List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: July 29, 1997.

Janet L. Andersen,

Director, Biopesticides and Pollution Prevention Division, Office of Pesticide Programs.

[FR Doc. 97–20987 Filed 8–7–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPP–30418A; FRL–5734–4]

Thermo Trilogy, Inc.; Approval of Pesticide Product Registrations

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces Agency approval of applications to register the pesticide products Daza Technical and Daza 4.5 WDG, containing an active ingredient not included in any previously registered products pursuant to the provisions of section 3(c)(5) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended.

FOR FURTHER INFORMATION CONTACT: By mail: Rita Kumar, Biopesticides and Pollution Prevention Division (7501W), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. CS51B6, Westfield Building North Tower, 2800 Crystal Drive, Arlington, VA 22202, (703) 308–8291; e-mail: kumar.rita@epamail.epa.gov.

SUPPLEMENTARY INFORMATION:

Electronic Availability: Electronic copies of this document and the Fact Sheet are available from the EPA home page at the Environmental Sub-Set entry for this document under ''Regulations'' (http://www.epa.gov/fedrgstr/).

EPA issued a notice, published in the **Federal Register** of September 4, 1996 (61 FR 46642; FRL–5391–9), which announced that AgriDyne Technologies, Inc., 2401 S. Foothill Drive, Salt Lake City, UT 84109, had submitted applications to register the pesticide products Daza Technical and Daza 4.5 WDG (EPA File Symbols 62552–RE and 62552–RU), containing the new active ingredient dihydroazadirachtin at 17.5 and 4.5 percent respectively, an active ingredient not included in any previously registered products.

The applications for Daza Technical and Daza 4.5 WDG were later transferred to Thermo Trilogy, Inc., 1500 Grace Drive, Columbia, MD 21044–4098. The products were designated new EPA File Symbols (70051–RE and 70051–RU), containing the same active ingredient dihydroazadirachtin at 17.5 and 4.5 percent respectively.

The applications were approved on June 11, 1997 and June 23, 1997, respectively, as Daza Technical for manufacturing use only (EPA Registration Number 70051–29) and Daza 4.5 WDG for indoor and outdoor use in ornamentals, turf, agronomic and horticultural crops (EPA Registration Number 70051–31).

The Agency has considered all required data on risks associated with the proposed use of dihydroazadirachtin, and information on social, economic, and environmental benefits to be derived from use. Specifically, the Agency has considered the nature of the chemical and its pattern of use, application methods and rates, and level and extent of potential exposure. Based on these reviews, the Agency was able to make basic health and safety determinations which show that use of dihydroazadirachtin when used in accordance with widespread and commonly recognized practice, will not generally cause unreasonable adverse effects to the environment.

More detailed information on these registrations is contained in an EPA Pesticide Fact Sheet on dihydroazadirachtin.

A copy of this fact sheet, which provides a summary description of the pesticides, use patterns and formulations, science findings, and the Agency's regulatory position and rationale, may be obtained from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161.

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are available for public inspection in the Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Rm. 1132, CM #2, Arlington, VA 22202 (703-305–5805). Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of Information Office (A-101), 401 M St., SW., Washington, D.C. 20460. Such requests should: (1) Identify the product name and registration number and (2) specify the data or information desired.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: July 29, 1997.

Janet L. Andersen,

Director, Biopesticides and Pollution Prevention Division, Office of Pesticide Programs.

[FR Doc. 97–20989 Filed 8–7–97; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[PF–752; FRL–5732–6]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–752, must be received on or before September 8, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7506C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

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

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

comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
George LaRocca (PM 13).	Rm. 204, CM #2, 703–305–6100, e-mail:larocca.george@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Mary Waller Acting (PM 21).	Rm. 265, CM #2, 703–308–9354, e-mail: waller.mary@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–752] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–752] and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

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

Dated: July 28, 1997.

Donald R. Stubbs,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

1. E.I. du Pont de Nemours & Co. (Dupont)

PP 4F3023

EPA has received a request to amend pesticide petition (PP 4F3023) from E.I. du Pont de Nemours & Co. (Dupont), P. O. Box 80038, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of esfenvalerate, (*S*)-cyano-(3-phenoxyphenyl)methyl (*S*)-4-chloro-alpha-(1-methylethyl) benzeneacetate in or on the raw agricultural commodity, celery. The enforcement analytical method for determining residue is gas chromatography with nitrogen phosphorus detection. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism and chemical nature of residues of fenvalerate in plants is adequately understood. The fate of fenvalerate has been extensively studied using radioactive tracers in plant and animal metabolism/nature of the residue studies previously submitted to the Agency. These studies have demonstrated that the parent compound is the only residue of toxicological significance.

2. *Analytical method*. There is a practical analytical method utilizing electron-capture gas chromotography available for enforcement with a limit of detection that allows monitoring food with residues at or above tolerance levels.

