calcium did not show any mutagenic activity when tested under the conditions of the studies mentioned above. Therefore, prohexadione calcium does not pose a mutagenic hazard to humans.

3. *Reproductive and developmental toxicity*. The reproductive and developmental toxicity of prohexadione calcium was investigated in a 2-generation rat reproduction study as well as in rat and rabbit teratology studies. The 2-generation rat reproduction study was conducted at dose levels of 0, 500, 5,000, and 50,000 ppm. There were no adverse effects on reproduction parameters seen even at the dose level of 50,000 ppm (5164 milligram kilogram bodyweight (mg/kg bw) for males and 5,600 mg/kg bw for females). The no-observed adverse effect level (NOAEL) for parental systemic toxicity was 500 ppm (48 mg/kg bw for males and 51 mg/kg bw for females) and the NOAEL for developmental toxicity was 5,000 ppm (270 mg/kg bw for females). Stomach lesions were observed at 5,000 ppm. Two mid-dose males and two males and one female of the high-dose from the Fo died. Body weight and food consumption changes and slight transient reduction in offspring growth were noted at 50,000 ppm. No impairment of reproductive function was observed at any of the dose levels tested.

Prohexadione calcium had no teratogenic potential at dose levels as high as 1,000 mg/kg bw in the rat and 350 mg/kg bw in the rabbit. The NOAEL for maternal toxicity in the teratogenicity studies is 100 mg/kg bw (rabbit) and 1000 mg/kg bw (rat), and the NOAEL for fetotoxicity in the teratogenicity studies is 350 mg/kg bw (rabbit) and 1,000 mg/kg bw (rat). The reproductive and developmental studies are summarized below.

A developmental study was conducted via oral gavage in rats at dose levels of 0, 100, 300, and 1,000 highest dose tested (HDT) mg/kg bw. The NOAEL for developmental and maternal toxicity was 1,000 mg/kg bw, the HDT. This was based on the fact that there were no signs of maternal toxicity, fetotoxicity or teratogenic effects.

A developmental study was conducted via oral gavage in rabbits at dose levels of 0, 40, 200, and 750 HDT mg/kg bw. The NOAEL for development toxicity was 40 mg/kg bw and the NOAEL for maternal toxicity was 40 mg/kg bw based on the following findings. Toxicity in the form of maternal mortality with values 16/20 and 4/20 was excessive in the mid- and high-dose group, respectively. Fetal deaths also occurred. Dose levels believed to exceed maximum tolerance dose (MTD); NOAELs for maternal and developmental effects are not considered reliable and useful for risk characterization. No teratogenic effects were noted in this study.

An additional teratology study in the same strain of rabbits was conducted at dose levels of 0, 30, 75, and 150 mg/kg bw. The NOAEL for development toxicity was 150 mg/kg bw and the NOAEL for maternal toxicity was 30 mg/kg bw based on the following findings. One low-, two mid-, and three high-dose animals died prior to day 29, however, at the high dose group one died of gavage error and another pneumonia, and the reason for the other deaths could not be determined. No teratogenic or fetoxtoxic effects were noted in this study.

An oral range-finding gavage teratology study in the same strain of rabbits (5 animals/dose level) was conducted in another independent laboratory. The dose levels selected were 0, 20, 100, 250, 500, and 1,000 mg/kg bw. This range finding study was conducted with a limited number of animals and a limited scope of examination. Based on these results the dose levels selected for the main study at this independent laboratory were 0, 30, 100, and 350 mg/kg bw. The NOAEL for development toxicity was 350 mg/kg bw and the NOAEL for maternal toxicity was 100 mg/kg bw based on the following findings. At the 350 mg/kg bw dose group transient bw decreases and two abortions were observed. No teratogenic or fetotoxic effects were noted in this study.

