1995, imidacloprid was not detected in seventeen 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.

2. *Non-dietary exposure*— i. *Residential turf*. Bayer Corporation 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 are 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 NOAEL 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 - 33.3 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 Corporation. 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 x 10^7 and 2.4 x 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 two percent 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 NOAEL 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 Occupational and Residential Exposure Branch (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. Cumulative Effects

No other chemicals having the same mechanism of toxicity are currently registered, therefore, there is no risk from cumulative effects from other substances with a common mechanism of toxicity.

E. 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. The TMRC from exposure to field corn for the general population, is 0.000055 mg/kg/bwt/day, which represents 0.1% of the RfD. 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 2 generations, as well as any observed systemic toxicity.

FFDCA Section 408 provides that the EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for prenatal and postnatal effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for imidacloprid relative to prenatal and postnatal effects is complete. Further for imidacloprid, the NOAEL 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 NOAELs 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, EPA has concluded that the TMRC from use of imidacloprid from published uses is 0.008358 mg/kg/bwt/day utilizing 14.7% of the RfD for the general population. For the most highly exposed subgroup in the population, non-nursing infants (less than 1 year old), the TMRC for the published tolerances is 0.01547 mg/kg/day. This is equal to 27.1% of the RfD. The TMRC from exposure to field corn to non-nursing infants is 0.000131 mg/kg/bwt/day, which represents 0.2% of the RfD. The TMRC for children ages 1 to 6 years is 0.000130 mg/kg/bwt/day, which represents 0.2% of the RfD. For nursing infants, the TMRC is 0.000032 mg/kg/bwt/day, which is 0.1% of the RfD. For children ages 7 to 12 years, the TMRC is 0.000098 mg/kg/bwt/day, which is 0.2% of the RfD. Thus, it can be concluded that there is a reasonable certainty that no harm will result from additional exposure of infants and children.

F. International Tolerances

No CODEX Maximum Residue Levels (MRLs) have been established for residues of imidacloprid on any crops at this time.

[FR Doc. 01–370 Filed 1–4–01; 8:45 am]
BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–989; FRL–6761–4]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket control number PF–989, must be received on or before February 5, 2001.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION.** To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–989 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Indira Gairola, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–8375; e-mail address: gairola.indira@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations,'' ''Regulations and Proposed Rules,'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this action under docket control number PF–989. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–989 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically.* You may submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–989. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the

name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical 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 this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

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

Dated: December 21, 2000.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by the petitioner and represents the view of the petitioner. EPA is publishing the petition summary verbatim without editing it 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.

Morflex Inc.

PP 8E4966, PP 8E4967

EPA has received two pesticide petitions (PP 8E4966, PP 8E4967) from Morflex, Inc., 2110 High Point Road, Greensboro, North Carolina 27403. proposing, pursuant to section 408(d) of the (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish an exemption from the requirement of a tolerance for acetyl tributyl citrate (Citroflex® A4) and triethyl citrate (Citroflex®) when used as inert ingredients in or on growing crops, when applied to raw agricultural commodities (RAC) after harvest or when applied to animals (40 CFR 180.1001(c), and (e)). 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

Residue chemistry data are generally not required by EPA regarding decisions relevant to exemptions from the requirement of a tolerance for inert ingredient. However, applicable dietary modeling data and environmental fate data have been completed and is used for the assessments included in these petitions. Since Morflex is requesting an exemption from the requirement of a tolerance, an analytical method is not required.

B. Toxicological Profile

1. *Acute toxicity*—i. *Oral LD_{50} in rats.* Acetyl tributyl citrate (ATBC). The acute oral LD_{50} for ATBC is 31.5 grams/kilograms body weight (g/kg bwt). Rising doses of ATBC were administered to groups consisting of 5 rats per group of from 10.5 to 31.5 g/kg bwt. Some animals appeared sluggish, however, they recovered during the 21–day post dosing observation period. There were no mortalities at any dose.

ii. *Triethyl citrate (TEC).* The acute oral LD_{50} of TEC in rats was determined to be 7 milligrams/Liters (mL)/kg bwt. The technical material triethyl citrate was administered to groups of 5 rats by stomach tube at doses ranging from 5 to 15 mL/kg bwt. Signs of toxicity occurred within 1–hour and included weakness, depression, ataxia, hyperexcitability, unrest, urinary incontinence, irregular, and labored respiration, convulsions preceeding death in some animals. Mortalities occurred in 2 hours to 3 days, while survivors recovered within 15 hours to 4 days.