3. *Magnitude of residues*. Current tolerances are based on the sum of all isomers of fenvalerate. Fenvalerate is a racemic mixture of four isomers (about 25% each). This product was registered as Pydrin. However since 1992, an *S,S*-isomer enriched formulation, Asana (esfenvalerate), has been the only fenvalerate formulation sold in the U.S. Since the *S,S*-isomer is the insecticidally active isomer, the use rate for Asana is four times lower than that for Pydrin. A petition is pending (PP 4F4329), to convert tolerances based on the use rates for Asana (still to be expressed as the sum of all isomers). Bridging studies have shown Asana residues to be 3-4 times lower than Pydrin residues.

Residue trials were conducted on celery at four sites using Asana XL and at two sites using Pydrin Insecticides in order to bridge data from 14 residue studies previously conducted using Pydrin alone. The mean esfenvalerate residue in untrimmed celery samples treated with Asana XL was 4.40 ppm (range 1.39 to 6.51 ppm). The mean fenvalerate residue in untrimmed celery samples treated with Pydrin was 12.0 ppm (range 4.78 to 19.1 ppm). Total fenvalerate residues were approximately three times lower after application of Asana XL Insecticide than after application of Pydrin Insecticide.

Since there are no processed commodities of celery, processing studies were not conducted. In addition, celery is not an animal feed item and, therefore, secondary residues will not be an issue.

B. Toxicological Profile

The following studies have been submitted to EPA:

1. *Acute toxicity*. A rat acute oral study on esfenvalerate technical with an LD_{50} of 87.2 mg/kg. A rabbit acute dermal study on esfenvalerate with an LD_{50} of > 2,000 mg/kg. Acute inhalation on technical grade a.i. waived due to negligible vapor pressure. A primary eye irritation test using esfenvalerate in the rabbit which showed mild irritation (conjunctivitis) that cleared by day 7. A primary dermal irritation test using esfenvalerate in the rabbit which showed minimal irritation that reversed within 72 hours after treatment (MRID 00156510). A dermal sensitization test on esfenvalerate in guinea pigs which showed no sensitization (MRID 41215203).

2. *Genotoxicty*. Esfenvalerate was not mutagenic in reverse mutation assays in *Salmonella* and *E. Coli* or in HGPRT *in vitro* assay in Chinese hamster lung cells. Esfenvalerate did not induce chromosome aberrations in an *in vitro* assay in Chinese hamster ovary cells. Esfenvalerate did not induce micronuclei in bone marrow of mice given up to 150 mg/kg intraperitoneally. Esfenvalerate did not induce unscheduled DNA synthesis in HeLa cells.

3. *Reproductive and developmental toxicity*. A pilot developmental study in the rat with doses of 0, 1, 2, 3, 4, 5, and 20 mg/kg/day esfenvalerate. The maternal NOEL was 3 mg/kg/day based on maternal clinical signs of abnormal gait or mobility at 4 mg/kg/day and above. A developmental study in the rat with doses of 0, 2.5, 5, 10, and 20 mg/kg/day esfenvalerate by gavage. There was no maternal NOEL but a maternal NOEL was established in the pilot study. Maternal signs observed at 2.5 mg/kg/day were erratic jerking and extension of forelimbs, rapid side-to-side head movement and excessive grooming. There were no fetal or developmental effects in either study at 20 mg/kg/day, the highest dose tested. Therefore, the fetal/developmental NOEL was > 20 mg/kg/day.

A pilot developmental study in the rabbit with doses of 0, 2, 3, 4, 4.5, 5, and 20 mg/kg/day esfenvalerate by gavage. The maternal NOEL was 2 mg/kg/day based on excessive grooming at 3 mg/kg/day and above. A developmental study in the rabbit with doses of 0, 3, 10, and 20 mg/kg/day esfenvalerate by gavage. There was no maternal NOEL but a maternal NOEL was established in the pilot study. There were no fetal or developmental effects in either study at the highest dose tested. Therefore, the fetal/developmental NOEL was > 20 mg/kg/day.

A 2-generation feeding study with esfenvalerate in the rat at dietary levels of 0, 75, 100, or 300 ppm. The high dietary concentration was lowered to 150 ppm for the second generation. Very mild body weight effects and sores at 75 ppm in both generations were considered secondary effects caused by scratching related to skin stimulation from dermal exposure. Therefore 75 ppm (4.2 mg/kg/day for first generation parental males, 5.6 mg/kg/day for first generation parental females, 6.0 mg/kg/day for second generation parental males, and 7.3 mg/kg/day for second generation parental females) was considered an NOAEL for both adult rats and their offspring. Effects were observed in adults and pups of both generations at 100 ppm and above. Pups were no more sensitive than adult animals.

