post-natal effects is complete. Further for imidacloprid, the NOEL of 5.7 mg/kg/bwt from the 2–year rat feeding/carcinogenic study, which was used to calculate the RfD (discussed above), is already lower than the NOELs from the developmental studies in rats and rabbits by a factor of 4.2 to 17.5 times. Since a hundredfold 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 percent 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 percent 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 percent of the RfD. The TMRC for children ages 1 to 6 years is 0.000130 mg/kg/bwt/day, which represents 0.2 percent of the RfD. For nursing infants, the TMRC is 0.000032 mg/kg/bwt/day, which is 0.1 percent of the RfD. For children ages 7 to 12 years, the TMRC is 0.000098 mg/kg/bwt/day, which is 0.2 percent 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. Other Considerations

The nature of the imidacloprid residue in plants and livestock is adequately understood. The residues of concern are combined residues of imidacloprid and its metabolites containing the 6-chloropyridinyl moiety, all calculated as imidacloprid. The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloropyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary GC-MS selective ion monitoring. There is an additional confirmatory method available. Imidacloprid and its metabolites have been shown to be stable for at least 24 months in frozen storage.

G. International Tolerances

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

[FR Doc. 97–16655 Filed 6–24–97; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–739; FRL–5721–7]

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–739, must be received on or before July 25, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), 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 regulatory action leaders listed in the table below:

Product Manager	Office location/telephone number	Address
Sheryl Reilly (PM 90)	Rm. 5-W29, 5th Floor, CS-1, 703-308-8265 e-mail: reilly.sheryl@epamail.epa.gov	2800 Jefferson Davis Hwy., Arlington, VA 22202
Mike Mendelsohn (PM 90).	Rm. 5-W44, 5th Floor, CS-1, 703-308-8715 e-mail: mendelsohn.mike@epamail.epa.gov	Do.
Linda Hollis (PM 90)	Rm 5-J, 5th Floor, CS-1, 703-308-8733 e-mail: hollis.linda@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–739] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:
opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in

Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number (insert docket number) and appropriate petition number. Electronic comments on 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: June 12, 1997.

Janet L. Andersen,

Director, Biopesticides and Pollution Prevention 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. Kemira Agro Oy

PP 7F4137

A. Proposed Use Practices

Registration of PRIMASTOP containing *Gliocladium catenulatum* Strain J1446 is being proposed for the following sites: Vegetables, herbs and spices, ornamentals, tree and shrub seedlings, turf, home and garden.

PRIMASTOP is used for the control of damping-off, seed rot, root and stem rot, and wilt diseases caused by *Rhizoctonia, Pythium, Phytophthora, Fusarium, Didymella, Botrytis, Verticillium, Alternaria, Cladosporium, Helminthosporium, Penicillium and Plicaria* on vegetables, herbs, ornamentals and tree and forest seedlings grown in greenhouse or outdoors.

PRIMASTOP can be incorporated in the growth substrate as a dry powder or as an aqueous suspension or applied by drenching, spraying or dipping.

1. *Incorporation into potting media.* The recommended rate for incorporation of PRIMASTOP in potting media is 5 to 30 oz/yd^3 (0.2 to 1 g/L) of growing media. If the incorporation is done with the aqueous suspension, mix 3.5 oz (100 g) of PRIMASTOP powder in 1.0 gallon (or 4 L) of water and carefully mix the suspension with the growth substrate (1.5-8.5 gal/yd^3). Incorporation of PRIMASTOP can be followed with a drench application within 2 to 6 weeks.

2. *Drench application.* Drenching treatment can be done using a 0.2-0.5% suspension. Seedling trays or beds can be drenched with PRIMASTOP at the recommended rate of 2 to 10 oz./100 ft^2 (5-25 g/m^2). Drenching at sowing is recommended for vegetables (except for tomato and leek), herbs and ornamentals (except pansy) grown in peat or soil mixture. Drenching after emergence is recommended for tomato, leek, pansy and all seedlings grown in rockwool, such as cucumbers. Repeat treatment at transplanting.