Conclusions from teratology studies. More than one definitive rabbit teratology study was conducted because issues associated with exceeding the MTD in the first study and spurious deaths, apparently not compound-related, in the second study confounded the determination of a NOAEL for maternal toxicity. There were no signs of teratogenic or fetotoxic effects in any study other than the first definitive study in which maternal deaths above the MTD apparently occurred. It is BASF's and K-1 Chemicals' opinion based on a thorough review of the teratology studies that the following overall NOAELs can be derived for the

teratology studies: - NOAEL maternal toxicity: 100 mg/kg bw (rabbit) and, 1,000 mg/kg bw (rat). - NOAEL prenatal toxicity: 350 mg/kg bw (rabbit) and, 1,000 mg/kg bw (rat).

The overall NOAEL of 100 mg/kg bw for maternal toxicity in rabbits is based on the last rabbit study, and is based on reduction of bw gain and food intake at dose levels of 250 mg/kg bw onwards. The NOAEL of 350 mg/kg bw for fetotoxic effects in the rabbit is also based on a reduction in bw gain. Based on the overall study results, it is concluded that there are no developmental effects of concern.

Based on preliminary discussions with EPA concerning the rabbit teratology studies, EPA concluded that the definitive NOAEL for maternal toxicity considering all of the studies ranges from 30 to 100 mg/kg bw. Agency scientists further stated that they needed to review the studies in detail to ultimately determine the definitive NOAEL for maternal toxicity. This uncertainty associated with maternal toxicity in the rabbit teratology studies does not impact risk considerations since the risk assessment is based on a lower NOAEL (20 mg/kg bw) in the chronic dog study.

4. *Subchronic toxicity*. The subchronic toxicity of prohexadione calcium was investigated in 90-day feeding studies with rats, mice, and dogs. In all these studies, prohexadione calcium displayed low toxicity. Prohexadione calcium showed no signs of neurotoxicity in a 90-day neurotoxicity rat study. Additionally, the results seen in four week feeding range-finding studies for rats and dogs were similar to the findings observed in the 90-day studies in the same animals.

5. *Chronic toxicity*. Based on review of the available data, the reference dose (RfD) for prohexadione calcium will be based on a 1-year feeding study in dogs with a threshold NOAEL of 20 mg/kg/day. Using an uncertainty factor of 100, the RfD is calculated to be 0.2 mg/kg/day. The following are summaries of studies submitted to EPA. Prohexadione calcium was administered to Beagle dogs at dietary concentrations of 0, 20, 200, and 1,000 mg/kg bw for 12 months. Slight changes were observed for hematological and clinical chemical parameters and dilated basophilic renal tubules (without histopathological concurrence) at dose levels greater than 200 mg/kg bw. The NOAEL was 20 mg/kg bw for the males and female dogs.

The 24-month Fisher 344 rat chronic/carcinogenic feeding study was conducted at dose levels of 0, 400, 2,000, 10,000, and 20,000 ppm with 80 male and 80 female animals per dose group. After 26, 52, and 78 weeks, 10 animals were sacrificed (satellite groups). The remaining animals were autopsied after 104 weeks of diet administration. The NOAEL for chronic toxicity was 2,000 ppm for males (93.9 mg/kg bw) and, 2,000 ppm for females (114 mg/kg bw). The following effects were observed in the 10,000, and 20,000 ppm groups: (1) Decreased bws were observed in both male and female rats at the 20,000 ppm dose level; (2) clinical chemical effects (i.e., lower potassium, bilirubin, and glucose levels) were observed in male and female rats at the 20,000 ppm dose level, in the 10,000 ppm dose level, reduced glucose levels were only seen in the males, and increased albumin/globulin ratios, sodium, chloride and calcium levels were observed only in the females; (3) increased urine volumes and lower specific gravity were observed in the mid-high and high-dose groups for both male and female rats; (4) minor changes in organ weights were noted for animals of the high dose group only, which consisted of increased relative liver, adrenal and kidney weights, the latter also absolute in females only, at week 26; at the end of the study decreased liver weights and increased relative brain and testis weights were noted and these changes were considered to be associated with the decreased bws; (5) macroscopic findings revealed an increase of pituitary nodules in the high dose group for both male and female rats which was not confirmed histopathologically and submucosal ectopic tissue in the glandular stomach was found in both male and female rats in the highest dose levels that was confirmed by histopathology which showed an increase of squamous cell hyperplasia in males and of basal cell hyperplasia in the forestomach; (6) a higher incidence of cellular hyperplasia was observed in the thyroid in the mid-high and high dose levels for male and female rats; and (7) no increased incidence of neoplasms occurred at any dose levels tested in this study.