4. *Subchronic toxicity*. A 90-day feeding study in rats conducted at 0, 75, 100, 125, and 300 ppm esfenvalerate with a NOEL of 125 ppm (6.3 mg/kg/day). This study provided intermediate dose levels to supplement a 90-day feeding study in rats conducted at 0, 50, 150, 300 and 500 ppm esfenvalerate with a NOEL of 50 ppm (2.5 mg/kg/day) based on jerky leg movements at 150 ppm (7.5 mg/kg/day) and above.

A 90-day feeding study in mice conducted at 0, 50, 150, and 500 ppm esfenvalerate and 2,000 ppm fenvalerate with a NOEL of 50 ppm esfenvalerate (10.5 mg/kg/day) based on lower glucose and triglycerides at 150 ppm. Neurologic symptoms were observed with 500 ppm esfenvalerate and 2,000 ppm fenvalerate.

Three-month subchronic study in dogs is satisfied by 1-year oral study in dogs, in which the NOEL was 200 ppm esfenvalerate (5 mg/kg/day). A 21-day dermal study in rabbits with fenvalerate conducted at 100, 300, and 1,000 mg/kg/day of fenvalerate with an NOAEL of 1,000 mg/kg/day fenvalerate.

5. *Chronic toxicity*. A 1-year study in which dogs were fed 0, 25, 50, or 200 ppm esfenvalerate with no treatment related effects at any dietary level. The NOEL was 200 ppm (5 mg/kg/day). An effect level for dietary administration of esfenvalerate for dogs of 300 ppm had been established earlier in the 2-week pilot study used to select dose levels for the chronic dog study.

A 20-month study with fenvalerate in mice fed 0, 10, 30, 100, and 300 ppm fenvalerate. The NOEL was 30 ppm (6mg/kg/day) based on red blood cell effects and granulomatous changes at 100 ppm. Fenvalerate was not carcinogenic at any concentration.

An 18-month study with esfenvalerate in mice fed 0, 35, 150, and 350 ppm esfenvalerate. Mice fed the 350 ppm dose were sacrificed within the first 2 months of the study, after excessive morbidity and mortality due to self-trauma induced by pharmacological effects on dermal sensory nerves. Therefore, data collected from the 350 ppm group were not used in the evaluation of the oncogenic potential of esfenvalerate. The NOEL was 35 ppm (4.29 and 5.75 mg/kg/day for males and females, respectively) based on lower body weight and body weight gain at 150 ppm. Esfenvalerate was not carcinogenic at either the 35 ppm or 150 ppm concentrations.

A 2-year study with fenvalerate in rats fed 1, 5, 25, and 250 ppm. A 1,000 ppm group was added to establish an effect level. The NOEL was 250 ppm (12.5 mg/kg/day). At 1,000 ppm, hind limb weakness, lower body weight, and higher organ-to-body weight ratios were observed. Fenvalerate was not carcinogenic at any concentration.

6. *Animal metabolism*.After oral dosing, fenvalerate was eliminated from rats within 5 days after dosing. The metabolic pathway involved cleavage of the ester linkage followed by hydroxylation, oxidation, and conjugation of the acid and alcohol moieties.

7. *Metabolite toxicology*.The parent molecule is the only moiety of toxicological significance which needs regulation in plant and animal commodities.

8. *Other potential toxicology considerations - endocrine effects*. Estrogenic effects have not been observed in any studies conducted on fenvalerate or esfenvalerate. In subchronic or chronic studies there were no lesions in reproductive systems of males or females. In the recent reproduction study with esfenvalerate, full histopathological examination of the pituitary and the reproductive systems of males and females was conducted. There were no compound-related gross or histopathological effects. There were also no compound-related changes in any measures of reproductive performance including mating, fertility, or gestation indices or gestation length in either generation.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing dietary exposure, chronic and acute dietary assessments have been conducted using all existing and pending tolerances for esfenvalerate. The toxicological endpoints used in both dietary assessments are derived from maternal NOEL's of 2.0 mg/kg/day

from rat and rabbit teratology studies. There were no fetal effects.

2. *Food*. A chronic dietary exposure assessment using anticipated residues and assuming that 100% of all crops are treated, found the percentages of the Reference Dose (RfD) utilized by the two most sensitive sub-populations to be 44% (Non-Nursing Infants <1 yr.) and 48% (Children 1-6 yrs.). This assessment also included all food tolerances for incidental food handling establishments which were set at 0.05 ppm (the limit of quantitation) since there were no detectable residues. The results have been adjusted from the study previously submitted to reflect the new RfD selected by EPA.