iii. *Oral LD_{50} in cats—ATBC.* The acute oral LD_{50} of ATBC was determined to be greater than 50 mL/kg bwt. The animals showed signs of slight nausea, and within a few hours they developed a diarrhea with oozing of the oily material from the rectum. The diarrhea subsided in less than 24 hours. There were no systemic toxicity signs as judged by the general appearance and behavior of the animals for periods up to 2 months.

iv. *TEC.* The acute oral LD_{50} of TEC was determined to be approximately 4 g/kg bwt in cats. TEC was administered by stomach tube to cats fasted for 24 hours in doses ranging from 1.1 to 10.8 g/kg bwt. Signs of toxicity consisted of nausea, vomiting, ataxia, weakness, muscle twitching, tremors, lowered body temperature, gasping, and shallow respiration, prostration, convulsions, respiratory failure and death. Mortalities occurred in about 2 hours to 2 days. Animals surviving recovered within 4 hours to 3 days depending upon the dose administered. Postmortem examinations showed no abnormalities of the thoracic abdominal organs related to the toxic signs.

v. *Intraperitoneal LD_{50} in mice—ATBC.* The acute intraperitoneal LD_{50} of ATBC was determined to be greater than 4g/kg bwt in Swiss Albino mice. The animals were observed for gross effects on appearance and behavior for 72 hours after dosing.

vi. *TEC.* The intraperitoneal LD_{50} of TEC was determined to be 1.75 g/kg bwt in Swiss Albino mice. Signs of toxicity included rapid loss of righting reflex without loss of consciousness, increased respiration rate, and clonic convulsions. Mortalities occurred during the first hour post dosing.

vii. *Intraperitoneal LD_{50} in rats.* The acute intraperitoneal LD_{50} of TEC in rats is 4.2 mL/kg bwt for females and 4.0 mL/kg bwt for males. Most deaths occurred within one hour post dosing following a depression of respiration and clonic convulsions. Pathological examinations of the animals that died indicated hemorrhage of the lung, pancreas and thymus, and marked congestion in the kidneys and liver.

viii. *Acute subcutaneous LD_{50} in rats.* The subcutaneous administration of TEC to rats resulted in LD_{50} of 6.7 mL/kg bwt in females and 6.6 mL/kg bwt in males. Mortalities typically occurred within 24 hours of dosing. Pathological examinations showed extensive hemorrhage in the lungs, and thymus, loss of hair, edema, and crust formation at injection sites. In surviving animals, at the end of the 14–day observation period, necrotic ulcers were noted at injection sites.

ix. *Acute dermal LD_{50} in guinea pig and rabbit.* The dermal LD_{50} of TEC was determined to be greater than 11.4 g/kg bwt in guinea pigs and greater than 5.7 mg/kg bwt in rabbits.

x. *Acute inhalation LC_{50} in rats.* The 6–hour inhalation LC_{50} of TEC in rats was determined to be approximately 1,300 ppm. In this study, groups of rats were exposed to vaporized TEC for 6 hours at concentrations between 1,300 and 3,500 ppm.

xi. *Skin irritation in rabbits—ATBC.* ATBC was found to be non-irritating to rabbit skin when applied as the undiluted technical material. The abdomens of 3 male Albino rabbits were clipped and 1 mL of ATBC was applied

to the intact skin daily for 4 days. The animals were observed for a period of 36 hours after the last application. There was no evidence of irritation.

xii. *TEC*—TEC was determined to be non-irritating to rabbit skin. Undiluted TEC was applied to intact or abraded rabbit skin for 24 hours under occlusion before scoring for irritation.

xiii. *Guinea pig sensitization—ATBC.* ATBC was found to be non sensitizing to the skin of Guinea pigs following the method Magnusson and Kligman's Guinea pig maximumization test. Sensitization was induced in guinea pigs by intradermal injections of the test substance and complete Freunds Adjuvant. The induction process was supplemented 7 days later by application of ATBC to the shoulder injection sites under occlusion. Fourteen days later the animals were challenged by occluded patches. Challenges were repeated after 1–week. Evaluations for contact sensitization were performed at 24 and 48 hours after patch removal.