In the 24-month B6C3F1 mouse feeding study, conducted at dose levels of 0, 400, 2,000, 20,000, and 40,000 ppm with interim sacrifices at 52 and 78 weeks, prohexadione calcium was negative for oncogenicity. The NOAEL for chronic toxicity was 2,000 ppm for males (279 mg/kg bw) and 2,000 ppm for females (351 mg/kg bw). The following effects were observed in the 20,000 and 40,000 ppm groups: (1) Statistically significant decreases in body weights were observed in male mice at the 20,000 ppm dose level and in female mice at the 40,000 ppm dose level; (2) a variety of changes in hematological parameters were noted in the respective investigations at weeks 52, 78, and 104, however, most of the changes were not dose related or consistent over time; (3) increased absolute and/or relative heart, brain, testes, liver, ovary, and kidney weights were observed in the mid-high and highest dose groups with a slight progression of severity to the highest dose group; (4) a higher incidence of splenomegaly was observed only in the male mice of the highest dose group; (5) histopathological examinations revealed an ectopic proliferation of the mucosal and glandular epithelium in the submucosal layer of the glandular stomach in male and female mice in the highest dose group tested, these changes were assessed to represent heteroplastic, ectopic proliferative changes accompanied by lumen dilatation and cytological degeneration; (6) a higher incidence of hyperkeratosis of the forestomach was observed in both male and female mice and hyperplasia of the squamous epithelium of the forestomach of female male mice was observed in the highest dose group tested; (7) vacuolic changes in the exocrine pancreas of the high dose female were observed; and (8) no increased incidence of neoplasms occurred at any dose levels tested in this study.

6. *Endocrine disruption*. No specific tests have been conducted with prohexadione calcium to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there were no significant findings in other relevant toxicity studies (i.e., subchronic and chronic toxicity, teratology and multi-generation reproductive studies) which would suggest that prohexadione calcium produces endocrine related effects.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. For purposes of assessing the potential dietary exposure, K-I Chemical has estimated aggregate exposure based on the Theoretical Maximum Residue Contribution (TMRC) from the proposed tolerances for prohexadione calcium in/on peanut nutmeat at 1.0 ppm and apples (pome fruit) at 3.0 ppm. A maximum residue level of 1.0 ppm was used for pears. The TMRC is a ''worse case'' estimate of dietary exposure since it is assumed that 100% of all crops for which tolerances are established are treated and that pesticide residues are always found at the tolerance levels. The TMRC from the proposed use of

prohexadione calcium on peanuts, pears and apples is 0.002570 mg/kg bw/day and utilizes 1.3% of the RfD for the overall U.S. population. The exposure of the most highly exposed subgroup in the population, non-nursing infants (< 1 year old), is 0.025758 mg/kg bw/day and utilizes 12.9% of the RfD.

Dietary exposure to residues of prohexadione calcium in or on food will be limited to residues on peanuts, apples and pears. Apple pomace, peanut meal and hay are fed to animals; thus exposure of humans to residues in feed items might result if such residues carry through to meat, milk, poultry, or eggs. However, K-I Chemical has concluded that there is no reasonable expectation that measurable residues of prohexadione calcium will occur in meat, milk, poultry, or eggs from this use but residues can be expected to be slightly above the limit of quantitation for cow kidney. Therefore, K-I Chemical is proposing a tolerance in/on cattle meat byproduct (kidney) at 0.1 ppm. There are no currently registered uses for prohexadione calcium on food or feed crops in the U.S. and thus, there are no established U.S. tolerances.

The following table summarizes the mean dietary exposures and the percents of RfD occupied by these exposures.