The Tier 3 acute dietary assessment has been rerun to incorporate current EPA thinking on processing studies and secondary residues that has arisen since the original study was submitted. The most sensitive sub-populations were determined to be: Non-Nursing Infants (< 1 yr.) with a Margin of Exposure (MOE) of 914 at the 95th percentile of exposure and an MOE of 254 at the 99th percentile of exposure; and Children (1-6 yrs.) with an MOE of 698 at the 95th percentile of exposure and 321 at the 99th percentile. The MOE's for the general population were 1,803 at the 95th percentile of exposure and 676 at the 99th percentile. This analysis used field trial residue data and market share data for the percent of crop treated. It also used Monte Carlo sampling and applied appropriate processing factors for apple juice and apple juice concentrate. Monte Carlo distribution was also used for meat and milk residues. Food handling establishment commodities were not included in the analysis because EPA methodology does not include them in Tier 3 exposure modeling.

3. *Drinking water*. Esfenvalerate is immobile in soil and, therefore, will not leach into groundwater. Additionally, due to the insolubility and lipophilic nature of esfenvalerate, any residues in surface water will rapidly and tightly bind to soil particles and remain with sediment, therefore not contributing to potential dietary exposure from drinking water. In addition, a screening evaluation of leaching potential of esfenvalerate has been conducted using DuPont's Tier 1 Ground Water Exposure Model (TIGEM, Version December 30, 1996) which is based on results from EPA's Pesticide Root Zone Model (PRZM, Version 2.0). Based on this screening assessment, the potential concentrations of esfenvalerate in shallow ground water are judged to be negligible.

4. *Non-dietary exposure*. Dietary exposure is the only significant route of chronic non-occupational exposure to esfenvalerate. However, esfenvalerate is registered for non-crop uses including spray treatments in and around commercial and residential areas, treatments for control of ectoparasites on pets, home care products including foggers, pressurized sprays, crack and crevice treatments, lawn and garden sprays, and pet and pet bedding sprays. For the non-agricultural products, the very low amounts of active ingredient they contain, combined with the low vapor pressure (1.5×10^{-9} mm Mercury at 25° C.) and low dermal penetration, would result in minimal inhalation and dermal exposure.

D. Cumulative Effects

The potential for cumulative effects of esfenvalerate and other pyrethroid insecticides that have a common mechanism of toxicity must also be considered. While risk assessment methodology has not been developed to estimate cumulative exposure to multiple pyrethroids, their similar insecticidal efficacy results in the substitution of one pyrethroid for another, rather than addition of pyrethroids. Because of the breadth of exposures included in the assumptions for esfenvalerate risk assessment, it is unlikely that there will be significant additive exposure to other pyrethroids.

These issues are extremely complex and require an extensive evaluation of a wealth of proprietary and published data across a broad range of pyrethroid insecticides in order to provide a scientifically sound interpretation upon which to base any regulatory judgments. The Pyrethroid Working Group is currently awaiting guidance from the Agency on cumulative effects. They anticipate having some preliminary evaluation data available for the Agency by August, 1997. For any interim decisions, the Agency should take into consideration the relatively benign toxicological profiles of pyrethroid insecticides and their long history of safe use.

E. Safety Determination

1. *U.S. population*.A chronic dietary exposure assessment using anticipated residues and assuming that 100% of all crops are treated, found the percentage of the RfD utilized by the General Population to be 16%. There is generally 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. Therefore, there is a reasonable certainty that no harm will result from aggregate exposure to esfenvalerate residues.

A Tier 3 acute dietary exposure assessment found the General Population to have MOE's of 1,803 at the 95th percentile of exposure and 676 at the 99th percentile of exposure. These values were generated using actual field trial residues and market share data for percentage of crop treated. These results depict an accurate exposure pattern at an exaggerated daily dietary exposure rate. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to esfenvalerate residues.

2. *Infants and children*. The chronic dietary assessment using the same assumptions described above, found the two most sensitive sub-populations to be non-nursing infants (<1 yr.) and children (1-6 yrs.) utilizing 44% and 48% of the RfD, respectively. In the Tier 3 acute dietary assessment that was rerun using the assumptions described above, non-nursing infants were found to have an MOE of 914 at the 95th percentile of exposure and an MOE of 254 at the 99th percentile. Children (1-6 yrs.) were determined to have an MOE of 698 at the 95th percentile and 321 at the 99th percentile. Therefore, there is a reasonable certainty that no harm will result from aggregate exposure to esfenvalerate residues.

F. International Tolerances

Codex maximum residue levels (MRL's) have been established for residues of fenvalerate on a number of crops that also have U.S. tolerances. Several of these MRL's are different than the proposed U.S. tolerances for esfenvalerate. Therefore, some harmonization of these maximum residue levels is still needed. (George LaRocca)

2. Elf Atochem North America, Inc.

PP 5F4550

EPA has received a pesticide petition (PP 5F4550) from Elf Atochem North America, Inc., 2000 Market Street, Philadelphia, PA 19103-3222, 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 Thiophanate-methyl in or on the raw agricultural commodities grapes at 5.0 parts per million (ppm) and pears at 7 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 of thiophanate-methyl (TM) in plants is well understood. Results of testing in wheat, lima beans, sugar beets and apples indicate that TM can be converted to methyl benzimidazole carbamate (MBC), allophanate (or FH-432), and DX-105 (sulfonated allophanate). TM, MBC, allophanate, and DX-105 are reflected in the tolerance as petitioned.