xiv. *TEC.* TEC was found to be a strong sensitizer in 9 of 10 Guinea pigs after the first challenge and in all 10 Guinea pigs after the second challenge. TEC was tested for the potential to induce contact dermatitis according to the Magnusson-Kligman's Guinea pig maximization test method. Sensitization was induced by intradermal injections of both test substance and Freunds Adjuvant and the induction process supplemented 7 days later by the test substance applied to the shoulder injections sites under occlusion. The animals were challenged by occluded patch 14 days later.

xv. *Human repeated insult patch test—ATBC.* ATBC was evaluated in 59 human subject panelists (males and females) in the repeated insult patch test of Draize. The test substance was found not to induce dermal irritation or contact sensitization. For this test, each of the 59 panelists received a test patch (20x20 cm) moistened with 0.4 mL of ATBC to the upper arms 3 times a week for 3 weeks. Patches were secured in place for 24 hours before removal. Duplicate challenges were made 2 weeks after the final serial applications, 1 set of patches to original sites and 1 set to adjacent sites. Patch sites were scored prior to patch applications and scored at 48 and 96 hours after applications.

xvi. *TEC.* Triethyl citrate was tested in an adaptation of the repeat insult patch test of Draize in 59 human subject panelists (males and females). A quantity of 0.4 mL of undiluted TEC was applied to each test patch prior to application. Patches were applied to each panelist 3 times a week for 3 consecutive weeks. Instructions were given to each panelist to keep the patches dry and to remove them 24 hours after application. Duplicate challenge applications were made 2 weeks after the final serial applications; 1 at the original site and 1 at an adjacent site. The patch sites were evaluated at 48 and 96 hours after application. There was no evidence of dermal irritation and no reactions suggestive of contact sensitization in any of the panelists.

2. *Genotoxicty*—i. *ATBC. Ames Salmonella/microsome reverse mutation assay.* ATBC did not exhibit mutagenic activity in the Ames assay with or with metabolic activation. ATBC was tested in a preincubation modification of the Ames assay with *Salmonella typhimurium* tester strains TA98, TA100, TA1535, and TA1537. Tests were performed in all strains, both with and without metabolic activation using S–9 rat liver systems. Assays were repeated twice in all strains. Another test was performed with ATBC using *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538, with and without metabolic activation using rat liver S–9 mix or hamster liver S–9 mix. Results were negative for mutagenicity in all 5 strains in the presence of both rat and hamster liver S–9 mix and in the absence of metabolic activation.

ii. *Mouse lymphoma mutagenesis assay.* ATBC produced a negative response in cultures with and without metabolic activation using Arochlor induced rat liver S–9 mix. The test article was assayed for mutagenic potential using thymidine kinase locus of L51784 TK+/-mouse lymphoma cells.

iii. *In vitro chromosomal aberration assay in rat lymphocytes.* ATBC did not exhibit clastogenic activity (increases in chromosomal aberrations) in cultured rat lymphocytes as compared with negative controls, either in the presence or absence of metabolic activation. ATBC was evaluated in a cytogenic assay using rat lymphocyte cells with and without rat liver S–9 mix metabolic activation. Frequencies of chromosomal aberrations, based upon mitotic indicies were determined from ATBC treated cultures and were found not to be significantly different than negative controls. Based upon the results of this study, ATBC did not exhibit clastogenic activity in cultured rat lymphocytes.

iv. *Chinese hamster ovary cell/hypoxanthine-guanine-phosphoribosyl transferase (CHO/HGPRT) forward mutation assay.* In this forward mutation assay, ATBC in 2 independent tests, did not induce a mutagenic response. ATBC was evaluated both in the absence and presence of rat liver S–9 mix metabolic activation. The forward mutation frequencies of ATBC treated cultures were not significantly different from those of negative controls, indicating no mutagenic response.

v. *Unscheduled DNA synthesis in rats.* ATBC did not induce unscheduled DNA systhesis (UDS) in livers from rats treated with commercial material at a dose of 10 mL/kg.