3. *Foliar spray.* PRIMASTOP can be sprayed or spread on the plant stems or foliar parts for control of Didymella or Botrytis with an aqueous suspension. Recommended concentration is up to 5%.

4. *Treatment of cuttings, bulbs or tubers.* Cuttings, bulbs or tubers can be dipped in or sprayed with PRIMASTOP suspensionn before planting or storage. The product can also be incorporated in the storage mixture, such as sand or peat at a rate up to 1 g/L.

B. Product Identity/Chemistry

1. *Identity of pesticide and corresponding residues.* The active ingredient in Primastop is *Gliocladium catenulatum* Strain J1446. The mechanism by which *Gliocladium catenulatum* Strain J1446 controls diseases appears to be enzymatic. *Gliocladium catenulatum* Strain J1446 does not produce toxins or antibiotics. Further, *Gliocladium catenulatum* Strain J1446 is a naturally occurring microorganism. *Gliocladium catenulatum* is widespread in the environment.

2. *Magnitude of residue anticipated at the time of harvest and method used to determine the residue.* No residues of *Gliocladium catenulatum* Strain J1446 are anticipated in treated crops at harvest. Subdivision M - Series 153A-3(a) indicates that ''if Tier I toxicology tests indicate no toxic or other harmful properties, then no residue data would be indicated.'' Studies with *Gliocladium catenulatum* Strain J1446 demonstrated low mammalian toxicity. No pathogenicity or infectivity was observed in any of the tests conducted with *Gliocladium catenulatum* Strain J1446. Further, *Gliocladium catenulatum* Strain J1446 is a naturally occurring microorganism. *Gliocladium catenulatum* is widespread in the environment.

3. *Statement of why an analytical method for detecting and measuring the levels of the pesticide residue are not needed.* Subdivision M - Series 153A-3(a) indicates that ''if Tier I toxicology tests indicate no toxic or other harmful properties , then no residue data would be indicated and thus a recommendation for an exemption from the requirement of a tolerance can be made.'' Studies with *Gliocladium catenulatum* Strain J1446 demonstrated low mammalian toxicity. No pathogenicity or infectivity was observed in any of the tests conducted with *Gliocladium catenulatum* Strain J1446. Further, *Gliocladium catenulatum* Strain J1446 is a naturally occurring microorganism. *Gliocladium catenulatum* is widespread in the environment.

C. Health and Safety

Kemira Agro Oy conducted the required toxicology studies to support its petition for an exemption from the requirement of tolerance and associated registrations. The studies conducted indicate a low mammalian toxicity for *Gliocladium catenulatum* Strain J1446. No pathogenicity or infectivity was observed in any of the tests conducted with *Gliocladium catenulatum* Strain J1446. The mechanism by which *Gliocladium catenulatum* Strain J1446 controls diseases appears to be enzymatic. *Gliocladium catenulatum* Strain J1446 does not produce toxins or antibiotics.

Toxicology data in support of the exemption from the requirement of a tolerance for *Gliocladium catenulatum* Strain J1446 included studies with the cell mass (technical) and with the formulated product as follows:

1. *Acute toxicity and/or pathogenicity.— a. Gliocladium catenulatum Strain J1446 Cell Mass (Technical).* i. Acute oral toxicity and pathogenicity in rats (acute oral LD_{50} > 4.04 to 5.86 × 108 cfu/kg, clearance: < 3 days).

ii. Acute pulmonary toxicity/ pathogenicity in rats (acute pulmonary LC_{50} > 6.60 to 7.98 × 108 cfu/kg, clearance: < 7 days).

iii. Acute intraperitoneal toxicity/ pathogenicity in rats (acute intraperitoneal LD_{50} > 4.2 × 108 cfu/kg, clearance: < 7 days).

b. *Gliocladium catenulatum Strain J1446 Formulation (Primastop).* i. Acute oral LD_{50} toxicity in rats (> 2,000 mg/kg, EPA toxicity category III).

