requested EPA delay in taking action for one year in order to determine the geographical extent and severity of the CO problem. EPA agreed to this request.

According to the Montana DEQ, data from the Idaho and Main CO monitoring site in conjunction with the results from the CO saturation study suggested the CO problem was a traffic corridor problem extending six- to eight-blocks in either direction along Idaho and Main. Montana DEQ determined that the CO saturation study did not sufficiently look at the effects of CO in the surrounding neighborhoods. Therefore, an additional CO monitoring site was installed next to Laser School, a residential site located one block north of Highway 2 and approximately five blocks north and east of the Idaho and Main site. Data collection at Laser School began on November 1, 1996.

II. Finding of Inadequacy

On January 10, 1980 (45 FR 2036), EPA approved Montana's plans for the attainment and maintenance of the national standards under section 110 of the Clean Air Act. EPA now finds [1] the SIP inadequate based on the reported exceedances of the CO NAAQS in Kalispell.

III. Call for SIP Revision

This finding of SIP inadequacy requires Montana to submit a SIP revision no later than 18 months from the date of EPA's letter to the Governor. To ensure that the SIP deadline is met, EPA requested the State to submit an action plan for the development of the SIP revision within 60 days from receipt of EPA's letter to the Governor. The State submitted an action plan to EPA on September 9, 1997. Any control strategies adopted and implemented as part of this SIP revision must provide for attainment and maintenance of the CO NAAQS within 5 years from the date of EPA's letter to the Governor. (See, e.g., section 110(n)(2) of the Act.)

IV. Final Action

This finding of inadequacy does not constitute a final agency action that is ripe for judicial review. EPA's action is a first step in an administrative process that will not be sufficiently concrete for judicial resolution until additional action is taken by EPA on a plan submittal by the State of Montana.

The 60-day time period for filing a petition for review under section 307(b) of the CAA is tolled until EPA makes the finding ripe by taking additional action in reliance on it, such as imposing sanctions on the State of Montana for failure to submit a SIP revision or promulgating approval of a SIP revision. A time limitation on petitions for judicial review can only run against challenges ripe for review.

A technical support document (TSD) is available from the contact person listed above. The TSD discusses in more detail the ambient standard and its health effects, the SIP call and legal authority, and the SIP revision schedule.

Authority: Sections 101, 107, 110, 116 and 301(a) of the Clean Air Act, as amended [42 U.S.C. 7401, 7407, 7410, 7416 and 7610(a)].

Dated: November 17, 1997.

William P. Yellowtail,

Regional Administrator.

[FR Doc. 97–32931 Filed 12–16–97; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[PF–781; FRL–5758–3]

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–781, must be received on or before January 16, 1998.

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. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 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
James Tompkins (PM 25).	Rm. 265, CM #2, 703–305–7801, e-mail:tompkins.james@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Elizabeth Haeberer	Rm. 207, CM #2, 703–308–2891, e-mail: haeberer.elizabeth@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–781]

[1] The finding is made pursuant to sections 110(a)(2)(H) and 110(k)(5) of the Clean Air Act, 42 U.S.C. 7410(a)(2)(H) and 7410(k)(5).

(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 PF-781 and appropriate petition number. Electronic comments on notice may be filed online at many Federal Depository Libraries.

List of Subjects

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

Dated: December 4, 1997

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

1. Bayer Corporation

PP 5F4480

EPA has received a pesticide petition (PP 5F4480) from Bayer Corporation, 8400 Hawthorn Rd., P.O. Box 4913, Kansas City, MO 64120-0013. proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of imidacloprid in or on the raw agricultural commodity pecans at 0.05 parts per million (ppm). The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using UV detection. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of imidacloprid in plants is adequately understood for the purposes of these tolerances. The residues of concern are combined residues of imidacloprid and its metabolites containing the 6-chloro-pyridinyl moiety, all calculated as imidacloprid.

2. *Analytical method*. The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloropyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary GC-MS selective ion monitoring. This method has successfully passed a petition method validation in EPA labs. There is a confirmatory method specifically for imidacloprid and several metabolites utilizing GC/MS and HPLC-UV which has been validated by the EPA as well. Imidacloprid and its metabolites are stable for at least 24 months in the commodities when frozen.