2. *Analytical method*. A proposed enforcement method for crop residue was submitted to the Agency in April 1996. The new method replaces the acid digestion method currently in widespread use. In contrast to the older method which involves acid hydrolysis of TM to MBC, the new method is capable of analyzing for TM directly and its three metabolites: MBC, allophanate, and DX-105. A proposed enforcement method for animal tissue will be submitted to the Agency in July 1997. The new method will entirely replace the current enforcement method.

3. *Magnitude of residues*—i. *Grapes*. Elf Atochem North America has conducted magnitude of the residue studies on grapes. The petition for the addition of a grape tolerance of 5 ppm was submitted June 16, 1995.

ii. *Pears*. Elf Atochem North America has conducted magnitude of the residue studies on pears. The petition for the addition of a pear tolerance of 7 ppm was submitted June 16, 1995.

B. Toxicological Profile

1. *Acute toxicity*. Technical thiophanate-methyl is practically non-toxic (Toxicity Category III) after administration by the oral, dermal and respiratory routes. Thiophanate-methyl is a skin sensitizer. Exposure to the technical product is not expected to occur to the general public or to infants or children.

2. *Genotoxicty*. Thiophanate-methyl has been extensively tested for genotoxicity and is not genotoxic. This further supports the threshold nature of the thyroid and liver effects. MBC has been tested in a wide range of genotoxicity assays. It is not a heritable gene mutagen. It does not interact with DNA, induce point mutations or result in germ cell mutations. Carbendazim (MBC) does cause numerical chromosome aberrations in experimental systems *in vitro* and *in vivo* as a result of interference with cellular tubulin rather than DNA.

3. *Reproductive and developmental toxicity*. Thiophanate-methyl induced no maternal effects and there were no teratogenic or fetotoxic effects in rats at any of the doses tested up to 2,500 ppm thiophanate-methyl. The maternal No Observable Effect Level, NOEL, is considered to be 250 ppm (12.5 mg/kg/day) based on body weight in the initial dosing phase of the study. The fetal NOEL was 2,500 ppm (125 mg/kg/day).

Thiophanate-methyl was also fed to pregnant rabbits at 0, 2, 6, and 20 mg/kg/day. The NOEL for maternal toxicity is tentatively defined as 6 mg/kg/day based on minimal body weight and food intake changes and the incidence of abortion/total litter loss. The NOEL for developmental effects is tentatively defined by EPA as 2 mg/kg/day. The Lowest Observable Effect Level, LOEL, was tentatively set at 6 mg/kg/day based on non-statistically significant dose-related increases in the incidence of asymmetric pelvis. These effects at the high dosage, 20 mg/kg/day, were well within historical control rates. This effect is not considered a harbinger of more significant findings at higher dosages. There was no evidence of any major teratogenicity. Based on this information, a NOEL of 6 mg/kg/day can be set for developmental effects.

In a 2-generation reproduction study, the thiophanate-methyl NOEL for systemic toxicity is <200 ppm based on hepatocellular hypertrophy/hyperplasia at all dose levels, decreased body weight gain in males, and increased liver and thyroid weights in both sexes at the highest dose tested. This LOEL is considered to be borderline NOEL/LOEL because the effects on the thyroid and liver at 2,100 ppm were minimal. The effects were less (fewer animals and less severe) in the succeeding generation. The NOEL is 200 ppm based on reduced body weights of the F2b pups during lactation at 630 ppm.

4. *Endocrine effects*. Thiophanate-methyl has been evaluated in both reproductive and developmental studies. No effects were observed that would indicate that the endocrine system is disrupted with regard to the reproductive system (i.e., anti-estrogenic, estrogenic, androgenic, anti-androgenic). TM does alter thyroid function through the thyroid stimulating hormone. This effect has been studied further and is documented in the rat chronic/oncogenicity study.

5. *Chronic toxicity*. Thiophanate-methyl was administered by capsule to beagle dogs for one year. Based on the decreased body weight gain in both sexes, decreased T4 levels in males and increased thyroid-to-body weight ratio and hypertrophic histologic changes in the thyroid gland in both sexes, the LOEL for thiophanate-methyl is 40 mg/kg/day and the NOEL is 8 mg/kg/day.