ii. Acute dermal LD_{50} toxicity in rats (>2,000 mg/kg, EPA toxicity category III).

iii. Acute dermal irritation in rabbits (minimal dermal irritant, EPA toxicity category IV).

iv. Acute inhalation LC_{50} toxicity in rats (> 5.57 mg/L, EPA toxicity category V).

v. Primary eye irritation (minimal eye irritant, EPA toxicity category IV).

vi. Skin sensitization (sensitizer).

vii. No hypersensitivity effects have been observed.

c. The inert ingredients contained in the *Gliocladium catenulatum* Strain J1446 formulation, Primastop, are all minimal risk (List 4)(59 FR 49400, September 28, 1994).

2. *Genotoxicity*. Subdivision M Guidelines do not require the conduct of genotoxicity studies to support the registration of a microbial pest control agent, such as *Gliocladium catenulatum* Strain J1446.

3. *Reproductive and developmental toxicity.* Subdivision M Guidelines do not require the conduct of reproductive and developmental toxicity studies to support the registration of a microbial pest control agent, such as *Gliocladium catenulatum* Strain J1446.

4. *Subchronic toxicity.* Subdivision M Guidelines do not require the conduct of subchronic toxicity studies to support the registration of a microbial pest control agent, such as *Gliocladium catenulatum* Strain J1446.

5. *Chronic toxicity.* Subdivision M Guidelines do not require the conduct of chronic toxicity studies to support the registration of a microbial pest control agent, such as *Gliocladium catenulatum* Strain J1446.

Sufficient data exist to assess the hazards of *Gliocladium catenulatum* Strain J1446 and to make a determination on aggregate exposure, consistent with section 408(c)(2), for the exemptions from the requirement of a tolerance. The exposures, including dietary exposure, and risks associated with establishing the requested exemption from the requirement of a tolerance follows.

D. Threshold Effects

Gliocladium catenulatum is a naturally occurring microorganism that is widespread in the environment. Both the cell mass (technical) and the formulation of *Gliocladium catenulatum* Strain J1446 demonstrated low toxicity. No pathogenicity or infectivity was observed in any of the tests conducted with *Gliocladium catenulatum* Strain J1446. No threshold effects were observed or are anticipated for *Gliocladium catenulatum* Strain J1446.

E. Non-threshold Effects

Gliocladium catenulatum is a naturally occurring microorganism that is widespread in the environment. Both the cell mass (technical) and formulation of *Gliocladium catenulatum* Strain J1446 demonstrated low toxicity. No pathogenicity or infectivity was observed in any of the tests conducted with *Gliocladium catenulatum* Strain J1446. Non-threshold effects were not observed nor are any anticipated for *Gliocladium catenulatum* Strain J1446.

F. Aggregate Exposure

Gliocladium catenulatum is naturally occurring and widespread in the environment. The low toxicity and non-pathogenicity/infectivity of *Gliocladium catenulatum* Strain J1446 is demonstrated by the data summarized above. The product will be applied by incorporation into growing media and/or by drenching at seeding or in the early growing stages of the treated plants.

1. *Dietary exposure*— a. *food*. It is not anticipated that residues of *Gliocladium catenulatum* Strain J1446 will occur in treated raw agricultural commodities.

b. *Drinking water*. It is not anticipated that residues of *Gliocladium catenulatum* Strain J1446 will occur in drinking water.

2. *Non-dietary exposure.* The potential for non-occupational, non-dietary exposure to the general population is not expected to be significant.

G. Cumulative Exposure

There is no anticipated potential for cumulative effects of *Gliocladium catenulatum* Strain J1446 and other substances that have a common mechanism of toxicity. Clearance of *Gliocladium catenulatum* Strain J1446 from test species was < 3 days in two studies and < 7 days in a third study. Toxic effects produced by *Gliocladium catenulatum* Strain J1446 should not be cumulative with those of any other chemical compounds.