3. *Genotoxicity—TEC.* Microbial assays, *Salmonella typhimurium and Saccaromyces cerevisiae.* TEC was not mutagenic in *Salmonella typhimurium* strains TA1535, TA1537, and TA1538 and in *Saccharomyces cerevisiae* strain D4, without metabolic activation, and with metabolic activation using S–9 mix from male mouse, rat and monkey livers. Plate tests and suspension tests were performed with the indicator strains of both test organisms. Based upon cell toxicity studies, concentrations from 0.4 to 1.7% were employed as the dose levels in the mutagenicity assays. Results were negative for mutagenicity with both bacteria and yeast organisms, with both the plate and suspension tests, with and without metabolic activation.

4. *Reproductive and developmental toxicity*—i. *ATBC. A 2–generation reproduction study in rats.* A 2–generation reproduction study conducted with ATBC in Sprague Dawley rats resulted in a no observed effect level (NOEL) of 100 milligrams/kilogram body weight mg/kg bwt/day based upon the lowest observed effect level (LOEL) of 300 mg/kg bwt/day for decreased maternal bwts gains and water consumption and reduced bwts and slightly higher mortalities among their offspring. This 2–generation reproduction study was conducted in Sprague Dawley rats with ATBC at dietary levels of 100, 300, or 1,000 mg/kg bwt/day to evaluate the potential effects on reproductive performance and on the survival and growth of offspring through 2–generations. In this study, 4 groups of male and female rats received control or 1 of the 3 dietary levels of ATBC continuously. Prior to mating, males were treated for 77 days and females for 21 days. After mating, males of the F_0 generation were removed and pregnant females were continued on diet through gestation, delivery and lactation. Subsequent F_1 offspring were maintained on the same diets as their parents for at least 10 weeks prior to mating within groups. The resulting F_2 generation litters were also maintained on the same diets as their parents for at least 14 days.

ii. *TEC. Developmental toxicity in the developing chicken embryo.* Treatment

of chicken embryos with TEC resulted in a negative teratogenic response. In this study, TEC was dissolved in ethanol to deliver a maximum of 10 mg per egg. The test substance in solution was administered by 2 routes, into the yolk and through the air sac. For each route, eggs were treated at 2 stages of incubation: preincubation (0–hour), and at the fourth day (96– hour).

5. *Subchronic toxicity*—i. *ATBC.* Fourteen–day range finding dietary toxicity in rats. In a 14–day range finding feeding study with ATBC, the NOEL was determined to 1,000 mg/kg bwt/day. In this study ATBC was administered in the diet at concentrations of 1%, 2.5% and 5% equivalent to doses of 1,000, 2,700 and 5,000 mg/kg bwt/day. Observations included clinical signs of toxicity, bwts, food intake, test substance intake, complete gross pathology including organ weights, and histopathologic examinations of livers. Food intake was initially decreased in all test groups, however, differences persisted in only among males of the 5,000 mg/kg bwt/day group. The initial differences are likely related to the unpalatability of the diet. Body weights were significantly lower among animals of the 2,700 mg/kg bwt/day and 5,000 mg/kg bwt/day treatment groups throughout the study. Organ weight determinations resulted in significantly increased relative liver weights among high dose females. Upon microscopic examinations of the livers there were increased cytoplasmic eosinophilia and a concomitant reduction of glycogen content of hepatocytes in periportal areas from animals of the 2,700 mg/kg bwt/day and 5,000 mg/kg bwt/day dose groups.

ii. *Ninety–day dietary toxicity in rats.* The results of a 90–day feeding study with ATBC resulted in a NOEL of 300 mg/kg bwt/day based upon the LOEL of 1,000 mg/kg bwt/day for minor changes is relative liver weights, liver enzymes and bilirubin levels. This study was conducted Sprague Dawley rats receiving dietary levels of ATBC of 0, 100, 300, or 1,000 mg/kg bwt/day for 90 days. All animals were observed daily for clinical signs of toxicity. Ophthalmoscopic observations were conducted in all animals of the highest dose group at pretest, and just prior to the treatment period. Body weights were recorded daily for all animals on day 1 of treatment and weekly thereafter. Food consumption was measured over 1 week periods, while water consumption was measured in each animal during the first and eleventh week of dosing. The results of clinical chemistries, hematology and urinalysis were recorded and complete necropsies with histological examinations were performed. A few statistically significant differences were noted between animals of the high dose group (1,000 mg/kg bwt/day) and controls including increased relative liver weights, liver enzymes, and bilirubin levels. However, there were no histopathological findings indicative of treatment related effects.