SUMMARY OF CHRONIC DIETARY EXPOSURE TO PROHEXADIONE CALCIUM

Group	DRES (Dietary Risk Evaluation System)	
	mg/kg bw/day	% RfD
U.S. Population	2.6	1.3
Nursing Infants (< 1 Year Old)	19.3	9.7
Non-Nursing Infants (< 1 Year Old)	25.8	12.9
Children 1-6 Years Old	8.7	4.4
Children 7-12 Years Old	3.5	1.8

ii. *Drinking water.* Based on studies submitted to EPA for assessment of environmental risk, K-I Chemical does not anticipate exposure to residues of prohexadione calcium in drinking water. There is no established Maximum Concentration Level (MCL) or Health Advisory Level (HAL) for prohexadione calcium under the Safe Drinking Water Act (SDWA).

2. *Non-dietary exposure.* K-I Chemical has not estimated non-occupational exposure to prohexadione calcium since the only pending registration is limited to commercial crop production. Prohexadione calcium products are not labeled for any residential uses, therefore eliminating the potential for residential exposure. Thus, potential for non-occupational exposure of the general population to prohexadione calcium is not present.

D. Cumulative Effects

K-I Chemical is aware of only one other registered compound, trinexapac-ethyl [4-(cyclopropyl-a-hydroxymethylene)-3,5-dioxo-cyclohexanecarboxylic acid ethylester], that has a structure similar to prohexadione calcium. However, K-I Chemical has no information that would indicate that the two compounds have a common mechanism of toxicity. Furthermore, trinexapac is registered for use only on turf. Therefore, even if the compounds were considered similar there would be no cumulative dietary exposure issue because of the differences in use patterns. In summary, dietary exposure to prohexadione calcium should not result in cumulative toxicity with other known chemical compounds.

E. Safety Determination

1. *U.S. population.* Using the conservative exposure assumptions described above and based on the completeness and the reliability of the toxicity data, K-I Chemical has estimated that aggregate exposure to prohexadione calcium will utilize 1.3% of the RfD for the U.S. population. K-I Chemical concludes that there is a reasonable certainty that no harm will result from the aggregate exposure to residues of prohexadione calcium, including anticipated dietary exposure and non-occupational exposures.

2. *Infants and children*—i. *Developmental toxicity in the rat.* A developmental study was conducted via oral gavage in rats with dosages of 0, 100, 300, and 1,000 HDT mg/kg/day with a NOAEL of 1,000 mg/kg/day the HDT for developmental and maternal toxicity based on the fact that no effects were observed for any test parameter measured in this study. Therefore, these NOAEL values are significantly higher than the NOAEL from the 1-year feeding study in dogs used to establish the RfD.

ii. *Developmental toxicity in the rabbit.* A series of developmental studies were conducted via oral gavage in rabbits with dosages ranging from 0 to 750 mg/kg/day with a development toxicity NOAEL of 350 mg/kg/day and a maternal toxicity NOAEL of 100 mg/kg/day based on bw gain reductions. These NOAEL values are higher than the NOAEL from the 1-year feeding study in dogs used to establish the RfD.

iii. *Reproductive toxicity.* A 2-generation reproduction study with rats fed dosages of 0, 500, 5,000, and 50,000 mg/kg/day resulted in a reproductive NOAEL of 50,000 ppm (5,300 mg/kg bw/day), a developmental NOAEL of 5,000 ppm (270 mg/kg bw/day), and a maternal toxicity NOAEL of 500 ppm (50 mg/kg bw/day). The developmental NOAEL was based on a slight, transient reduction in offspring growth. The maternal NOAEL is similar and the reproductive NOAEL is significantly higher (above the limit dose of 1,000 mg/kg/day) than the NOAEL from the 1-year feeding study in dogs used to establish the RfD.

3. *Reference dose.* Since developmental and reproductive toxicity occurs at levels above the levels shown to exhibit parental toxicity and since these levels are significantly higher than those used to calculate the RfD, K-I Chemical believes the RfD of 0.20 mg/kg/day (20 mg/kg/day and an Uncertainty Factor of 100) is an appropriate measure of safety for infants and children.

Dietary exposure of the most highly exposed subgroup in the population, non-nursing infants (< 1 year old) is 0.025758 mg/kg bw/day. This accounts for 12.9% of the RfD. There are no residential uses of prohexadione calcium and contamination of drinking water is extremely unlikely. In addition, there were no significant findings in relevant toxicity studies (i.e., subchronic and chronic toxicity, teratology and multi-generation reproductive studies) which would suggest that prohexadione calcium produces endocrine related effects. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, K-I Chemical concludes that there is a

reasonable certainty that no harm will result to infants and children from aggregate exposure to the residues of prohexadione calcium, including all anticipated dietary exposure and all other non-occupational exposures.