H. Determination of Safety for U.S. Population

Gliocladium catenulatum Strain J1446 is a naturally occurring microorganism. *Gliocladium catenulatum* is widespread in the environment. The low toxicity of *Gliocladium catenulatum* Strain J1446 is demonstrated by the data summarized above. Based on this information, the aggregate exposure to *Gliocladium catenulatum* Strain J1446 over a lifetime should not pose appreciable risks to human health. There is a reasonable certainty that no harm will result from aggregate exposure to *Gliocladium catenulatum* Strain J1446 residues. Exempting *Gliocladium catenulatum* Strain J1446 from the requirement of a tolerance should be considered safe and pose insignificant risk.

I. Determination of Safety for Infants and Children

The toxicity and exposure data are sufficiently complete to adequately address the potential for additional sensitivity of infants and children to residues of *Gliocladium catenulatum* Strain J1446. There is a reasonable certainty that no harm will result to infants and children from aggregate exposure to *Gliocladium catenulatum* Strain J1446 residues.

J. Estrogenic Effects

No specific tests have been conducted with *Gliocladium catenulatum* Strain J1446 to determine whether it may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, it is not likely that *Gliocladium catenulatum* strain J1446 would have estrogen or endocrine effects because:

1. It is a naturally occurring microorganism. *Gliocladium catenulatum* is widespread in the environment.

2. It has demonstrated low mammalian toxicity.

3. No pathogenicity or infectivity was observed in any of the tests conducted with *Gliocladium catenulatum* Strain J1446.

4. The mechanism by which *Gliocladium catenulatum* Strain J1446 controls diseases appears to be enzymatic.

5. *Gliocladium catenulatum* Strain J1446 does not produce toxins or antibiotics.

K. Existing Tolerances

No tolerances or exemptions from the requirement of tolerance have been established or applied for domestically or internationally other that subject petition.

L. Environmental Fate

Environmental fate data are not required to support the registration of a biopesticide unless results from Tier I studies indicate that risks would be expected from use of the product. *Gliocladium catenulatum* GStrain J1446 is a naturally occurring microorganism. *Gliocladium catenulatum* is widespread in the environment. Extensive literature searches revealed an absence of any ecological effects or environmental fate

data from *Gliocladium catenulatum*. (Sheryl Reilly)

2. Monsanto Company

PP 6E4657

A. Background Information and Use Profile

The development of plant varieties containing useful new traits introduced by plant genetic engineering such as insect protection depends upon an effective means to select for the rare transformed plant cells containing the added gene. For example, a gene required for the production of an insecticidal protein in the plant tissue cannot be efficiently selected for several weeks after the transformation event as it does not, itself, provide a readily selectable property to the cell which carries it. Regenerating each cell from that transformation experiment to test for the presence of the gene would be both impractical and prohibitory, as the frequency that transformed cells are obtained is often as low as 1 in 10,000 or 1 in 100,000 of the cells treated (Fraley et al., 1984). Therefore, a selectable marker gene and a selective agent are used to identify the rare transformed cells for regeneration.

A selectable marker gene allows a cell expressing that marker gene to grow in the presence of a selective agent by inactivating or neutralizing the agent which would otherwise inhibit the growth or kill the cell. The marker gene also permits tracking of linked traits that are difficult to identify at the cellular or whole plant level.

For insect-protected corn plants, the gox gene was used as a selectable marker gene conferring tolerance to glyphosate. The glyphosate oxidoreductase (gox) gene from Achromobacter sp. strain LBAA (new genus *Ochrobactrum anthropi*) produces a protein (GOX) which degrades glyphosate. The GOX protein confers tolerance to glyphosate and provides a selectable marker used in initial laboratory stages of plant cell selection to identify plant cells containing agronomic genes of interest such as the cryIA(b) gene which imparts protection from certain lepidopteran insect pests.

B. Risk Assessment and Statutory Findings Toxicology Profile

1. *Data summary.* Monsanto Company has requested an exemption from the requirement of a tolerance for glyphosate oxidoreductase (GOX) as a plant pesticide formulation inert ingredient. Included in the Monsanto submission to the EPA were several toxicology studies in support of the GOX protein as a pesticide formulation inert ingredient.