iii. *TEC. Subchronic oral* toxicity in mice. TEC was evaluated for subchronic toxicity in a group of 20 mice receiving 350 mg/kg bwt/day of commercial grade test substance (purity >99%) in 3% acacia intraperitoneally, daily for 14 consecutive days. A control group consisting of the same number of mice received 3% acacia daily under the same schedule. Body weight gains of TEC treated mice were significantly lower as compared with controls by day 7. There were no significant differences in red and white blood cell counts, clotting times, and hemoglobin levels between treated and control mice. Under the conditions of the study, the LOEL was established at 350 mg/kg bwt/day, when given intraperitoneally for 14 days.

iv. *Subchronic dietary toxicity in rats.* In an 8 week dietary feeding study in rats with TEC, the NOEL was established at 4 g/kg bwt/day. Groups of approximately 4 males and 4 females were administered TEC in the diet at concentrations of 0, 0.5, 1.0, or 2.0%. These dietary concentrations were estimated to be equivalent to 0, 1, 2, or 4 g/kg bwt/day TEC. TEC administered daily in the diet at doses up to approximately 1/2 of the rat oral LD_{50} had no significant effect on growth. Blood counts including red and white blood cell counts, differential cell counts were not significantly among treatment and control groups. There were no, gross findings in thoracic or abdominal organs at necropsy. Histological sections of organs, including the heart, lungs, gastrointestinal tract, liver, pancreas, spleen, and kidneys, revealed no differences between treatment and control animals.

v. *Subchronic toxicity in dogs.* In this study, 4 dogs were given daily doses of 2.5 to 3.5 mL/kg bwt/day (2,840 to 3,975 mg/kg bwt/day) as rising doses for 7 to 12 weeks. The study report indicates bwt gains were normal as were results of urinalysis and serum chemistries. Hematology results suggested a tendency to anemia. Organ weights were normal except for one abnormally heavy liver. At these doses severe and widespread liver pathology was evident. Other organs were reportedly normal. As the purpose of the study was to determine the toxic dose for repeated administrations of TEC, the NOEL was not established.

6. *Chronic toxicity*—i. *ATBC. 2–year chronic toxicity in rats.* A 2–year chronic toxicity study conducted with ATBC in Sherman rats at dietary concentrations of 0, 200, 2,000, or 20,000 ppm (equivalent to 0, 10, 100, or 1,000 mg/kg bwt/day) resulted in a NOEL of 1,000 mg/kg bwt/day. Animals were observed for physical appearance and behavior throughout the study as were individual bwts. All animals that died and those sacrificed at the end of the study were examined for gross and histological changes. No differences in behavior or physical appearance was noted among treated and control animals. There were no statistically significant differences between the growth of animals treated with ATBC and controls. There were no statistical differences in mortalities among treatment and control animals. Inflammatory disease of the lungs was the most common finding at autopsy, however, there was no treatment related differences. There were no differences in tumor frequencies among treatment and control animals. There was no reported evidence of effects on the endocrine system.

ii. *TEC. 2–year chronic dietary toxicity in rats.* In this study, TEC administered to rats for 2 years via dietary administration resulted in no significant effects at the highest dose tested, equivalent to 1,500 mg/kg bwt/day. Sprague Dawely rats (15 per sex per dose group) were fed diets containing TEC at concentrations of 0, 0.33, 1.0, or 3.0% for 2 years. These dietary concentrations are estimated to be equivalent to 0, 165, 500, or 1,500 mg/kg bwt/day. Clinical observations were made daily and individual bwts were measured weekly. Blood and urine evaluations were conducted at specified intervals. Scheduled interim sacrifices of animals included macroscopic examinations of thoracic and abdominal organs and microscopic examinations of the kidney and liver tissues. All animals that died spontaneously during the study, as well as all animals remaining at the termination of study (1 or 2 years), were examined by a pathologist. At terminal sacrifice, microscopic examinations were made of kidney, liver, heart, lungs, spleen, stomach, small intestine, adrenals, ovaries, uterus, testes, and seminal vesicles. There were transiently lower bwts among males of the high dose group animals, possibly related to the unpaletibility of the diet. There were no significant differences observed between treated and control groups for the