F. International Tolerances

A maximum residue level (MRL) has not been established for prohexadione calcium in peanuts, apples or pears by the Codex Alimentarius Commission.

[FR Doc. 99–21944 Filed 8–23–99; 8:45 am]

BILLING CODE 6560–50–F

FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collection(s) Being Reviewed by the Federal Communications Commission, Comments Requested

August 18, 1999.

SUMMARY: The Federal Communications Commission, 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, Pub. L. 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 clarity 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: Written comments should be submitted on or before October 25, 1999. If you anticipate that you will be submitting comments, but find it difficult to do so within the period of time allowed by this notice, you should advise the contact listed below as soon as possible.

ADDRESSES: Direct all comments to Les Smith, Federal Communications Commission, 445 12th Street, SW, Room 1–A804, Washington, DC 20554 or via the Internet to lesmith@fcc.gov.

FOR FURTHER INFORMATION CONTACT: For additional information or copies of the information collections contact Les Smith at (202) 418–0217 or via the Internet at lesmith@fcc.gov.

SUPPLEMENTARY INFORMATION:

OMB Approval Number: 3060–0798.

Title: FCC Application for Wireless Telecommunications Bureau Radio Service Authorization.

Form Number: FCC 601.

Type of Review: Revision of an existing collection.

Respondents: Individuals or households; Business or other for-profit; Not-for-profit institutions; State, Local or Tribal Government.

Number of Respondents: 240,320.

Estimated Time Per Response: 1.25 hours.

Total Annual Burden: 210,280 hours.

Needs and Uses: FCC 601 is used as the general application (long form) for market based licensing and site-by-site licensing in the Wireless Telecommunications Radio Services. The purpose of this revision is to make the necessary changes to convert the Private Operational and Fixed Microwave Services to ULS. We sought emergency clearance and received approval on these changes and are now seeking a 3 year clearance. The information is used by the Commission to determine whether the applicant is legally, technically and financially qualified to be licensed.

Respondent costs are estimated to be $48,364,000, which includes application filing fees.

Federal Communications Commission.

Magalie Roman Salas,

Secretary.

[FR Doc. 99–21900 Filed 8–23–99; 8:45 am]

BILLING CODE 6712–01–P

FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collection(s) Being Reviewed by the Federal Communications Commission

August 17, 1999.

SUMMARY: The Federal Communications Commission, 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 clarity 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: Written comments should be submitted on or before October 25, 1999. If you anticipate that you will be submitting comments, but find it difficult to do so within the period of time allowed by this notice, you should advise the contact listed below as soon as possible.

ADDRESSES: Direct all comments to Les Smith, Federal Communications Commissions, 445 12th Street, SW, Room 1–A804, Washington, DC 20554 or via the Internet to lesmith@fcc.gov.

FOR FURTHER INFORMATION CONTACT: For additional information or copies of the information collections contact Les Smith at (202) 418–0217 or via the Internet at lesmith@fcc.gov.

SUPPLEMENTARY INFORMATION:

OMB Control Number: 3060–0855

Title: Telecommunications Reporting Worksheet and Associated Requirements—CC Docket No. 98–171

Form Number: FCC Forms 499–A and FCC 499–S

Type of Review: Extension.

Respondents: Business or other for-profit entities.

Number of Respondents: 5500 respondents

Estimated Time Per Response: 7.27 hours per response (avg.)

Total Annual Burden: 40,000 hours.

Estimated Annual Reporting and Recordkeeping Cost Burden: $9,200.00.

Frequency of Response: Annually, Semi-Annually, On occasion, Third party disclosure.

Needs and Uses: In a Report and Order issued in CC Docket No. 98–171, released July 14, 1999, the Commission simplified and consolidated four Commission reporting requirements so that carriers need only file one worksheet to satisfy the contributor reporting requirements associated with: the universal service support