The GOX protein used in these studies was produced in an *E. coli* over-expression system and partially purified. The GOX protein expressed in *E. coli* was characterized and shown to be substantially equivalent to the GOX expressed in insect-protected corn where it was utilized as a selectable marker protein.

The following mammalian toxicity studies have been conducted to support this exemption from the requirement of a tolerance:

a. A mouse acute oral gavage study was performed in which the No-Observed-Effect-Level (NOEL) for toxicity of GOX protein administered as a single dose was considered to be 100 milligrams per kilogram (mg/kg) (the highest tested target dose). The protein was administered by gavage to three groups of male and female mice at target dose levels of 1, 10, and 100 mg/kg body weight. Appropriate hollow vector and vehicle controls were used. Mice were observed twice daily for signs of toxicity and food consumption was recorded daily. Food and water were provided ad libitum. All animals were sacrificed on post-dosing day seven and subjected to gross necropsy. Approximately 40 tissues were collected and saved for each animal in the test. There were no statistically significant differences in body weight, cumulative body weight or food consumption between the controls or GOX protein treated groups. No grossly observable pathologic changes were observed in mice at necropsy that were related to treatment.

When proteins are toxic, they are known to act via acute mechanisms and at very low dose levels (Sjoblad, et al., 1992). The acute oral toxicity data submitted support the prediction that the GOX protein is non-toxic to humans.

b. An *in vitro* digestive fate study of the GOX protein in simulated gastric and intestinal fluids demonstrated rapid protein degradation. In gastric fluid, the GOX protein degraded extremely rapidly; more than 90% of the initially added GOX protein degraded after 15 seconds incubation as detected by western blot analysis. GOX enzymatic activity also dissipated readily; more than 96% of the added GOX activity dissipated after one minute incubation, the earliest time point measured.

In intestinal fluid, GOX protein degraded rapidly; more than 90% of the initially added GOX protein degraded after 30 seconds incubation as detected by western blot analysis. GOX enzymatic activity also dissipated readily; more than 95% of the added GOX enzymatic activity dissipated after 60 minute incubation. The difference in dissipation of the enzymatic activity of GOX when compared to detection by western blot analysis suggests the antigenic sites on the GOX protein for the particular antibody used in this study were more sensitive to proteolytic degradation in simulated intestinal fluid under these conditions than is the functional active site of the GOX protein. The GOX protein degraded readily, though, as assessed by both western blot analysis and enzymatic activity.

The results of this study established that the GOX protein and its associated functional activity will be efficiently degraded upon exposure to gastric and intestinal fluids in the mammalian digestive tract. Known protein allergens are often resistant to digestion.

c. A homology assessment of the amino acid sequence of the GOX protein has been performed comparing this protein to known allergens or gliadin proteins. Monsanto has searched the amino acid sequences of the 219 allergens present in public domain genetic databases (GenBank, EMBL, PIR, and SwissProt) for similarity to the amino acid sequences of the GOX protein using the FASTA computer program (Pearson and Lipman, 1988). Monsanto concludes (i) that the gox gene introduced does not encode a known allergen, and (ii) that the introduced GOX protein does not share immunologically significant sequences with known allergens.

The GOX protein is produced at low levels (ppm) by insect-protected corn plants and is contained within the cells of the corn plant. In documentation provided to the Agency, the range of GOX protein levels in insect-protected corn line samples as assessed using a validated ELISA specific to the GOX protein ranged from < 1.8 to 19.32 µg/g fresh weight (fwt) in leaf tissue, < 1.5 to 11.7 µg/g fwt in grain, and < 0.6 to 2.46 µg/g fwt in whole plant tissue. Western blot analysis indicated that the GOX protein was not present in corn pollen.

The genetic material encoding the GOX protein and the regulatory regions associated with the gene have been well characterized. Nucleic acids (DNA) is common to all forms of plant and animal life and there are no known instances of toxic effects related to their consumption. No mammalian toxicity is expected from dietary exposure to the genetic material necessary for the production of the GOX protein in corn.