3. *Magnitude of residues*. Field studies were conducted to determine imidacloprid residues on pecans following treatment with either a single soil or two foliar applications. Seven field studies were conducted using a single soil application of 0.5 lb active ingredient per acre. 5–field studies were conducted using two foliar applications at a rate of 0.17 lb active ingredient per acre, with a 10–day interval. After the final foliar application or the soil application, samples were collected at earliest harvest which ranged from 4 to 21–days for the foliar application or 99 to 150 days for the soil application. Maximum residues, in pecans, detected following either 2 foliar applications or 1 soil application were >0.05 ppm. Therefore, a tolerance of 0.05 ppm of pecans is being proposed with a preharvest interval defined as earliest harvest (shuck split). CBTS has concluded that existing poultry meat and egg tolerances are adequate to support the proposed new uses of imidacloprid.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral LD_{50} values for imidacloprid technical ranged from 424 - 475 milligrams/kilogram/bodyweight (mg/kg/bwt) in the rat. The acute dermal LD_{50} was greater than 5,000 mg/kg in rats. The 4–hour rat inhalation LC_{50} was >69 mg/m^3 air (aerosol). Imidacloprid was not irritating to rabbit skin or eyes. Imidacloprid did not cause skin sensitization in guinea pigs.

2. *Genotoxicty*. Extensive mutagenicity studies conducted to investigate point and gene mutations, DNA damage and chromosomal aberration, both using *in vitro* and *in vivo* test systems show imidacloprid to be non-genotoxic.

3. *Reproductive and developmental toxicity*. A 2–generation rat reproduction study gave a no-observed-effect level (NOEL) of 100 ppm (8 mg/kg/bwt). Rat and rabbit developmental toxicity studies were negative at doses up to 30 mg/kg/bwt and 24 mg/kg/bwt, respectively.

4. *Subchronic toxicity*. 90–day feeding studies were conducted in rats and dogs. The NOEL's for these tests were 14 milligrams/kilogram/bodyweight/day (mg/kg/bwt/day) (150 pm) 5 mg/kg/bwt/day (200 ppm) for the rat and dog studies respectively.

5. *Chronic toxicity*. A 2–year rat feeding/carcinogenicity study was negative for carcinogenic effects under the conditions of the study and had a NOEL of 100 ppm (5.7 mg/kg/bwt in male and 7.6 mg/kg/bwt female) for noncarcinogenic effects that included decreased body weight gain in females at 300 ppm and increased thyroid lesions in males at 300 ppm and females at 900 ppm. A 1–year dog feeding study indicated a NOEL of 1,250 ppm (41 mg/kg/bwt). A 2–year mouse carcinogenicity study that was negative for carcinogenic effects under conditions of the study and that had a NOEL of 1,000 ppm 208 milligrams/kilogram/day (mg/kg/day).

Imidacloprid has been classified under ''Group E'' (no evidence of carcinogenicity) by EPA's OPP/HED's Reference Dose (RfD) Committee. There is no cancer risk associated with exposure to this chemical. The reference dose (RfD) based on the 2–year rat feeding/carcinogenic study with a NOEL of 5.7 mg/kg/bwt and 100-fold uncertainty factor, is calculated to be 0.057 mg/kg/bwt. The theoretical maximum residue contribution (TMRC) from published uses is 0.008187 mg/kg/bwt/day utilizing 14.4% of the RfD.

6. *Animal metabolism*. The metabolism of imidacloprid in animals

is adequately understood. The residues of concern are combined residues of imidacloprid and its metabolites containing the 6-chloro-pyridinylmoiety, all calculated as imidacloprid.

C. Aggregate Exposure

Imidacloprid is a broad-spectrum insecticide with excellent systemic and contact toxicity characteristics with both food and non-food uses. Imidacloprid is currently registered for use on various food crops, tobacco, turf, ornamentals, buildings for termite control, and cats and dogs for flea control.

1. *Dietary exposure*. The EPA has determined that the reference dose (RfD) based on the 2–year rat feeding/ carcinogenic study with a NOEL of 5.7 mg/kg/bwt and 100-fold uncertainty factor, is calculated to be 0.057 mg/kg/ bwt.

