Billing: The rates listed above will be applied to the amount of capacity reserved, payable whether utilized or not.

For Losses: Capacity and energy losses incurred in connection with the transmission and delivery of capacity and energy under this rate schedule shall be supplied by the customer in accordance with the transmission service agreement.

[FR Doc. 99–3114 Filed 2–8–99; 8:45 am]

BILLING CODE 6450–01–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–856; FRL–6058–3]

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–856, must be received on or before March 11, 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 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 I. Miller (PM 23)	Rm. 237, CM #2, 703–305–6224, e-mail:miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Sidney Jackson (PM 23)	Rm. 233, CM #2, 703–305–7610, e-mail: jackson.sidney@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–856] (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. Comments and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number (insert docket number) 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: January 29, 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. Dow AgroSciences LLC

PP 8F 3600

EPA has received a pesticide petition (8F 3600) from Dow AgroSciences LLC, 9330 Zionsville Road, Indianapolis, IN 46268, 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 the herbicide clopyralid in or on the raw agricultural commodity sugar beet, roots at 2.0 parts per million (ppm) and sugar beet, tops at 3.0 ppm and on the processed agricultural commodity (PAC) sugar beet, molasses at 16.0 ppm. at sugar beet, roots at 2.0 ppm and sugar beet, tops at 3.0 ppm and on the processed agricultural commodity (PAC) sugar beet, molasses at 16.0 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*. The metabolism in plants is adequately understood. No metabolites of significance were detected in plant metabolism studies.

2. *Analytical method*. There is a practical analytical method for detecting and measuring levels of clopyralid in or on food with a limit of quantitation (LOQ) of 0.05 ppm that allows monitoring of food with residues at or above the levels set in these tolerances. EPA has provided information on this method to FDA. The method is available to anyone who is interested in pesticide residue enforcement.

3. *Magnitude of residues*. Tolerances for residues of the herbicide clopyralid in or on the following raw agricultural commodities, sugar beet roots and tops and the processed agricultural commodity molasses, were established on August 12, 1988 (53 FR 33488, 33489) at 0.5, 0.5, and 7.0 ppm, respectively, based upon residue data generated by Craven Laboratories. The validity of these data were in question and Dow AgroSciences repeated the residue studies. The last of the required residue data using a 105 day pre-harvest interval (PHI) were submitted to the Agency in June 1994. This pesticide petition proposes increased tolerances based upon data using a 45 day PHI. Residues of clopyralid were determined in roots and tops from several varieties of sugar beets, which were harvested from plots treated at the currently labeled season-maximum rate of 0.25 lb ae/acre in one application. Field test plots were located at thirteen sites representing the major U.S. production areas. Highest residues at the 45 day PHI for roots averaged 0.91 µg/g with the highest individual value of 1.47 µg/g, and for tops averaged 1.52 µg/g with the highest individual value of 2.48 µg/g. Based on the data, it is expected that residues of clopyralid in or on sugar beets as a raw agricultural commodity will not exceed proposed revised tolerances of 2 µg/g in roots and 3 µg/g in tops when the PHI is 45 days or longer. With a concentration factor of 8, the proposed tolerance for sugar beet molasses is 16 µg/g. The proposed revised tolerances would adequately cover these anticipated residues.

B. Toxicological Profile

1. *Acute toxicity*. Clopyralid has low acute toxicity. The rat oral LD_{50} is 5,000 milligram/kilograms (mg/kg) or greater for males, and females. The rabbit dermal LD_{50} is greater than 2,000 mg/kg and the rat inhalation LC_{50} is greater than 1.0 mg/L air (the highest attainable concentration). In addition, clopyralid is not a skin sensitizer in guinea pigs and is not a dermal irritant. Technical clopyralid is an ocular irritant but ocular exposure to the technical material would not normally be expected to occur to infants or children or the general public. End use formulations of clopyralid have similar low acute toxicity profiles and most have low ocular toxicity as well. Therefore, based on the available acute toxicity data, clopyralid does not pose any acute dietary risks.

2. *Genotoxicty*. Clopyralid is not genotoxic. The following studies have been conducted and all were negative for genotoxic responses. Ames bacterial mutagenicity assay (with and without exogenous metabolic activation); Host-Mediated assay *In vivo* cytogenetic test, rat; *In vivo* cytogenetic test, mouse; *In vivo* dominant lethal test, rat; *In vitro* unscheduled DNA synthesis assay in primary rat hepatocyte cultures; *In vitro* mammalian cell gene mutations assay in Chinese hamster ovary cell cultures (with and without exogenous metabolic activation).