In summary, the safety of the GOX protein to mammals, including humans, was confirmed by demonstrating the rapid degradation of the GOX protein

under conditions which simulate mammalian digestion, by establishing the lack of toxicity to rodents in an acute gavage study and by establishing the lack of allergenic concerns.

2. *Acute toxicity.* An acute mouse gavage study with GOX protein was performed to directly assess potential acute toxicity associated with this protein. No adverse effects were observed in mice dosed with GOX protein. Based on this study, in which the No-Observed-Effect-Level (NOEL) for toxicity of the protein administered as a single dose was considered to be 100 mg/kg (the highest tested target dose), no acute dietary risks are posed for infants, children or adults.

3. *Developmental/reproductive effects.* No instances of reported adverse reproductive or developmental effects to humans, domestic animals or wildlife as a result of exposure to the GOX protein or the microbial source of the gox gene, Achromobacter, are known. Enzyme proteins have not been reported in literature to produce teratogenic effects or reproductive deficiency when fed to animals or man (Pareza and Foster, 1983).

4. *Chronic effects.* In an *in-vitro* digestive fate study, the GOX protein was rapidly degraded in simulated gastric and intestinal fluid with more than 90% of the initially added GOX protein degraded after 15 and 30 seconds incubation, respectively, as detected by western blot analysis. Consequently, no chronic effects are expected for infants, children or adults.

5. *Carcinogenicity.* Protein enzymes are not considered to be carcinogenic (Pareza and Foster, 1983) and consequently, there is no carcinogenic risk associated with the GOX protein for infants, children or adults.

6. *Endocrine effects.* Not applicable. Enzyme proteins are not known to interact or bind directly with the estrogen receptor to produce endocrine effects. Further, there is little opportunity for systematic absorption of the GOX protein due to rapid degradation by digestive enzymes. Therefore, no adverse effects to the endocrine system is known.

C. Aggregate Exposure

1. *Dietary exposure.* Oral exposure to the GOX protein at very low levels may occur from ingestion of processed corn products; however, the lack of mammalian toxicity and the digestibility of the protein have been demonstrated as cited above.

2. *Drinking water exposure.* Transfer of the GOX protein to drinking water is highly unlikely given containment of the protein in plant cells and natural degradation upon plant senescence. Western blot analysis has indicated that the GOX protein was not present in corn pollen.

3. *Non-occupational exposure.* The GOX protein is produced at low levels as a selectable marker and is contained within the cells of the plant. Consequently, negligible exposure to the protein is expected from handling corn seed, leaf or other plant tissue at planting, during growth, or at harvest. In addition, negligible exposure to the GOX protein would be expected during storage, transportation, or disposal of insect-protected corn seed as the protein cannot drift or volatilize from the plant and its bioactivity is rapidly lost upon decomposition of the plant tissue.

D. Cumulative Risk

The GOX enzyme was isolated from an Achromobacter species and catyalyzes the degradation of glyphosate to aminomethylphosphonic acid (AMPA) and glyoxylate. This conversion of glyphosate to AMPA and glyoxylate is the primary route for the degradation of glyphosate. This degradation inactivates the herbicide and allows the plant cell expressing the GOX protein to grow in the presence of glyphosate. This mechanism is not shared by other known selectable markers used in initial laboratory stages of plant cell selection. Consideration of a common mode of toxicity is not appropriate given that there is no indication of mammalian toxicity of the GOX protein and no information that indicates that toxic effects would be cumulative with any other compounds.

E. Safety Determinations

1. *U.S. general population.* The toxicity profile for the GOX protein indicates no risk from exposure to the GOX protein by the overall U.S. population. Monsanto believes that the lack of acute toxicity, rapid digestibility of the GOX protein and lack of homology to known proteinaceous allergens or toxins provide evidence for the lack of toxicity and allergenicity and support an exemption from the requirement of a tolerance for the GOX protein.