2. *Food*. The theoretical maximum residue contribution (TMRC) from this proposed use on Pecans as well as all published uses and pending uses is 0.008149 mg/kg/bwt/day utilizing 14.3% of the RfD for the general population. For the most highly exposed subgroup in the population, children (1–6 years), the TMRC for the all uses is 0.018367 mg/kg/day. This is equal to 32.2% of the RfD. Therefore, dietary exposure from the existing uses including the currently proposed tolerance will not exceed the reference dose for any subpopulation (including infants and children).

3. *Drinking water*. Although the various imidacloprid labels contain a statement that this chemical demonstrates the properties associated with chemicals detected in ground water, the Registrant is not aware of imidacloprid being detected in any wells, ponds, lakes, streams, etc. from its use in the U.S. In studies conducted in 1995, imidacloprid was not detected in 17 wells on potato farms in Quebec, Canada. In addition, ground water monitoring studies are currently underway in California and Michigan. Therefore, contributions to the dietary burden from residues of imidacloprid in water would be inconsequential.

4. *Non-dietary exposure*— i. *Residential turf*. Bayer has conducted an exposure study to address the potential exposures of adults and children from contact with imidacloprid treated turf. The population considered to have the greatest potential exposure from contact with pesticide treated turf soon after pesticides are applied is young children. Margins of safety (MOS) of 7,587 - 41,546 for 10–year–old children and 6,859 - 45,249 for 5–year–old children were estimated by comparing dermal exposure doses to the imidacloprid no-observable effect level of 1,000 mg/kg/ day established in a 15–day dermal toxicity study in rabbits. The estimated safe residue levels of imidacloprid on treated turf for 10–year–old children ranged from 5.6 - 38.2 g/cm^2 and for 5–year–old children from 5.1 - 33.5 g/cm^2. This compares with the average imidacloprid transferable residue level of 0.080 g/cm^2 present immediately after the sprays have dried. These data indicate that children can safely contact imidacloprid-treated turf as soon after application as the spray has dried.

ii. *Termiticide*. Imidacloprid is registered as a termiticide. Due to the nature of the treatment for termites, exposure would be limited to that from inhalation and was evaluated by EPA's Occupational and Residential Exposure Branch (OREB) and Bayer. Data indicate that the Margins of Safety for the worst case exposures for adults and infants occupying a treated building who are exposed continuously (24 hours/day) are 8.0×10^7 and 2.4×10^8, respectively - and exposure can thus be considered negligible.

iii. *Tobacco smoke*. Studies have been conducted to determine residues in tobacco and the resulting smoke following treatment. Residues of imidacloprid in cured tobacco following treatment were a maximum of 31 ppm (7 ppm in fresh leaves). When this tobacco was burned in a pyrolysis study only 2% of the initial residue was recovered in the resulting smoke (main stream plus side stream). This would result in an inhalation exposure to imidacloprid from smoking of approximately 0.0005 mg per cigarette. Using the measured subacute rat inhalation NOEL of 5.5 mg/m^3, it is apparent that exposure to imidacloprid from smoking (direct and/or indirect exposure) would not be significant.

iv. *Pet treatment*. Human exposure from the use of imidacloprid to treat dogs and cats for fleas has been addressed by EPA's OREB who have concluded that due to the fact that imidacloprid is not an inhalation or dermal toxicant and that while dermal absorption data are not available, imidacloprid is not considered to present a hazard via the dermal route.

D. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, it can be concluded that total aggregate exposure to imidacloprid from all current uses including those currently proposed will utilize little more than 15% of the RfD for the U.S. population. EPA generally has no concerns for exposures below 100% of the RfD, because the RfD represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of imidacloprid, the data from developmental studies in both rat and rabbit and a 2–generation reproduction study in the rat have been considered. The developmental toxicity studies evaluate potential adverse effects on the developing animal resulting from pesticide exposure of the mother during prenatal development . The reproduction study evaluates effects from exposure to the pesticide on the reproductive capability of mating animals through two generations, as well as any observed systemic toxicity.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post- natal effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for imidacloprid relative to pre- and post-natal effects is complete. Further for imidacloprid, the NOEL of 5.7 mg/kg/bwt from the 2–year rat feeding/ carcinogenic study, which was used to calculate the RfD (discussed above), is already lower than the NOELs from the developmental studies in rats and rabbits by a factor of 4.2 to 17.5 times. Since a 100-fold uncertainty factor is already used to calculate the RfD, it is surmised that an additional uncertainty factor is not warranted and that the RfD at 0.057 mg/kg/bwt/day is appropriate for assessing aggregate risk to infants and children.