following blood examinations: hemoglobin, erythrocyte count, non-protein nitrogen, and sugar determination. Urine tests for reaction, albumin, reducing substances, and microscopic evaluation were all considered to be normal. Terminal and interim autopsies disclosed no findings that were significant or attributable to TEC treatment. Size and weight of organs of the principal tissues at the time of autopsy were unremarkable. There were no significant differences between treated and control animals in comparison to the pathological findings.

iii. *Six months dietary toxicity in dogs*. In a 6 month dietary toxicity study in dogs, TEC did not exhibit any toxic effects and the NOEL is greater than 280 mg/kg bwt/day the highest dose tested (HDT). Groups of 4 Beagle or Beagle type dogs (males and females) were administered 6 days per week for 6 months at dietary levels of TEC equivalent to 55 or 280 mg/kg bwt/day. The dogs were observed daily, weighed weekly and urinalysis were conducted at 3 and 6 months after initiation of the study. Blood samples were taken at 2, 4, and 6 months after initiation of dosing for hematological examinations. Dogs were sacrificed at the end of the in-life dosing phase and necropsied. Body weight gain and clinical observations were normal throughout the study. No significant changes or abnormalities were reported in hematology, serum chemistry or urinalysis during the course of the study. Gross examinations of major organs and organ weights at necropsy were normal. Histopathologic examinations of the major organs did not show any abnormalities.

7. *Animal metabolism*—i. *ATBC*. Metabolism and disposition of acetyl tributyl citrate in male Sprague Dawley rats. The metabolism of ATBC using ^{14}C–ATBC in rats receiving single oral doses of 70 mg/kg. ATBC was determined to be rapidly absorbed and excreted with an elimination half-life of 3.4 hours. Greater than 98% of administered ^{14}C was achieved via urine, feces and in expired air 48 hours after dosing. Urinary metabolites identified in this study include acetyl citrate, monobutyl citrate, acetyl monobutyl citrate, dibutyl citrate, and acetyl dibutyl citrate.

ii. *Metabolism of acetyltributylcitrate (ATBC) and tributylcitrate (TBC) in human serum and rat liver homogenates*. The metabolism of ATBC and the intermediate deacetylated metabolite tributylcitrate (TEC), was studied *in vitro* using human serum and rat liver homogenates. At a concentration of 100 µg/mL in human serum, ATBC was found to undergo extensive metabolism with a half-life of approximately 32 hours. Also, at a concentration of 100 µg/mL in rat liver homogenate, ATBC was found to undergo extensive and complete metabolism with a half-life of approximately 10 minutes. There is very little or no emonstrable TBC in the 2 test systems because of the rapid further metabolism of this intermediate metabolite. The metabolic half-life of TBC in human serum and rat liver homogenate was approximately 4 hours and a few seconds, respectively. These studies confirm the ready and complete conversion of ATBC and TBC via ester hydrolysis to acetic acid, citric acid and butanol. Butanol would be expected to undergo oxidation to butyric acid and further metabolism by b-oxidation.

iii. *TEC. Absorption, distribution, metabolism and excretion of tiethyl citrate in the rat*. Following a single oral 2 mg/kg dose of ^{14}C–TEC in rats, a peak blood concentration of about 1.48 µg eq./g blood was achieved at 15 minutes post-administration, blood concentration rapidly decreased to about 0.05 µg eq./gm blood after 1 hour and was barely detectable after 24 hours. Tissue distribution was examined after single oral administration of a 2 mg/kg dose of ^{14}C–TEC to rats. At 15 minutes post-administration, relatively high ^{14}C concentrations were found in the didney (37.81+ 5.02 µg eq./g tissue), stomach (10.00+ 3.53 µg eq./g tissue), small intestines including contents (10.65 + 3.15 µg eq./g tissue) and liver (4.40 + 0.77 µg eq./g tissue). By 24 hours after dosing, the ^{14}C concentrations detected in most tissues had decreased to near the detection limit (0.01 µg eq./g tissue), with the exception of the large intestine including contents. Cumulative urinary, fecal and expiratory excretions of ^{14}C–TEC were 93, 0.2 and 1%, respectively, 8 hours after administration of a single 2 mg/kg dose of ^{14}C–TEC. At 120 hours after dosing, the total ^{14}C excretion of urine, feces and expiration had reached 99%. Metabolism of ^{14}C–TEC was investigated using the 24–hour urine of rats after a single oral administration of a 2 mg/kg dose. Three major metabolites were separated by thin-layer chromatography and identified using gas chromatography (GC/MS). Two of the metabolites were isomers of diethyl citrate and 1 was found to be monoethyl citrate.