3. *Reproductive and developmental toxicity*. Developmental toxicity was studied using rats and rabbits. The developmental study in rats resulted in a developmental no-observed adverse effect level (NOAEL) of > 250 milligram/kilograms/day (mg/kg/day) (a maternally toxic dose) and a maternal toxicity NOAEL of 75 mg/kg/day. A 1974 study in rabbits revealed no evidence of developmental or maternal toxicity at 250 mg/kg/day; thus the developmental and maternal NOAEL was > 250 mg/kg/day. A more recent study in rabbits (1990) resulted in developmental and maternal NOAELs of 110 mg/kg/day based on maternal toxicity at 250 mg/kg/day. Based on all of the data for clopyralid, there is no evidence of developmental toxicity at dose levels that do not result in maternal toxicity. In a 2-generation reproduction study in rats, pups from the high dose group which were fed diets containing clopyralid had a slight reduction in body weight during lactation and an increase in liver weights in F1a and F1b weanlings. The NOAEL for parental systemic toxicity was 500 mg/kg/day. There was no effect on reproductive parameters at > 1,500 mg/kg/day nor was there an adverse effect on the morphology, growth or viability of the offspring; thus, the reproductive NOAEL is > 1,500 mg/kg/day.

4. *Subchronic toxicity*. The following studies have been conducted using clopyralid. In a rat 90 day feeding study, Fischer 344 rats were fed diets containing clopyralid at doses of 5, 15, 50 or 150 mg/kg/day with no adverse effects attributed to treatment. In a second study, Fischer 344 rats were fed diets containing clopyralid at doses of 300, 1,500 and 2,500 mg/kg/day. Effects at the highest doses were decreased food consumption accompanied by decreased body weights and weight gains in both males and females. Slightly increased mean relative liver and kidney weights were noted in males of all doses, and in females at the top 2 doses. Because there were no other effects, the kidney and liver weight effects were judged as being adaptive rather than directly toxic. The no-observed adverse effect level (NOAEL) was 1,500 mg/kg/day for males and females. The NOAEL was 300 mg/kg/day for females. In a mouse 90 day feeding study, B6C3F1 mice were fed diets containing clopyralid at doses of 200, 750, 2,000 or 5,000 mg/kg/day. A slight decrease in body weight occurred at the top dose in both sexes. The liver was identified as the target organ based on slight increases in liver weights and minimal microscopic alterations at the higher dose levels. The liver changes were considered to be reversible and adaptive. The NOAEL for males was 2,000 mg/kg/day, and for females was 750 mg/kg/day. In a 180 day feeding study, beagle dogs were fed diets containing clopyralid at doses of 15, 50 or 150 mg/kg/day; there were no adverse effects. In a second dietary study, dogs also were fed diets containing clopyralid at doses of 15, 50 or 150 mg/kg/day; the only effect was an increase in the mean relative liver weight in females at the 150 mg/kg/day. In a 21 day dermal study, clopyralid was applied by repeated dermal application to New Zealand White rabbits at dose levels up to 1,000 mg/kg/day. Treatment produced no systemic effects.

5. *Chronic toxicity*. In a chronic toxicity and oncogenicity study, Sprague-Dawley rats were fed diets containing clopyralid at doses of 5, 15, 50 or 150 mg/kg/day. The only effect was a trend toward a decreased body weight of female rats receiving the 150 mg/kg/day dose with a NOAEL of 50 mg/kg/day. In a second study clopyralid was fed to Fischer 344 rats in the diet at doses of 15, 150 or 1,500 mg/kg/day. The effects were confined almost entirely to the 1,500 mg/kg/day dose groups and included slightly decreased food consumption and body weights, slightly increased liver and kidney

weights and macroscopic and microscopic changes in the stomach. No tumorigenic response was present. The NOAEL for this study was 150 mg/kg/day. B6C3F1 mice were maintained for 2 years on diets formulated to provide targeted dose levels of 10, 500 or 2,000 mg/kg/day. The only evidence of toxicity was body weight depression in males dosed at 2,000 mg/kg/day. There was no evidence of tumorigenic response at any dose level. Based on the chronic toxicity data, EPA has established the RfD for clopyralid at 0.5 mg/kg/day. The RfD for clopyralid is based on a 2 year chronic oncogenicity study in rats with a no-observed-effect level (NOAEL) of 50 mg/kg/day and an uncertainty (or safety) factor of 100. Thus, it would not be necessary to require the application of an additional uncertainty factor above the 100-fold factor already applied to the NOAEL. Using its Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), clopyralid would be classified as Group E for carcinogenicity (no evidence of carcinogenicity) based on the results of the carcinogenicity studies. There was no evidence of carcinogenicity in 2 year feeding studies in mice and rats at the dosage levels tested. The doses tested are adequate for identifying a cancer risk. Thus, a cancer risk assessment would not be appropriate.