A combined chronic/oncogenicity feeding study was performed in rats at dosages of 0, 75, 200, 1,200 and 6,000 ppm TM for 2 years. No clinical signs attributable to TM were noted in the first 52 weeks. It was concluded that the effects of the treatment with TM included growth depression, anemia, morphological and functional changes in the thyroid and pituita, hepatocellular hypertrophy with lipofuscin, acceleratednephropathy andlipidosis of the adrenal cortex. The MTD was determined to be 1,200 ppm for both males and females. At 6,000 ppm, approximately five times the MTD, an increase in thyroid follicular cell adenomas was observed in males. Thyroid hyperplasia and hypertrophy were observed only at or above the MTD. These effects are considered to be related to the treatment related changes in hormonal homeostasis of the pituitary-thyroid axis. The NOEL is 200 ppm (8.8 mg/kg/day in males and 10.2 mg/kg/day in females) when fed for 104 weeks.

6. *Carcinogenicity*. Thiophanate-methyl was fed to male and female CD-1 mice for 18 months. At 3,000 ppm the males showed an increased incidence of hepatocellular hypertrophy and a small, but statistically significant, decrease in body weight (<8%). Transient increases in serum thyroid stimulating hormone (TSH) and in absolute and relative thyroid weights were also observed in males. At the highest dose tested (7,000 ppm) both males and females showed increased mortality and increased liver weight at both weeks 39 and 78. Females at 7,000 ppm showed a statistically significant decrease in body weight (<8%), decreased serum thyroxine (T4) at week 39, and increased heart weight at weeks 39 and 78. A dose-related statistically significant increase in the incidence of hepatocellular adenomas was observed in both sexes at 3,000 and 7,000 ppm. The systemic NOEL is 150 ppm (23.7 mg/kg/day in males and 28.7 mg/kg/day in females). The LOEL is 640 ppm based on an increased incidence of hepatocellular hypertrophy in females.

Mechanistic studies have been performed in rats and mice to elucidate the role of TM in the disruption of the thyroid. TSH, T3 and T4 are altered by TM treatment. The thyroid effects are alleviated by the addition of T4. The effects noted in both the rat, mouse and dog studies fit the threshold consideration category outlined by the Agency in the document ''Thyroid

Follicular Carcinogenesis: Mechanistic and Science Policy Considerations.''

7. *Animal metabolism*. The metabolism of thiophanate-methyl in animals is well understood. In animal studies in laying hens and lactating goats, some of the metabolites are subsequently hydroxylated. Thiophanate-methyl was also orally administered to male and female rats at dose levels of 10, 13, and 150 mg/kg. The absorption and excretion of the radioactivity was rapid. The maximum concentrations in blood were reached after about 1 to 3 hours in the two lower dose groups and in 4 to 7 hours in the higher. Less than 0.5% of the administered dose was associated with the rat's body. Among the tissues examined, the residue level of thiophanate-methyl equivalents was the highest in the thyroid and liver. About 70% of the radioactivity was quantitatively identified in the urine and feces as TM, 4-OH-TM, 5-OH-MBC and 5-OH-MBC-S (enzymatic hydrolysis from conjugated material).

8. *Metabolite toxicology*. There are three primary plant metabolites of thiophanate-methyl: MBC, allophanate, and DX105 (sulfonated allophanate). The toxic metabolite, MBC, is well understood and documented in the report of the International Programme on Chemical Safety (Environmental Health Criteria 149: Carbendazim, World Health Organization, 1993). MBC is marketed outside the U. S. under the trade name of Carbendazim.

The NOEL for MBC is 500 mg/kg/day in the rat chronic/oncogenicity and 300 mg/kg in the dog chronic studies. Three mouse oncogenicity studies were performed in three different strains of mice with mixed results. In CD-1 mice, MBC induced hepatocellular adenomas in females with a NOEL of 500 mg/kg/day. In SPF mice there was an increase in the incidence of combined hepatocellular adenomas and carcinomas. A study in NMRKf mice showed no carcinogenic effects up to a dose of 5,000 mg/kg/day. The rat oncogenicity study showed no carcinogenicity. The Agency has categorized MBC (carbendazim) as a C oncogen and assigned a Q* of 4.2×10^{-3}.

C. Aggregate Exposure

1. *Dietary exposure*. Dietary exposure is the primary route of exposure to TM.

2. *Drinking water*. Thiophanate-methyl is not expected to be found in water. The half-life of TM is very short in soil and water. When metabolized or chemically converted to MBC, none is expected to leave the soil. Little to no TM exposure is expected in drinking water. In the ''EPA Pesticide in Groundwater Database: A Compilation of Monitoring Studies: 1971-1991: National Summary'' no TM was detected. Based on the environmental fate data, TM or its metabolite MBC is not expected to leach into water systems. There are no uses of TM that are expected to impact water.