2. *Infants and children.* Monsanto considers the acute toxicity data, the rapid degradation of the GOX protein in the mammalian digestive system and the lack of homology to known proteinaceous allergens as evidence to support the safety of this protein to infants and children. Based upon this evidence, it is not expected that infants and children would be more susceptible to this protein than is the adult population.

F. Residue Chemistry Data Summary

As a plant pesticide formulation inert ingredient, the gox gene was used to produce the GOX protein which confers tolerance to glyphosate as the selectable marker. The GOX protein is produced in plant tissues including grain and forage at low levels as documented above. Mammalian safety of the protein has been demonstrated in acute oral toxicity test of the GOX protein. Analytical methods for the detection and measurement of the GOX protein are not needed as Monsanto is petitioning for an exemption from the requirement of a tolerance on the basis of mammalian safety. The GOX protein is not on the Food and Drug Administration's Generally Recognized as Safe (GRAS) list.

G. Environmental Fate Data Summary

The GOX gene was cloned from an Achromobacter species, reported to be one of the most frequently occuring bacteria in the rhizosphere (Joos et al., 1988). The GOX protein is produced at low levels within the cells of the plant and expected to degrade at plant senescence and exposure to physical, chemical and microbial processes in the environment. (Mike Mendelsohn)

3. Seminis

PP 4E4310

A. Proposed Use Practices

Recommended application method and rate(s), frequency of application, and timing of application. The inserted genes are under the control of a constitutive promoter. Therefore, the viral coat proteins will be produced within the tissues of the genetically engineered plant and will not be applied externally. In information provided to commercial growers, the resistance of the engineered plants to specific plant viruses will be described. However, Seminis states that no special instructions for use will be necessary. Appropriate cultural practices for growing seed with genetically engineered virus resistance will be determined by individual growers, as such practices are for all other plant varieties.

B. Product Identity/Chemistry

1. *Identity of the pesticide and corresponding residues.* The pesticide consists of a pair of viral coat proteins that are produced by the genetically engineered plant. One protein consists of a fusion of 16 amino acids of the cucumber mosaic virus coat protein and 281 amino acids of the watermelon mosaic virus 2 coat protein. The molecular weight of the chimeric coat

protein is approximately 33,203. The second protein consists of 279 amino acids of the zucchini yellow mosaic virus coat protein with a molecular weight of approximately 31,458.

2. *Magnitude of residue anticipated at the time of harvest and method used to determine the residue.* The viral coat proteins are expressed in plant tissues and, therefore, are not residues in the same manner as a pesticide applied externally to growing crop plants. Seminis believes that little concern exists for the presence of viral coat proteins remaining on or in genetically engineered plants as they are ubiquitous in nature, found in soil, water, terrestrial plants and raw produce.

3. *A statement of why an analytical method for detecting and measuring the levels of the pesticide residue are not needed.* The Enzyme-Linked Immunoabsorbent Assay (ELISA) test can be used to determine expression levels of viral coat proteins in transformed plants, fruits and leaves. However, the available scientific literature indicates that viral coat proteins do not pose a threat to human health or the environment at any level. Therefore, Seminis states that an analytical method for detecting and measuring the level of engineered viral coat proteins is not needed.

C. Mammalian Toxicological Profile

Viral coat proteins are substances that viruses produce during a plant infection to encapsulate and protect their genetic material. When the genetic material encoding the coat protein for a plant virus is introduced into a plant's genome, the plant is able to resist subsequent infections by that same virus as well as strains closely related to the donor virus. Virus-infected plants currently are and have been a part of both the human and domestic animal food supply, and Seminis agrees with EPA's finding that plant viruses are not known to be harmful to humans (59 FR 60519-60535, November 23, 1994)(FRL–4755–3). All available data from the scientific literature indicates that plant viruses are not toxic to humans or other vertebrates. Additionally, plant viruses are unable to replicate in mammals or other vertebrates, eliminating the possibility of human infection. This has been shown by injections of purified whole virus into laboratory animals to develop antibodies for ELISA tests.