6. *Animal metabolism*. Disposition and metabolism of clopyralid were tested in male and female rats at a dose of 5 mg/kg (oral). The majority of a radioactive dose was excreted in 24 hours of all dose groups. Fecal elimination was minor. Detectable levels of residual radioactivity were observed in the carcass and stomach at 72 hours post-dose. HPLC and TLC analysis of urine and fecal extracts showed no apparent metabolism of clopyralid.

7. *Metabolite toxicology*. There are no clopyralid metabolites of toxicological significance.

8. *Endocrine disruption*. There is no evidence to suggest that clopyralid is neurotoxic.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure under these tolerances, exposure is estimated based on the theoretical maximum residue contribution (TMRC) from the existing and this proposed amended tolerance for clopyralid on food crops. The TMRC is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of those food products eaten by various population subgroups. Exposure of humans to residues could also result if such residues are transferred to meat, milk, poultry or eggs. The following assumptions were used in conducting this exposure assessment. 100% of the crops were treated, the raw agricultural commodities (RAC) residues would be at the level of the tolerance, certain processed food residues would be at anticipated (average) levels based on processing studies and all current and pending tolerances were included. This results in an over estimate of human exposure and a conservative assessment of risk. Based on a NOAEL of 50 mg/kg/day in a 2 year chronic feeding/oncogenicity study in the rat and a hundredfold safety factor, the RfD would be 0.5 mg/kg/day. Consequently, these tolerances have a TMRC of 0.010277 mg/kg/day and would utilize approximately 2.1% of the RfD for the general U.S. population.

i. *Food*. The Toxicology database indicates there is no concern regarding acute and chronic dietary risk since the available data do not indicate any evidence of significant toxicity from exposure by the oral route.

ii. *Drinking water*. Another potential source of dietary exposure to residues of pesticides are residues in drinking water. There is no established maximum concentration level (MCL) for residues of clopyralid in drinking water. Although there has been limited detections at parts per billion (ppb) levels in some of the specially designed studies under highly vulnerable test conditions, no ongoing monitoring studies (U.S. Geological Survey, Selected Water Resources Abstracts; Pesticides in Ground WaterDatabase - A Compilation of Monitoring Studies: 1971-1991 National Summary; U.S. Department of Agriculture, AGRICOLA database; and, U.S. Department of Commerce, National Technical Information Service) have reported residues of clopyralid in ground or surface waters.

Based on the physical and chemical characteristics of clopyralid, such as water solubility and its stability under hydrolysis and photolysis, it has potential for downward movement through the soil profile. However, the behavior of the compound under field conditions demonstrates fairly rapid degradation and limited downward movement. Degradation based on 20 field dissipation sites indicated an average half-life of 25 days. Degradation is driven primarily by microbial processes. Downward movement through the soil profile was generally confined to the upper 18 inches of the soil profile. Validated computer modeling also predicted the maximum depth of residues to be 18-inches, with no detections predicted at 6 months after application.

Because the laboratory derived physical/chemical properties of clopyralid indicate a potential for downward movement, lysimeter studies were conducted. In a U.S. study, undisturbed soil columns (lysimeters), 8 inches in diameter, and 3 feet deep, were treated with 950 g ae/ha (about 5 x labeled use rates) in actual field conditions. Residues of clopyralid in soil as well as soil-solution (leachate) were collected in the closed system. The average depth of movement for the majority of clopyralid (center of mass) was 11 inches, and no detectable residues were observed in the leachate. In a European study, lysimeters 1-3 ft. diameter, and 3 ft. deep, were treated with 120 and 240 g ae/ha in actual field conditions. The average center of mass was 12 inches. No detectable residues were observed in the lysimeters. The amount of ^{14}C in leachate accumulated over 2 years in the degraded loess and silty sand lysimeters, was only 0.6% and 0.3% of applied, respectively. The leachate concentrations of ^{14}C-labeled clopyralid in degraded loess and silty sand throughout the first 10-16 months of the study ranged from 0.002-0.14 µg/l ppb and 0.003-0.02 ppb, respectively. A second European lysimeter study with silty sand lysimeters treated with 120 g ae/ha revealed a 2 year cumulative clopyralid leachate of only 0.1% of applied (0.04 ppb). These studies demonstrate that in lysimeter test systems, under field environmental conditions, clopyralid rapidly dissipates through mineralization to carbon dioxide. Also the very low levels observed in leachate demonstrate that there is very little potential for clopyralid to leach through soil and contaminate ground water.