Using the conservative exposure assumptions described above, it can be concluded that the TMRC from use of imidacloprid from published and pending uses is 0.008149 mg/kg/bwt/ day utilizing 14.3% of the RfD for the general population. For the most highly exposed subgroup in the population, children (1–6 years), the TMRC for the published tolerances is 0.018367 mg/kg/ day. This is equal to 32.2% of the RfD. Therefore, dietary exposure from the existing uses including the currently proposed tolerances will not exceed the reference dose for any subpopulation (including infants and children).

E. International Tolerances

No CODEX Maximum Residue Levels (MRL's) have been established for residues of Imidacloprid on any crops at this time. (Elizabeth Haeberer)

2. E. I. Du Pont de Nemours & Company

PP 5F4545

EPA has received a pesticide petition (PP 5F4545) from E. I. Du Pont de Nemours & Company (DuPont), P.O. Box 80038, Wilmington, DE 19880-0038. proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish an exemption from the requirement of a tolerance for quizalofop (2-[4-(6-chloroquinoxalin-2-yl)oxy) phenoxy]) - propanoic acid], and quizalofop ethyl [ethyl-2- [4-(6-chloroquinoaxalin-2-yl)oxy) phenoxy) propanoat in or on the raw agricultural commodities canola seed and canola meal . The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using UV detection. 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*. Quizalofop-p ethyl ester is metabolized by cleavage at three sites as follows:

(i) Primary pathway is hydrolysis of the ethyl ester to form the quizalofop-p acid, then (ii) Cleavage of the enol ether linkage in the acid, between the phenyl and quinoxalinyl rings, to form phenols, and (iii) Cleavage of the ether linkage between the isopropanic group and the phenyl ring to form a phenol.

The plant metabolism data show that quizalofop-p ethyl ester does not translocate, but is rapidly hydrolyzed to the corresponding acid; then the phenols conjugate with the plant sugars. Metabolism studies in soybeans using the racemic mixture quizalofop ethyl ester and the resolved D+ isomer show nearly identical pathways.

The nature of the quizalofop-p ethyl ester residue in plants is adequately understood. The residues of concern are quizalofop-p ethyl ester and its acid metabolite, quizalofop-p, and the *S* enantiomers of both the ester and the acid, all expressed as quizalofop-p ethyl ester.

2. *Analytical method*. An adequate analytical methodology (high-pressure liquid chromatography using either ultraviolet or fluorescence detection) is available for enforcement purposes in Vol. II of the Food and Drug Administration Pesticide Analytical Method (PAM II, Method I).

Adequately validated residue analytical methods, LAN-1 and LAN-3, were used to gather the magnitude of the quizalofop-p, its acid metabolite, phenols 1, 2, and 4, residue data on canola and canola processed commodities.

3. *Magnitude of residues*. Dupont proposes establishing tolerances for the combined residues of quizalofop (2-[4-(6-chloroquinoaxalin-2-yl)oxy) phenoxy])-propionic acid], and quizalofop ethyl [ethyl-2- [4-(6-chloroquinoxalin-2-yl)oxy) phenoxy) propanoat for the raw agricultural commodities canola seed at 1.0 parts per million (ppm) and canola meal at 1.5 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Several acute toxicology studies were conducted and the overall results placed technical grade quizalofop ethyl in toxicity Category III. These include the following studies in Category III: acute oral toxicity (LD_{50}s 1,480 and 1,670 for female and male rats, respectively) and eye irritation (mild effects; reversible within 4–days). Dermal toxicity (LD_{50} > 5,000 mg/kg; rabbit), inhalation toxicity (LC_{50} > 5.8 mg/L; rat) and dermal irritation were classified within Category IV. Technical quizalofop ethyl was not a dermal sensitizer.

2. *Genotoxicty*. Technical quizalofop ethyl was negative in the following genotoxicity tests: bacterial gene mutation assays with *E. coli* and *S. typhimurium*; gene mutation assays in Chinese hamster ovary(CHO) cells ; *in vitro* DNA damage assays with B. subtillis and in rat hepatocytes; and an *in vitro* chromosomal aberration test in CHO cells.