8. *Endocrine disruption*. Chronic and reproductive toxicity data conducted with ATBC and chronic toxicity data conducted with TEC are without adverse effects to reproductive or the endocrine system. Also, the compounds do not share structural similarities with currently known or chemicals suspected to have endocrine disruptive properties.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. ATBC and TEC are currently classified as generally recognized as safe (GRAS) for use in foods and food packaging, cosmetics, pharmaceuticals, and as plasticizers for consumer and packaging products. The current petition, requests the exemption from tolerances for these compounds when used as inert ingredients in agricultural formulations for use on growing crops for post harvest applications to food crops and applications to animals. Although residue data are generally not required for inert ingredient exemptions from tolerances, Morflex, Inc. has developed worst case assumptions using Novigen Sciences Dietary Exposure Evaluation Model (DEEM) with data inputs based upon the model of Kenaga and Hoergers: Maximum Expected Residues on Vegetation. The Kenega nomogram is used to predict maximum residue levels present on day 0 following different application rates of a chemical to 1 of 6 different categories of plants or plant parts. The 3 basic features of the Kenaga nomogram-catagories of plants and plant parts, maximum predicted residue levels, and a linear dose-residue relationship. Crops and crop groups selected for this analysis include the following: leafy vegetables (succulent or dried), fruiting vegetables, cucurbit vegetables, citrus fruits, pome fruits, stone fruits, berries, cereal grains, grapes, and bananas. The reference dose chosen for this analysis, was derived from the NOEL resulting from a chronic rat (2–year) study conducted with ATBC. This study was conducted at dietary concentrations of 0, 200, 2,000, and 20,000 ppm equivelant to 0, 10, 100, and 1,000 mg/kg bwt/day of ATBC. No effects were reported up to the HDT. Therefore, for the purposes of this assessment, a chronic reference dose (RfD) of 10 mg/kg bwt/day was used. The chronic RfD includes an uncertainty factor of 100 to account for intra-species and inter-species variations. Food consumption data from the United States Department of Agriculture (USDA) CSFII conducted in 1994 through 1996, were used to estimate dietary exposure. The levels of ATBC and TEC can vary depending upon the percent of ATBC and TEC in the formulation and/or the application rate of the product. For purposes of this screening level assessment, an application rate of 3 pounds per acre of ATBC or TEC was assumed. Also, no adjustment was made for percent crop

treated and all commodities contain residues at predicted day zero levels. For this screening level assessment with an application rate of 3 pounds ATBC or TEC per acre, the following 0–time level residues are predicted from the nomogram: leafy vegetables–375 ppm, legume vegetables–36 ppm, fruiting vegetables, cucurbit vegetables, citrus fruits, pome fruits, stone fruits, berries, cereal grains, grapes, and bananas–21 ppm.. Using the above modeling parameters, chronic exposure was estimated for the overall U.S. population and 25 population subgroups. Chronic exposure for the overall U.S. population was estimated to be 0.492873 mg/kg bwt/day, representing 4.9% of the RfD. The exposure estimate for the most highly exposed population subgroup, children 1-6 years of age, was 0.984312 mg/kg bwt/day, or 9.8%.

ii. *Drinking water*. Based upon the chemical and physical properties, and the environmental fate characteristics, ATBC and TEC are not expected to persist environmentally, nor result in significant concentrations in drinking water sources.

2. *Non-dietary exposure*. ATBC and TEC are currently used in non-food use pesticide formulations, as well as in food, food packaging, cosmetics, medical devices and pharmaceuticals, and as plasticizers.