In summary, these data on potential water exposure indicate insignificant additional dietary intake of clopyralid and any exposure is more than compensated for in the conservative dietary risk evaluation. Therefore, it is concluded that there is a reasonable certainty of no harm even at potential upper limit exposures to clopyralid from drinking water.

2. *Non-dietary exposure*. There is a non-dietary use registered under the Federal Insecticide, Fungicide and Rodenticide Act. The use is for weed control in residential turf and ornamentals. Potential exposures for children from non-occupational uses is therefore limited to turf and ornamental re-entry and this exposure is low.

3. *Short-term or intermediate-term*. The data for clopyralid does not indicate any evidence of significant toxicity by the dermal and inhalation routes. Consequently, there is no concern for short-term or intermediate-term residential risk. Therefore, a short-term or intermediate-term residential risk assessment would not be required.

4. *Chronic*. As part of a hazard assessment process an endpoint of concern is determined for the chronic occupational or residential risk assessment. However, as indicated, the exposures that would result from the use of clopyralid are of an intermittent nature. The frequency and duration of these exposures do not exhibit a chronic exposure pattern. The exposure does not occur often enough to be considered a chronic exposure; i.e., a continuous exposure that occurs for at least several months. Therefore, it would not be appropriate to aggregate exposure from the residential use with exposure from food and drinking water.

5. *Acute*. No concern would exist for an acute dietary assessment for clopyralid because the available data indicates no evidence of significant toxicity from a 1 day or single event exposure by the oral route. Therefore, an acute dietary risk assessment would not be required.

D. Cumulative Effects

The potential for cumulative effects of clopyralid and other substances that have a common mechanism of toxicity was considered. The mammalian toxicity of clopyralid is well defined. However, no reliable information exists to indicate that toxic effects produced by clopyralid would be cumulative with those of any other chemical compound. Additionally, clopyralid does not appear to produce a toxic metabolite produced by other substances. Therefore, consideration of a common mechanism of toxicity with other compounds is not appropriate at this time. Thus, only the potential exposures to clopyralid were considered in the aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Based on a NOAEL of 50.80 milligram/kilogram/body weight/day (mg/kg/bwt/day) from a 2 year rat feeding study with a decreased mean bwt gain effect, and using an uncertainty factor of 100 to account for the interspecies extrapolation and intraspecies variability, a RfD of 0.5 mg/kg/bwt/day was used for this assessment of chronic risk. As indicated, there is no endpoint of concern identified with acute and short- or intermediate-term exposures. Based on the known toxicity and exposure data, the proposed and existing tolerances would utilize approximately 2.1% of the RfD for the U.S. population. And, as indicated previously, whatever upper limit might be used for drinking water exposure, the exposure estimate for clopyralid would not exceed the RfD. 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 appreciable risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to clopyralid residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of clopyralid, data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat were considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism during prenatal development resulting from pesticide exposure to one or both parents. Reproduction studies provide (i) information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and (ii) data on systemic toxicity.

Developmental toxicity was studied using rats and rabbits. The developmental study in rats resulted in a developmental NOAEL of > 250 mg/kg/day (a maternally toxic dose), and a maternal toxicity NOAEL of 75 mg/kg/day. A 1974 study in rabbits revealed no evidence of developmental or maternal toxicity at 250 mg/kg/day; thus the developmental andmaternal NOAEL was > 250 mg/kg/day. A more recent study in rabbits (1990) resulted in developmental and maternal NOAEL's of 110 mg/kg/day based on severe maternal toxicity at 250 mg/kg/day. Based on all of the data for clopyralid, there is no evidence of developmental toxicity at dose levels that do not result in maternal toxicity.

In a 2-generation reproduction study in rats, pups from the high dose group which were fed diets containing clopyralid had a slight reduction in bwt during lactation and an increase in liver weights in F1a and F1b weanlings. The NOAEL for parental systemic toxicity was 500 mg/kg/day. There was no effect on reproductive parameters at > 1,500 mg/kg/day nor was there an adverse effect on the morphology, growth or viability of the offspring; thus, the reproductive NOAEL is > 1,500 mg/kg/day.

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 toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. These data suggest minimal concern for developmental or reproductive toxicity and do not indicate any increased pre- or post-natal sensitivity. Therefore, an additional uncertainty factor is not necessary to protect the safety of infants and children and that the RfD at 0.5 mg/kg/day is appropriate for assessing aggregate risk to infants and children.

The percent of the RfD that will be utilized by the aggregate exposure from all tolerances to clopyralid will be much less than 10% for non-nursing infants and for children (1-6 years of age). Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to clopyralid residues.

F. International Tolerances

There are no Codex maximum residue levels established for clopyralid.