3. *Reproductive and developmental toxicity*. Studies supporting the registration include: A developmental toxicity study in rats administered dosage levels of 0, 30, 100, and 300 milligrams/kilogram/day (mg/kg/day) highest dose tested (HDT). The maternal toxicity no-observed effect level (NOEL) was 30 mg/kg/day and a developmental toxicity NOEL was greater than 300 mg/kg/day (HDT). The maternal NOEL was based on reduced food consumption and increased liver weights. A developmental toxicity study in rabbits administered dosage levels of 0, 7, 20, and 60 mg/kg/day with no developmental effects noted at 60 mg/kg/day (HDT). The maternal toxicity NOEL was 20 mg/kg/day based on decreases in food consumption and body weight gain at 60/mg/kg/day (HDT). A 2-generation reproduction study in rats fed diets containing 0, 25, 100 or 400 ppm (or approximately 1, 1.25, 5, and 20 mg/kg/day, respectively) with a developmental (systemic effects) NOEL of 1.25 mg/kg/day for F_{2B} weanlings based on increased liver weights and increased incidence of eosinophilic changes in the livers at 5.0 mg/kg/day. These liver changes were considered to be physiological or adaptive changes to compound exposure among weanlings. When access to the mother's feed is available, it is a common observation that young rats will begin consuming chow prior to complete weaning at 21–days of age. Consumption could not be quantified; therefore, the maternal consumption was assumed as the NOEL (if normalized on a body weight basis, exposures to the weanling rats were likely higher). The parental NOEL of 5.0 mg/kg/day was based on decreased body weight and premating weight gain in males at 20 mg/kg/day (HDT).

4. *Subchronic toxicity*. A 90-day study was conducted in rats fed diets containing 0, 40, 128, 1,280 ppm (or approximately 0, 2, 6.4 and 64 mg/kg/day, respectively). The NOEL was 2 mg/kg/day. This was based on increased liver weights at 6.4 mg/kg. A 90–day feeding study in mice was conducted with diets that contained 0, 100, 316 or 1,000 ppm (or approximately 0, 15, 47.4, and 150 mg/kg/day, respectively). The NOEL was > 15 mg/kg/day lowest dose tested (LDT) based on increased liver weights and reversible histopathological effects in the liver at the LDT. A 6-month feeding study in dogs was conducted with diets that contained 0, 25, 100 or 400 ppm (or approximately 0, 0.625, 2.5, and 10 mg/kg/day, respectively). The NOEL was 2.5 mg/kg/day based on increased blood urea nitrogen at 10 mg/kg/day. A 21–day dermal study was conducted in rabbits at doses of 0, 125, 500 or 2,000 mg/kg/day. The NOEL was 2,000 mg/kg/day (HDT).

5. *Chronic toxicity*. An 18–month carcinogenicity study was conducted in CD-1 mice fed diets containing 0, 2, 10, 80 or 320 ppm (or approximately 0, 0.3, 1.5, 12, and 48 mg/kg/day, respectively). There were no carcinogenic effects observed under the conditions of the study at levels up to and including 12 mg/kg/day. A marginal increase in the incidence of hepatocellular tumors was observed at 48 mg/kg/day, the (HDT) which exceeded the maximum tolerated

dose (MTD). (Please see the discussion by the EPA HED Carcinogenicity Peer Review Committee.)

A 2–year chronic toxicity/carcinogenicity study was conducted in rats fed diets containing 0, 25, 100 or 400 ppm (or 0, 0.9, 3.7, and 15.5 mg/kg/day for males and 0, 1.1, 4.6, and 18.6 mg/kg/day for females, respectively). There were no carcinogenic effects observed under the conditions of the study at levels up to and including 18.6 g/kg/day (HDT). The systemic NOEL was 0.9 mg/kg/day based on altered red cell parameters and slight/minimal centrilobuler enlargement of the liver at 3.7 mg/kg/day.

A 1–year feeding study was conducted in dogs fed diets containing 0, 25, 100 or 400 ppm (or approximately 0, 0.625, 2.5, and 10 mg/kg/day, respectively). The NOEL was 10 mg/kg/day (HDT).