3. *Non-dietary exposure*. Thiophanate-methyl has turf use patterns which are primarily commercial (golf course, turf farms). Children are not primary users of golf courses and would have little opportunity for exposure as the result of this use pattern. Homeowner use is expected to be low. Based on sales figures use on lawns should not exceed 1%. Product is applied by commercial applicators. The dermal exposure studies showed no toxicity in a limit test at 2,000 mg/kg. The dermal absorption of thiophanate-methyl, and carbendazim is significantly lower than the oral route of exposure. The NOEL for a 21-day dermal exposure study in rats is 300 mg/kg/day and dermal irritation is 1,000 mg/kg/day dosage.

Based on the limited use of the product on lawns and the low dermal toxicity, little to no contribution to the TM risk cup is expected through non-occupational exposure.

D. Cumulative Effects

Benomyl, MBC, thiabendazole and TM have been evaluated for similar toxicity patterns because of the potential structure-activity relationship. TM, although displaying some similarities to each benzimidazole, is also very different. These benzimidazoles do not share a toxicity profile that would indicate there is common mode of action.

The toxic effects of TM are very different from those published on MBC or benomyl. TM toxicity primarily involves the thyroid. In contrast, no disruption of the thyroid-pituitary-liver axis is documented in either the carbendazim or the benomyl studies. Secondary effects on the liver could be seen in common, but these too are very different. If driven by MBC alone, TM should have a dose effect much higher than MBC. In fact, it is two to three times lower. Reproductive, developmental and genetic toxicity are also different between TM and MBC. Likewise, thiabendazole is different than TM. It does not metabolize to MBC and shows significant differences from TM in the type of toxicities observed. Therefore, there is no scientific basis for aggregating this class of fungicides, due to a lack of common mechanisms of toxicity.

E. Safety Determination

1. *U.S. population*. Assessments have been made for chronic, acute, and cancer risk. In all assessments, there is a reasonable certainty of no harm associated with TM residues on food.

2. *Non-cancer chronic dietary safety determination*. For chronic assessments other than cancer, the Reference Dose (RfD) is 0.08 mg/kg/day based on the results of the chronic dog study. Because the data base is complete, a 100-fold safety factor can be used. The maximum permitted intake (MPI) of TM for a 60 kg human is calculated to be 4.8 mg/day. The theoretical maximum residue contribution (TMRC) from existing tolerances for a 1.5 kg daily diet is calculated to be 0.24002 mg/day. Based on the Agency's calculations, this represents about 5% utilization of the MPI. The addition of grapes would add only 0.00000895 mg/kg/day and pears would add 0.00000512 mg/kg/day. Using anticipated residue rather than tolerances, the actual utilization of the MPI will be significantly lower.

3. *Acute dietary safety determination*. The acute dietary risk Tier 3 analysis has been performed using a Monte Carlo analysis. The NOEL used was from a developmental study in rabbits (6 mg/kg/day). For the total U.S. population, non-nursing infants, children aged 1 to 6, and women aged 13 to 50 all margins of exposure (MOE) exceeded 100 at any percentile evaluated. At the 95 percentile of per-capita days, the MOE for all uses including pending actions for the U.S. population is 3,468; for non-nursing infants the MOE is 1,123; for all infants the MOE is 1,260; children ages 1 to 6 it is 1,620; children ages 7 to 12 the MOE is 2,911 and for females 13 to 50 the MOE is 7,219. The highest exposed sub-population, non-nursing infants, had an MOE of 562 at the 99th percentile. There is an adequate acute dietary safety margin for all current and intended uses of TM.

4. *Cancer risk assessment*. Thiophanate-methyl is regulated based on the metabolite MBC with a designated Q* of 4.2×10^{-3} based on mouse liver tumors. The lifetime cancer dietary risk is calculated by summing all sources of MBC that would result from TM use. Residues measured as MBC on plants were added to the residues from TM that could be converted biologically upon ingestion to MBC. Residue values were averaged and adjusted for percent of the crop treated. The bio-conversion factor was 36.5% based on the rat metabolism study using the low dose preconditioned treatment. Using the USDA's Continued Survey of Food Intake by Individuals (CSFII) conducted

from 1989 through 1992 and field trial residue data, MBC exposure was calculated. This exposure multiplied by the cancer potency factor (Q*) generates the potential cancer risk attributable to MBC at the 95% confidence interval. Life-time cancer risk for the total U.S. population for all seasons is calculated to be 2.71 x 10^{-7}. With the addition of grape and pear uses the lifetime cancer risk is 2.89 x 10^{-7}. The most sensitive sub-population is non-hispanic other than black or white, with a cancer risk of 4.56 x 10^{-7}.

5. *Infants and children*. Based on the acute and chronic dietary assessments, there is reasonable certainty of no harm to children who consume food treated with TM. Potential exposure from water or non-occupational exposure is minimal. Inhalation and dermal exposure is unlikely. The acute MOEs for dietary ingestion are large.