2. IR4 Project

PP 8E4983, 8E5019, 8E5020,8E5021, and 8E5024

EPA has received pesticide petitions (PP 8E4983, 8E5018, 8E5019, 8E5020, 8E5021, and 8E5024) from the Interregional Research Project Number 4 (IR-4), 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 tolerances for residues of the insecticide, tebufenozide (benzoic acid, 3,5-dimethyl-, 1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl)hydrazide) in or on the raw agricultural commodities.

1. *PP 8E4983* proposed the establishment of a tolerance for blueberries at 2.0 parts per million (ppm), and PP 8E5018 proposed a tolerance for caneberries at 1.0 ppm. Subsequently, IR-4 amended these tolerance proposals to include a single tolerance at 3.0 ppm for berries (Crop Group 13) that will include both blueberries, and caneberries under PP 8E4983.

2. *PP 8E5024* proposes the establishment of tolerances for canola seed at 1.75 ppm, and canola oil at 3.75 ppm.

3. *PP 8E5019* proposes the establishment of a tolerance for cranberries at 1.0 ppm.

4. *PP 8E5021* proposes the establishment of a tolerance for mint at 10.0 ppm.

5, *PP 8E5020* proposes the establishment of tolerances for turnips tops at 9.0 ppm, and turnip roots at 0.25 ppm.

EPA has determined that the petitions contain data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of tebufenozide in plants (grapes, apples, rice, and sugar beets) is adequately understood for the purpose of these tolerances. The metabolism of tebufenozide in all crops was similar and involves oxidation of the alkyl substituents of the aromatic rings primarily at the benzylic positions. The extent of metabolism and degree of oxidation are a function of time from application to harvest. In all crops, parent compound comprised the majority of the total dosage. None of the metabolites were in excess of 10% of the total dosage.

2. *Analytical method*. High performance liquid chromatographic (HPLC) analytical methods using ultraviolet(UV) detection have been validated for blueberries, raspberries, canola seed and oil, cranberries, mint foliage and oil, and turnip roots and tops. The methods involve extraction by blending with solvents, purification of the extracts by liquid-liquid partitions and final purification of the residues using solid phase extraction column chromatography. The limits of quantitation (LOQ) is 0.005 ppm for blueberries, 0.01 ppm for canola seed and meal, mint foliage, raspberries, and turnip roots and tops, 0.02 ppm for mint oil, 0.03 ppm for canola soapstock and oil, and 0.05 ppm for cranberries.

3. *Magnitude of residues*. Field residue trials were conducted with a 70 wettable power (WP) formulation in geographically representative regions of the U.S. A total of 8 field residue trials were conducted in blueberries. The average blueberry residue value from all trials was 0.81 ppm.

A total of 7 field residue trials were conducted in canola. The average canola seed residue value from all trials was 0.84 ppm. Two processing studies were conducted. Average residues in meal, soapstock and oil were 0.11 ppm, 0.83 ppm, and 1.75 ppm, respectively. Residues did not concentrate in soapstock (Concentration Factor (CF) is less than 1), and a tolerance in soapstock is not needed. For oil, the average CF is 2.26, and the proposed tolerance is 3.75 ppm (2.26 times 1.58 ppm).

A total of 6 field residue trials were conducted in cranberries. The average cranberry residue value from all trials was 0.30 ppm.

A total of 5 field residue trials were conducted in mint. The average mint foliage residue value from all trials was 7.11 ppm. Mint oil was prepared from foliage from two residue trials. The average oil residue was 0.23 ppm. Since residues do not concentrate in oil, a tolerance is not needed.

A total of 5 field residue trials were conducted in raspberries. The average raspberry residue value from all trials was 0.62 ppm.

A total of 6 field residue trials were conducted in turnips. The average residue value from all trials was 0.10 ppm for roots, and 2.27 ppm for tops.

B. Toxicological Profile

1. *Acute toxicity*. Results of a battery of toxicological studies show tebufenozide has low acute toxicity. Tebufenozide Technical was practically non-toxic by ingestion of a single oral dose in rats, and mice (LD_{50} > 5,000 milligram/kilograms (mg/kg)) and was practically non-toxic by dermal application LD_{50} > 5,000 mg/kg. Tebufenozide Technical was not significantly toxic to rats after a 4 hour inhalation exposure with an LC_{50} value of 4.5 mg/L (highest attainable concentration), is not considered to be a primary eye irritant or a skin irritant, and is not a dermal sensitizer. An acute neurotoxicity study in rats did not produce any neurotoxic or neuropathologic effects.