The Carcinogenicity Peer Review Committee (CPRC) of HED has evaluated the rat and mouse cancer studies on quizalofop along with other relevant short-term toxicity studies, mutagenicity studies, and structure activity relationships. The CPRC concluded, after three meetings and an evaluation by the OPP Science Advisory panel, that the classification should be a Category D (not classifiable as to human cancer potential). No new cancer studies were required.

The first CPRC review tentatively concluded that quizalofop should be classified as a Category B2 (probable human carcinogen). That classification was based on liver tumors in female rats, ovarian tumors in female mice, and liver tumors in male mice. This classification was downgraded to a Category C (possible human carcinogen) at a second CPRC review. The change in classification was due to a reexamination of the liver tumors in female rats and ovarian tumors in female mice. The first peer review had found a statistically significant positive trend for liver carcinomas in female rats. Subsequent to this conclusion the tumor data was reevaluated, and the revaluation showed a reduced number of carcinomas. Although there remained a statistically significant positive trend for carcinomas in the study, the CPRC concluded that the carcinomas were not biologically significant given the few carcinomas identified (one at the mid-dose and two at the high dose). Noting that this level of carcinomas was within historical levels, the CPRC concluded that administration of quizalofop did not appear to be associated with the liver carcinomas.

As to the ovarian tumors in female mice, the CPRC had first attached importance to the fact that these tumors were statistically significant at the high dose as compared to historical control values although statistically significant when compared to concurrent controls. However, review of further historical control data showed that the level of ovarian tumors in the quizalofop study was similar to the background rate in several other studies. Given this information and that the quizalofop study showed no hyperplasia of the ovary, no signs of endocrine activity related to ovarian function, and no dose response relationship, the CPRC concluded that the ovarian tumors were probably not compound-related.

The findings of the second CPRC review were presented to EPA's Scientific Advisory Panel (SAP). The SAP concurred with the CPRC conclusion that the liver tumors in female rats and the ovary tumors in female mice showed no evidence of carcinogenicity. However, the SAP disagreed with CPRC's classification of quizalofop as a Category C based on the liver tumors in male mice. The SAP concluded that the mouse liver tumors did not support such a classification because the tumors occurred at a dose above the MTD and because they were not statistically significant if a ''p'' value of less than 0.05. The SAP believed that such greater statistical rigor was appropriate for variable tumor endpoints such as male mouse liver tumors.

Following the SAP review, the CPRC changed the classification for quizalofop to Category D. The Category D classification is based on an approximate doubling in the incidence of male mice liver tumors between controls an the high dose. This finding was not considered strong enough to warrant the finding of a Category C (possible human carcinogen) since the increase was of marginal statistical significance, occurred at a high dose which exceeded the predicted MTD, and occurred in a study in which the concurrent control for liver tumors was somewhat low as compared to the historical controls, while the high dose control group was at the upper end of previous historical control-groups.

EPA has found the evidence on the carcinogenicity of quizalofop-p ethyl ester in animals to be equivocal and therefore concludes that quizalofop-p ethyl ester does not induce cancer in animals within the meaning of the Delaney clause. Important to this conclusion was the following evidence: (1) The only statistically significant tumor response that appears compound-related was seen at a single dose in a single sex in a single species; (2) the response was only marginally statistically significant; (3) the response was only significant when benign and malignant tumors were combined; (4) the tumors were in the male mouse liver; (5) the tumors were within historical controls; and (6) the mutagenicity studies were negative. Although in some circumstances a finding of animal carcinogenicity would be made despite any one, or even several, of the six factors noted, the combination of all of these factors here cast sufficient doubt on the reproducibility of the response in the high dose male mouse that EPA concludes the evidence on carcinogenicity is equivocal.

6. *Animal metabolism*. The metabolism of quizalofop ethyl in animals (rat, goat and poultry) is well understood. ^{14}C-phenyl and ^{14}C-quinoxaline quizalofop ethyl ester metabolism studies have been conducted in each species. There are similarities among these species with respect to metabolism. Quizalofop ethyl is rapidly and extensively metabolized and rapidly excreted by rats. The principal metabolites were the quizalofop-p acid and two dechlorinated hydroxylated forms of the acid. Tissue residues were minimal and there was no evidence of accumulation of quizalofop ethyl or its metabolites in the rat.