The potential of TM to induce toxic effects in children at a greater sensitivity than the general population has been assessed by the rat and rabbit developmental and 2-generation reproduction studies. No major teratogenic or fetotoxic effects were present in the absence of maternal toxicity. The TM 2-generation reproduction study showed thyroid and liver effects in both the parental and first generation pups. The effects were greater in the parental animals than in subsequent generations. This would indicate that there is no greater sensitivity for neo-nates, infants and children to TM than the general population. The reproductive and developmental data base is complete. There is no need to impose an additional safety factor to protect infants and children. Based on the level of potential exposure and similar sensitivity to the adult population, infants and children are well protected by the current TM regulatory policy.

F. International Tolerances

The CODEX Maximum Residue Limits (MRL) for thiophanate-methyl are expressed as the metabolite MBC. The grape MRL is 10 mg/kg and the pear MRL is 5 mg/kg. (Mary Waller)

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–5871–7]

De Minimis Settlement Under Section 122(g) of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA), as amended, Peerless Industrial Paint Coatings Site, City of St. Louis, St. Louis County, Missouri; Notice of Request for Public Comment

AGENCY: Environmental Protection Agency.

ACTION: Notice of request for public comment.

SUMMARY: The Environmental Protection Agency (EPA) has entered into a *de minimis* administrative settlement to resolve claims under the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA), as amended, 42 U.S.C. 9622(g). The settlement is intended to resolve the liability of Westinghouse Electric Corporation (Westinghouse) for the response costs incurred and to be incurred at the Peerless Industrial Paint Coatings Site, City of St. Louis, St. Louis County, Missouri.

DATES: Written comments must be provided on or before September 8, 1997.

ADDRESSES: Comments should be addressed to the Regional Administrator, United States Environmental Protection Agency, Region VII, 726 Minnesota Avenue, Kansas City, Kansas 66101 and should refer to: In the Matter of the Peerless Industrial Paint Coatings Superfund Site, City of St. Louis, St. Louis County, Missouri, EPA Docket Nos. VII–97–F–0001.

FOR FURTHER INFORMATION CONTACT: Denise L. Roberts, Assistant Regional Counsel, United States Environmental Protection Agency, Region VII, 726 Minnesota Avenue, Kansas City, Kansas 66101, (913) 551–7559.

SUPPLEMENTARY INFORMATION: Westinghouse Electric Corporation (''Westinghouse'' or *de minimis* party''), the settling party, is a *de minimis* generator of hazardous substances found at the Peerless Industrial Paint Coatings Site, which is the subject Superfund Site. On April 21, 1997, Region VII entered into a *de minimis* administrative settlement to resolve claims under Section 122(g) of CERCLA, 42 U.S.C. 9622(g).

The Peerless Industrial Paint Coatings Site (the ''Site'') is located in St. Louis at 1265 Lewis Street, St. Louis, Missouri, approximately ¼ mile north of downtown St. Louis in an industrial section of the city. The *de minimis* party, Westinghouse, is a corporation that operated a facility in Manor, Pennsylvania from 1937 until July 1995 which manufactures and sells paints and resins to commercial customers. Westinghouse sold polyester resins and alkyds to Peerless Industrial Paint Coatings (''Peerless''), a St. Louis corporation, at very low prices. Westinghouse admitted that it sold secondary coatings or materials to Peerless at very low prices, which were less than the costs of disposal for hazardous wastes at an authorized permitted facility. Peerless was a manufacturer of paints and magazine coatings that purchased large quantities of paint materials at low prices and accumulated more materials on-site than could be used. In June 1993, the EPA began a removal action at the site. Approximately 3500 drums of hazardous substances that demonstrated the characteristic of ignitability were removed from the facility at the cost of $1,089,062.71.

The settlement has been approved by the U.S. Department of Justice because the response costs in this matter exceed $500,000.00. The EPA estimates the total past and future costs will be approximately $1,342,357.05. Pursuant to the Administrative Order on Consent, the *de minimis* party is responsible for its attributable share of 1.71 percent of the hazardous substances removed from the Site. Westinghouse had agreed to pay a total of $27,920.07 which is further detailed as follows: $17,720.07 is its attributable share of past costs, $5,100.00 is its attributable share of anticipated future costs; and $5,100.00 is a premium of 100% for future cost overruns. The EPA determined these amounts to be the *de minimis* party's fair share of liability based on the amount of hazardous substances found at the Site and contributed by the settling party. The settlement includes contribution protection from lawsuits by other potentially responsible parties as provided for under section 122(g)(5) of CERCLA, 42 U.S.C. 9622(g)(5).

The *de minimis* settlement provides that the EPA covenants not to sue the *de minimis* party for response costs at the Site or for injunctive relief pursuant to Sections 106 and 107 of CERCLA and section 7003 of the Resource Conservation and Recovery Act of 1980, as amended (RCRA), 42 U.S.C. 6973. The settlement contains a reopener clause which nullifies the covenant not to sue if any information becomes known to the EPA that indicates that the parties no longer meet the criteria for a