2. *Genotoxicty*. Tebufenozide technical was negative (non-mutagenic) in an Ames assay with and without hepatic enzyme activation and in a reverse mutation assay with *E. coli*. Tebufenozide technical was negative in a hypoxanthine guanine phophoribosyl transferase (HGPRT) gene mutation assay using Chinese hamster ovary (CHO) cells in culture when tested with and without hepatic enzyme activation. In isolated rat hepatocytes, tebufenozide technical did not induce unscheduled DNA synthesis (UDS) or repair when tested up to the maximum soluble concentration in culture medium. Tebufenozide did not produce chromosome effects *in vivo* using rat bone marrow cells or *in vitro* using Chinese hamster ovary cells (CHO). On the basis of the results from this battery of tests, it is concluded that tebufenozide is not mutagenic or genotoxic.

3. *Reproductive and developmental toxicity*. See discussion of studies under section E.2. Infant and Children.

4. *Subchronic toxicity*— i. The no-observed adverse effect level (NOAEL) in a 90 day rat feeding study was 200 ppm (13 mg/kg/day for males, 16 mg/kg/day for females). The lowest-observed adverse effect level (LOAEL) was 2,000 ppm (133 mg/kg/day for males, 155 mg/kg/day for females). Decreased body weight in males, and females was observed at the LOAEL of 2,000 ppm. As part of this study, the potential for tebufenozide to produce subchronic neurotoxicity was investigated. Tebufenozide did not produce neurotoxic or neuropathologic effects when administered in the diets of rats for 3 months at concentrations up to and including the limit dose of 20,000 ppm (NOAEL = 1,330 mg/kg/day for males, and 1,650 mg/kg/day for females).

ii. In a 90 day feeding study with mice, the NOAEL was 20 ppm (3.4 and 4.0 mg/kg/day for males and females, respectively). The LOAEL was 200 ppm (35.3 and 44.7 mg/kg/day for males and females, respectively). Decreases in body weight gain were noted in male mice at the LOAEL of 200 ppm.

iii. A 90 day dog feeding study gave a NOAEL of 50 ppm (2.1 mg/kg/day for males and females). The LOAEL was 500 ppm (20.1 and 21.4 mg/kg/day for males and females, respectively). At the LOAEL, females exhibited a decrease in rate of weight gain and males presented an increased reticulocyte.

iv. A 10 week study was conducted in the dog to examine the reversibility of the effects on hematological parameters that were observed in other dietary studies with the dog. Tebufenozide was administered for 6 weeks in the diet to 4 male dogs at concentrations of either 0 or 1,500 ppm. After the 6 weeks, the dogs receiving treated feed were switched to the control diet for 4 weeks. Hematological parameters were measured in both groups prior to treatment, at the end of the 6 weeks treatment, after 2 weeks of recovery on the control diet and after 4 weeks of recovery on the control diet. All hematological parameters in the treated/recovery group were returned to control levels indicating that the effects of tebufenozide on the hemopoietic system are reversible in the dog.

v. In a 28 day dermal toxicity study in the rat, the NOAEL was 1,000 mg/kg/day highest dose tested (HDT). Tebufenozide did not produce toxicity in the rat when administered dermally

for 4 weeks at doses up to and including the limit dose of 1,000 mg/kg/day.

5. *Chronic toxicity*— i. A 1 year feeding study in dogs resulted in decreased red blood cells, hematocrit, and hemoglobin and increased Heinz bodies, reticulocytes, and platelets at the LOAEL of 8.7 mg/kg/day. The NOAEL in this study was 1.8 mg/kg/day.

ii. An 18 month mouse carcinogenicity study showed no signs of carcinogenicity at dosage levels up to and including 1,000 ppm, the HDT.

iii. In a combined rat chronic/oncogenicity study, the NOAEL for chronic toxicity was 100 ppm (4.8 and 6.1 mg/kg/day for males and females, respectively), and the LOAEL was 1,000 ppm (48 and 61 mg/kg/day for males and females, respectively). No carcinogenicity was observed at the dosage levels up to 2,000 ppm (97 mg/kg/day and 125 mg/kg/day for males and females, respectively).

6. *Animal metabolism*. The adsorption, distribution, excretion and metabolism of tebufenozide in rats was investigated. Tebufenozide is partially absorbed, is rapidly excreted and does not accumulate in tissues. Although tebufenozide is mainly excreted unchanged, a number of polar metabolites were identified. These metabolites are products of oxidation of the benzylic ethyl or methyl side chains of the molecule. These metabolites were detected in plant and other animal (rat, goat, and hen) metabolism studies.