The primary pathway in ruminants is hydrolysis of the ethyl ester to form the quizalofop-p methyl ester. In poultry, the primary metabolic pathway is also the hydrolysis of the ethyl ester to form the quizalofop-p acid, then the methyl esterification to form the quizalofop methyl ester becomes a minor pathway.

The nature of the quizalofop ethyl ester residue in livestock is adequately understood. The residues of concern are quizalofop ethyl, quizalofop methyl, and quizalofop, all expressed as quizalofop ethyl.

7. *Metabolite toxicology*. There is no evidence that the metabolites of quizalofop ethyl as identified as either the plant or animal metabolism studies are of any toxicological significance.

C. Aggregate Exposure

1. *Dietary exposure*. Quizalofop ethyl is a herbicide with proposed use on canola. The only potential for non-occupational aggregate exposure would come from dietary intake.

An analysis of chronic dietary risk was conducted to determine the impact of the possible addition of canola to the Assure label. A Reference Dose (RfD) of 0.009 mg/kg/day was used in the analyses. Consumption data for canola

had to be estimated using various production and usage statistics.

2. *Food*. The first step in the analysis was to run the TAS (Tolerance Assessment System) program using current tolerances with an RfD of 0.009 mg/kg/day. The Theoretical Maximum Residue Concentration (TMRC), based on the current tolerances, was 0.000288 mg/kg/day for the U.S. population (48 states) and 0.000759 mg/kg/day for the population subgroup with the highest estimated exposure (non-nursing infants > 1–yr. old). For the U.S. population subgroup this represents approximately 3.2% of the RfD while for the most exposed population this represents approximately 8.4% of the RfD. Based on the risk estimates arrived at in this analysis, chronic dietary risk from the current uses of Assure is minimal.

Unfortunately the 1977–1979 food consumption database does not contain any consumption data for canola oil. At the time the survey was conducted, canola oil was not a significant part of the U.S. diet. Since 1977 more canola oil is used in U.S. homes, although total production and usage are still minor as compared to soybean oil. Conservative assumptions were used to estimate canola consumption in the U.S. The USDA's Oilseed Analysis Division indicated that an average of 1.1 billion pounds of canola oil was used in the U.S. annually over the past 5–years. The dietary exposures that might occur by way of consumption of canola oil can be estimated by taking the average annual consumption of canola oil in the U.S. (includes both domestically produced and imported canola oils) and dividing it by the approximate U.S. population of 266.3 million people. This gives a per-capita consumption estimate for the general population. To calculate exposure, this number is divided by the average number of days in a year and the average body weight of a person (60 kg). (This weight is the same that was used by EPA as part of their ''Food Factor'' system that predated the current Tolerance Assessment System). This value is also supported by taking the average weight of children between the ages of 6–months to 19–years (36 kg) and the average weight of adults (70 kg), and assuming that an average person lives to be 69–years old (Review Draft of the Exposure Factors Handbook, U.S. EPA). Using these assumptions, canola oil consumption was calculated to be 0.088 g/kg bw/day.

While this method provides a useful estimate of exposure, it is clearly a conservative estimate for risk assessment purposes, since this estimate assumes that all the canola oil used in the U.S. is indeed ingested. In reality some percentage of any commodity is lost between production and consumption. In addition, oil may be used in cooking activities such as deep-fat frying where most of the oil is not actually eaten but is discarded or recycled. With the understanding that the dietary analysis will be very conservative, the consumption data for canola used in the DRES analysis for all population subgroups was set at 0.088 g/kg bw/day. This was done by entering a consumption estimate of 0.088 for ''rapeseed'' for all population subgroups (there is no agricultural commodity in TAS for canola oil).

When a tolerance for canola (1.0 ppm) was added to the current tolerances, the TMRC was 0.000376 mg/kg/day for the U.S. population (48 states) and 0.000847 mg/kg/day for the highest population subgroup (non-nursing infants >1–yrs. old). When expressed as a percentage of the RfD, the U.S. population (48 states) was approximately 4.2% and the highest population subgroup was approximately 9.4%. These results indicate that predicted chronic exposure after the addition of a canola tolerance is well below the RfD even with the conservative (high) nature of the assumptions that were made in calculating consumption.