More importantly, however, this tolerance exemption will apply to that portion of the viral genome coding for the whole coat protein and any sub-component of the coat protein expressed in the plant. This coat protein component alone is incapable of forming infectious particles. Because whole intact plant viruses are not known to cause deleterious human health effects, Seminis believes that it is reasonable to assume that a subunit of these viruses likewise will not cause adverse human health effects.

D. Aggregate Exposure

1. *Dietary exposure.* a. *Food.* Seminis states that the use of viral coat protein-mediated resistance will not result in significant new dietary exposure to plant viruses. Entire infectious particles of zucchini yellow mosaic virus and watermelon mosaic virus, including the coat protein component, are already found in the fruit and tissues of many plants. Virus-infected food plants are and have been a part of the human and domestic animal food supply. Such food plants and food derived from them have been consumed, including by children and infants, with no detectable or observed adverse effects to human health.

b. *Drinking water.* Seminis states that the use of viral coat protein-mediated resistance will not result in significant new levels of viral coat proteins from engineered plants in drinking water. Plant viruses are already present in soil and water. Viral coat proteins expressed in genetically engineered plants are limited to plant tissues. Upon plant senescence, viral coat proteins are believed to degrade in the soil in the same manner as other proteins. Consequently, Seminis believes that viral coat proteins produced as plant-pesticides would represent a negligible addition to those existing in drinking water.

2. *Non-dietary exposure (lawn care, topical insect repellents, etc.).* The use of the genetically engineered viral coat proteins is for improved virus disease resistance in agricultural crops. Therefore, Seminis believes that non-dietary exposure to genetically engineered viral coat proteins will be minimal to non-existent.

E. Cumulative Exposure

Exposure through other pesticides and substances with the common mode of toxicity as this pesticide. Seminis believes that due to the lack of toxicity associated with plant viruses and plant viral coat proteins, cumulative effects with other pesticides and substances will be non-existent.

F. Safety Determination

1. *U.S. population.* There is no known toxicity associated with coat proteins from plant viruses. Consequently, a safety assessment is not needed for these proteins. Given the long history of mammalian consumption of the entire plant virus particle in foods, without any adverse human health effects, Seminis reasonably believes that consumption of a non-infectious component of the WMV plant virus is safe. There are no known data that indicate aggregate exposure to plant viral coat proteins under normal conditions will result in harm to any person.

2. *Infants and children.* Viral coat proteins are ubiquitous in foods, including those foods consumed by infants and children. Moreover, there is not reason to believe that plant viral coat proteins are likely to occur in different amounts in foods consumed by children and infants. Further, there is no scientific evidence that viral coat proteins used as plant pesticides would have a different effect on children than on adults. Viral coat proteins are not toxic and, therefore, Seminis believes with reasonable certainty that no harm will result to Infants and Children from aggregate exposure to coat proteins from plant viruses.

G. Existing Tolerances

An exemption from tolerance was granted for watermelon mosaic virus-2 and zucchini yellow mosaic virus coat proteins as expressed in Asgrow line ZW20 of *Cucurbita pepo L.* in November 1994 (59 FR 54824, November 2, 1994)(FRL–4908–1).

H. International Tolerance

No known international tolerance or exemption from tolerance has been granted for watermelon mosaic virus-2 and zucchini yellow mosaic virus coat proteins. Seminis Vegetable Seeds, Inc. concludes that plant viruses, including watermelon mosaic virus-2 and zucchini yellow mosaic virus coat proteins, are not harmful to humans, and that there is a reasonable certainty that no harm will result from aggregate exposure to coat proteins of watermelon mosaic virus-2 and zucchini yellow mosaic virus, and the genetic material necessary for production, including all anticipated dietary exposures and all other non-occupational exposures. Accordingly, Seminis believes that watermelon mosaic virus-2 and zucchini yellow mosaic virus coat proteins qualify for an exemption from the requirement of a tolerance in or on all raw agricultural commodities. (Linda Hollis)

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