7. *Metabolite toxicology*. Common metabolic pathways for tebufenozide have been identified in both plants (grape, apple, rice, and sugar beet), and animals (rat, goat, and hen). The metabolic pathway common to both plants and animals involves oxidation of the alkyl substituents (ethyl and methyl groups) of thearomatic rings primarily at the benzylic positions. Extensive degradation and elimination of polar metabolites occurs in animals such that residues are unlikely to accumulate in humans or animals exposed to these residues through the diet.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Tolerances have been established (40 CFR 180.482) for the residues of tebufenozide, in or on a variety of raw agricultural commodities. A permanent tolerance has been established for the residues of tebufenozide in/on walnuts at 0.1 ppm, and pecans at 0.05 ppm. Permanent tolerances at 0.5 ppm and 1.0 ppm have been established for imported wine grapes, and apples, respectively. Other proposed tolerances are pending. Risk assessments were conducted by Rohm and Haas to assess dietary exposures and risks from tebufenozide as follows:

ii. *Acute exposure and risk*. No acute endpoint was identified for tebufenozide and no acute risk assessment is required.

iii. *Chronic exposure and risk*. For chronic dietary risk assessment, it is assumed that 100% of all crops which are consumed will contain residues of tebufenozide at the tolerance levels. The Reference Dose (RfD) used for the chronic dietary analysis is 0.018 mg/kg/day. Potential chronic exposures were estimated using NOVIGEN'S Dietary Exposure Evaluation Model (DDEM Version 5.03b) which uses USDA food consumption data from the 1989-1992 survey. The existing and proposed tebufenozide tolerances result in a theoretical maximum residue contribution (TMRC) that is equivalent to 34.5% of the RfD for the U.S. population, 61.4% of the RfD for infants, 70.4% of the RfD for non-nursing infants (> 1 year old), and 79.8% of the RfD for chlidren 1 to 6 years old. The chronic dietary risks from these uses do not exceed EPA's level of concern.

iv. *Drinking water*. Submitted environmental fate studies suggest that tebufenozide is moderately persistent to persistent and mobile, and could potentially leach to groundwater and runoff to surface water under certain environmental conditions. However, in terrestrial field dissipation studies, residues of tebufenozide and its soil metabolites showed no downward mobility and remained associated with the upper layers of soil. Foliar interception (up to 60% of the total dosage applied) by target crops reduces the ground level residues of tebufenozide. There is no established Maximum Concentration Level (MCL) for residues of tebufenozide in drinking water. No drinking water health advisory levels have been established for tebufenozide. There is no entry for tebufenozide in the ''Pesticides in Groundwater Database'' (EPA 734-12-92-001, September 1992).

v. *Chronic exposure and risk*. There are insufficient water-related exposure data to complete a comprehensive drinking water assessment for tebufenozide at this time. However, in order to mitigate the potential for tebufenozide to leach into groundwater or runoff to surface water, precautionary language has been incorporated into the product label. Considering the precautionary language on the label and based on the Registrant's knowledge of environmental occurrence of the chemicals, significant exposure from residues of tebufenozide in drinking water is not anticipated.

2. *Non-dietary exposure*. Tebufenozide is not currently registered for any indoor or outdoor residential uses; therefore, no non-dietary residential exposure is anticipated.

D. Cumulative Effects

The potential for cumulative effects of tebufenozide with other substances that have a common mechanism of toxicity was considered. Tebufenozide belongs to the class of insecticide chemicals known as diacylhydrazines. The only other diacylhydrazine currently registered for non-food crop uses is halofenozide. Tebufenozide and halofenozide both produce a mild, reversible anemia following subchronic/chronic exposure at high doses; however, halofenozide also exhibits other patterns of toxicity (liver toxicity following subchronic exposure and developmental/systemic toxicity following acute exposure) which tebufenozide does not. Given the different spectrum of toxicity produced by tebufenozide, Rohm Haus concludes that there is no reliable data at the molecular/mechanistic level which would indicate that toxic effects produced by tebufenozide would be cumulative with those of halofenozide (or any other chemical compound).

Based on the overall pattern of toxicity produced by tebufenozide in mammalian and insect systems, the compound's toxicity appears to be distinct from that of other chemicals, including organochlorines, organophosphates, carbamates, pyrethroids, benzoylureas, and other diacylhydrazines. Thus, according to Rohm Haus, there is no evidence to date to suggest that cumulative effects of tebufenozide and other chemicals should be considered.

E. Safety Determination

1. *U.S. population*— i. *Acute exposure and risk*. Since no acute endpoint was identified for tebufenozide, no acute risk assessment is required.

ii. *Chronic exposure and risk*. Using the conservative exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of tebufenozide from existing, pending and proposed tolerances is 34.5% for the U.S. population. Aggregate exposure (food and water) are not expected to exceed 100%. EPA generally has no concern for exposures below 100% of

the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Rohm and Haas concludes that there is a reasonable certainty that no harm will result from aggregate exposure to tebufenozide residues to the U.S. population.