3. *Drinking water*. Another potential source of dietary exposure to pesticides is residues in drinking water. There is no established Maximum Concentration Level (MCL) for quizalofop ethyl in water. Based on the low use rate of quizalofop ethyl, and a use pattern that is not widespread (since the current and proposed uses are on minor crops), DuPont does not anticipate residues of quizalofop in drinking water and exposure from this route is unlikely.

4. *Non-dietary exposure*. Quizalofop ethyl is not registered for any use which could result in non-occupational, non-dietary exposure to the general population.

D. Cumulative Effects

There is no evidence to indicate or suggest that quizalofop p-ethyl has any toxic effects on mammals that would be cumulative with those of any other chemicals.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the most sensitive species chronic NOEL of 0.9 mg/kg and a reference dose (RfD) of 0.009 mg/kg/day, the existing tolerances and proposed use of quizalofop ethyl on canola are expected to utilize 4.2% of the RfD for the general U.S. population. Generally, exposures below 100% of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to quizalofop ethyl resulting from proposed agricultural use on canola.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of quizalofop ethyl, data were considered from developmental toxicity studies in the rat and rabbit, and a multi-generation reproduction study in rats. There were no developmental effects observed in the absence of maternal toxicity in the rat and rabbit developmental studies. Minimal adaptive or physiological effects were observed in livers of weanlings in the 2–generation rat reproduction study described earlier. However, this effect was only observed at a dose that far exceeds any expected human exposure. Further, the NOEL of 0.9 mg/kg/day from the 2–year rat study with quizalofop ethyl which was used to calculate the RfD (discussed above), is already lower than any of the NOEL's defined in the developmental and reproductive toxicity studies with quizalofop ethyl.

As mentioned previously, canola oil is a very minor component of the diet, and thus had not been included as part of the 1977–79 food survey used in EPA's DRES system. DuPont is not aware of specific food survey data concerning consumption of canola oil by infants and children. However, the 1977–79 food survey database does provide consumption data for other edible oils for each of the population subgroups, including infants and children. This data indicates that non-nursing infants consume more soybean and coconut oil than any of the other 22 population subgroups, specifically consuming 4.2 times more soybean oil and 49.1 times more coconut oil than the consumption by the general U.S. population. The data also show that children 1–6 consume more corn, cottonseed, peanut, and sunflower oil than any other subgroup listed, to a maximum of 2 times more than the general U.S. population. Using this data and making the most conservative assumption to extrapolate to canola oil, we can estimate that infants and children consume 49 times more canola oil than does the U.S. population, and calculate an approximate daily consumption of 4.3 grams canola oil/kg body weight. If we use the additional conservative assumptions that all the canola oil consumed contains

quizalofop ethyl residues at tolerance levels of 1.0 ppm, we calculate that the TMRC in the infants' and children's diets would be 0.000847 mg/kg/day or 9.4% of the RfD.

As indicated above, infants and children have a low potential for quizalofop ethyl exposure because of both the low levels of canola oil in the diet, and the absence of detectable residues in field-treated canola. The toxicology profile of quizalofop ethyl demonstrates low mammalian toxicity. Because there was no evidence that offspring were uniquely susceptible to the toxic effects of quizalofop ethyl, an additional 10-fold uncertainty factor should not be required to protect infants and children. Therefore, the RfD of 0.009 mg/kg/day, which utilizes a 100-fold safety factor, is appropriate to assure a reasonable certainty of no harm to infants and children from aggregate exposure to quizalofop ethyl.

F. International Tolerances

Harmonization of Tolerances: Since there are no Mexican or Codex MRLs/tolerances, compatibility is not a problem at this time. Compatibility cannot be achieved with the Canadian negligible residue type limit at 0.1 ppm at the USA use pattern, which had findings of real residues above 0.1 ppm. (James Tompkins)

[FR Doc. 97–32935 Filed 12–16–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–782; FRL–5759–1]

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–782, must be received on or before January 16, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides 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 to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 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
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224, e-mail: miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
James Tompkins (PM 25).	Rm. 239, CM #2, 703–305–5697, e-mail: tompkins.james@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–782] (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 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [PF–782] 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: December 3, 1997.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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