D. Cumulative Effects

Cumulative effects are not expected since ATBC and TEC are rapidly degraded to natural substances.

E. Safety Determination

1. *U.S. population*. Based upon the dietary residue exposure analysis using the Kenega nomogram, the most sensitive population, children 1–6 years, was 0.984312 mg/kg bwt/day or 9.8% of the RfD for the crops and crop groups used in this assessment. Results of a 2–generation reproduction study with ATBC did not reveal developmental or reproduction effects at doses up to 100 mg/kg bwt/day. Also, based on the absence of pup toxicity up to the dose level (1,000 mg/kg bwt/day) producing maternal effects, there is no evidence of special post-natal sensitivity to infants and children. It is concluded that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to acetyl tributyl citrate (ATBC) or triethyl citrate (TEC) when used as inert ingredients in agricultural formulations of pesticides.

2. *Infants and children*. No embryotoxic, developmental, or teratogenic effects have been associated with acetyltributyl citrate (ATBC) or triethyl citrate (TEC).

F. International Tolerances

Morflex Inc. is unaware of any International tolerances or CODEX maximum residue limits (MRL's) for acetyltributyl citrate (ATBC) or triethyl citrate (TEC) on any crop or livestock commodities.

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BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[FRL–6930–2]

Notice of Tentative Approval, Request for Comments and Solicitation of Requests for a Public Hearing for Public Water System Supervision Program Revision for the Commonwealth of Virginia

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of Tentative Approval and Solicitation of Requests for a Public Hearing.

SUMMARY: Notice is hereby given in accordance with the provision of section 1413 of the Safe Drinking Water Act as amended, and the rules governing National Primary Drinking Water Regulations that the Commonwealth of Virginia has revised its approved Public Water System Supervision Primacy Program. Specifically, Virginia has adopted Consumer Confidence Report regulations requiring annual drinking water quality reports from community water suppliers. EPA has determined that these regulations are no less stringent than the Federal provisions and satisfy the requirements of the Federal regulations. Therefore, EPA has decided to tentatively approve the program revisions. All interested parties are invited to submit written comments on this determination and may request a public hearing.

DATES: Comments or a request for a public hearing must be submitted by February 5, 2001. This determination shall become effective on February 5, 2001 if no timely and appropriate request for a hearing is received and the Regional Administrator does not elect to hold a hearing on his own motion, and if no comments are received which cause EPA to modify its tentative approval.

ADDRESSES: Comments or a request for a public hearing must be submitted to Patti Kay Wisniewski, Drinking Water Branch (3WP22), U.S. Environmental Protection Agency Region III, 1650 Arch Street, Philadelphia, PA 19103–2029.

All documents relating to this determination are available for inspection between the hours of 8:00 a.m. and 4:30 p.m., Monday through Friday, at the following offices:

- Drinking Water Branch, Water Protection Division, U.S. Environmental Protection Agency, Region III, 1650 Arch Street, Philadelphia, Pennsylvania 19103–2029; and
- Virginia Department of Health, Division of Water Supply Engineering, 1500 East Main Street, Richmond, Virginia 23218.

FOR FURTHER INFORMATION CONTACT: Patti Kay Wisniewski at the Philadelphia address given above; telephone (215) 814–5668 or fax (215) 814–2318.

SUPPLEMENTARY INFORMATION: All interested parties are invited to submit written comments on this determination and may request a public hearing. All comments will be considered, and, if necessary, EPA will issue a response. Frivolous or insubstantial requests for a hearing may be denied by the Regional Administrator. However, if a substantial request for a public hearing is made by February 5, 2001, a public hearing will be held. A request for public hearing shall include the following: (1) The name, address, and telephone number of the individual, organization, or other entity requesting a hearing; (2) a brief statement of the requesting person's interest in the Regional Administrator's determination and of information that the requesting person intends to submit at such a hearing; and (3) the signature of the individual making the request; or, if the request is made on behalf of an organization or other entity, the signature of a responsible official of the organization or other entity.

Dated: December 27, 2000.

Bradley M. Campbell,

Regional Administrator, EPA, Region III.

[FR Doc. 01–362 Filed 1–4–01; 8:45 am]
BILLING CODE 6560–50–M

FEDERAL COMMUNICATIONS COMMISSION

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

December 20, 2000.

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