2. *Infants and children-children*— i. *In general*. In assessing the potential for additional sensitivity of infants and children to residues of tebufenozide, data from developmental toxicity studies in the rat and rabbit, and 2-generation reproduction studies in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

ii. *Developmental toxicity studies*— a. *Rats*. In a developmental toxicity study in rats, the maternal (systemic) NOAEL was 250 mg/kg/day. The LOAEL was 1,000 mg/kg/day based on decrease body weight and food consumption. The developmental (pup) NOAEL as > 1,000 mg/kg/day HDT.

b. *Rabbits*. In a developmental toxicity study in rabbits, the maternal and developmental NOAELs were > 1,000 mg/kg/day HDT.

iii. *Reproductive toxicity study rats*. In a multigeneration reproductive toxicity study in rats, the parental (systemic) NOAEL was 0.85 mg/kg/day. Spleenic pigmentation changes and extramedullary hematopoiesis occurred at the LOAEL of 12.1 mg/kg/day. In addition to these effects, decreased body weight gain and food consumption occurred at 171.1 mg/kg/day. The reproductive (pup) NOAEL was 12.1 mg/kg/day. The reproductive LOAEL of 171.1 mg/kg/day was based on a slight increase in the number of pregnant females that did not deliver or had difficulty and had to be sacrificed. Additionally at the LOAEL, in F1 dams, the length of gestation increased and implantation sites decreased significantly. In a second study, reproductive effects were not observed at 2,000 ppm (the NOAEL equal to 149-195 mg/kg/day) and the NOAEL for systemic toxicity was determined to be 25 ppm (1.9-2.3 mg/kg/day).

iv. *Pre- and post-natal sensitivity*— a. *Pre-natal sensitivity*. The developmental NOAELs of > 1,000 mg/kg/day HDT from the developmental toxicity studies in rats and rabbits demonstrate that there is no developmental (prenatal) toxicity present for tebufenozide.

Additionally, these developmental NOAELs are greater than 500-fold higher than the NOAEL of 1.8 mg/kg/day from the 1 year feeding study in dogs which was the basis of the RfD.

b. *Post-natal sensitivity*. In the reproductive toxicity study in rats, the reproductive NOAEL (12.1 mg/kg/day from the first study; 149-195 mg/kg/day from the second study) is between 14-fold higher than the parental NOAEL (0.85 mg/kg/day) in the first study and 83-fold higher than the parental NOAEL (1.8-2.3 mg/kg/day) in the second study. These data indicate that post-natal toxicity in the reproductive studies occurs only in the presence of significant parental toxicity. These developmental and reproductive studies indicate that tebufenozide does not have additional post-natal sensitivity for infants and children in comparison to other exposed groups. Because these reproductive effects occurred in the presence of parental (systemic) toxicity and were not replicated at the same doses in a second study, these data do not indicate an increased pre-natalor post-natal sensitivity to children and infants (that infants and children might be more sensitive than adults) to tebufenozide exposure.

c. *Acute exposure and risk*. Since no acute endpoint was identified for tebufenozide, no acute risk assessment is required.

d. *Chronic exposure and risk*. With the existing, pending and proposed tolerances for tebufenozide, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of tebufenozide range from 39.9% for nursing infants less than 1 year old to 79.8.% for children 1 to 6 years old. Aggregate exposure (food and water) are not expected to exceed 100%. Rohm and Haas concludes that there is a reasonable certainty that no harm will result from aggregate exposure to tebufenozide residues to non-nursing infants.

F. International Tolerances

There are currently no CODEX, Canadian or Mexican maximum residue levels (MRLs) established for tebufenozide in blueberries, caneberries, canola, cranberries, mint or turnips so no harmonization issues are required for this action.

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FEDERAL COMMUNICATIONS COMMISSION

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

February 3, 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(s), 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 April 12, 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 Judy Boley, Federal Communications Commission, Room 1–C804, 445 12th Street, SW, DC 20554 or via the Internet to jboley@fcc.gov.

FOR FURTHER INFORMATION CONTACT: For additional information or copies of the information collection(s), contact Judy Boley at 202–418–0214 or via the Internet at jboley@fcc.gov.

SUPPLEMENTARY INFORMATION:

OMB Control No.: 3060–0812.

Title: Assessment and Collection of Regulatory Fees.

Form No.: N/A.

Type of Review: Extension of currently approved collection.

Respondents: Individuals or households, business or other for-profit, not-for-profit institutions.