Executive Order 12915, entitled ''Federal Implementation of the North American Agreement on Environmental Cooperation.'' The Committee is responsible for providing advice to the U.S. Representative on implementation and further elaboration of the agreement.

The Committee consists of 12 independent representatives drawn from among environmental groups, business and industry, public policy organizations and educational institutions.

DATES: The Committee will meet on Thursday, January 27, 2000 from 9 a.m. until 5:30 p.m., and on Friday, January 28, 2000 from 8:30 a.m. to 3 p.m.

ADDRESSES: The Riverwalk Plaza Hotel, 100 Villita Street, San Antonio, Texas. The meeting is open to the public, with limited seating on a first-come, first-served basis.

FOR FURTHER INFORMATION CONTACT: Mr. Mark Joyce, Designated Federal Officer, U.S. EPA, Office of Cooperative Environmental Management, at (202) 564–9802.

Dated: December 20, 1999.

Sonia Altieri,

Acting Designated Federal Officer National Advisory Committee.

[FR Doc. 00–357 Filed 1–7–00; 8:45 am]

BILLING CODE 6560–50–M

ENVIRONMENTAL PROTECTION AGENCY

[FRL–6520–6]

Governmental Advisory Committee to the U.S. Representative to the Commission for Environmental Cooperation

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: Pursuant to the Federal Advisory Committee Act (Public Law 92–463), the U.S. Environmental Protection Agency (EPA) gives notice of a meeting of the Governmental Advisory Committee (NAC) to the U.S. Government Representative to the Commission for Environmental Cooperation (CEC).

The Committee is established within the U.S. Environmental Protection Agency (EPA) to advise the Administrator of the EPA in her capacity as the U.S. Representative to the CEC. The Committee is authorized under Article 18 of the North American Agreement on Environmental Cooperation, and the North American Free Trade Agreement Implementation Act (NAFTA), Public Law 103–182. Federal government responsibilities relating to the committee are set forth in Executive Order 12915, entitled ''Federal Implementation of the North American Agreement on Environmental Cooperation.'' The Committee is responsible for providing advice to the U.S. Representative on implementation and further elaboration of the agreement.

The Committee consists of 12 independent representatives drawn from state, local and tribal governments.

DATES: The Committee will meet on Thursday, January 27, 2000 from 9 a.m. until 5:30 p.m., and on Friday, January 28, 2000 from 8:30 a.m. to 3 p.m.

ADDRESSES: The Riverwalk Plaza Hotel, 100 Villita Street, San Antonio, Texas. The meeting is open to the public, with limited seating on a first-come, first-served basis.

FOR FURTHER INFORMATION CONTACT: Ms. Sonia Altieri, Designated Federal Officer, U.S. EPA, Office of Cooperative Environmental management, at (202) 564–9788.

Dated: December 20, 1999.

Sonia Altieri,

Designated Federal Officer, Governmental Advisory Committee.

[FR Doc. 00–358 Filed 1–7–00; 8:45 am]

BILLING CODE 6560–50–M

ENVIRONMENTAL PROTECTION AGENCY

[PF–902; FRL–6394–6]

Notice of Filing Pesticide Petitions to Establish Tolerance for Certain Pesticide Chemicals in or on Food

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 docket control number PF–901, must be received on or before February 9, 2000.

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–901 in the subject line on the first page of your response.

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

Product Manager	Office location/telephone number/e-mail address	Address	Petition number(s)
James A. Tompkins (PM 25).	Rm. 239, CM #2, 703–305–5697, e-mail: tompkins.james@epamail.epa.gov.	1921 Jefferson Davis Hwy. Arlington, VA	PP 8F4973, 9F5096, 9F6007, and 0F6071
Joe Travano (PM 10)	Rm. 214, CM #2, 703–305–6411, e-mail: travano.joe@epamail.epa.gov.	Do.	PP 9F6033 and 9F6062

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	Examples of potentially affected entities
Industry	111	Crop production
	112	Animal production
	311	Food manufacturing
	32532	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'' 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–901. 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–901 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, 401 M St., SW., 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–901. 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 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.

List of Subjects

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

Dated: December 3, 1999.

Peter Caulkins, Acting

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

The 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 petitions 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. Monsanto Company

PP 8F4973, 9F5096, 9F6007, and 0F6071

EPA has received pesticide petitions (8F4973, 9F5096, 9F6007, and 0F6071) from Monsanto Company, 600 13th Street NW., Suite 660, Washington, DC 20005 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of glyphosate (N-(phosphonomethyl)glycine) in or on the raw agricultural commodities (RACs) corn, field, forage at 3.0 parts per million (ppm); (8F4973); alfalfa, hay at 400 ppm and alfalfa, forage at 175 ppm (9F5906); and stover and straw of cereal grains group at 100 ppm (9F6007). Monsanto proposes deletion of currently established tolerances on alfalfa at 200 ppm, alfalfa, fresh 0.2 ppm (9F5906); corn, field, stover at 100 ppm; sorghum, grain, stover at 40 ppm, and wheat straw at 85 ppm (9F6007). The proposed deletions are either no longer needed or are superceded by the proposed crop group tolerance.

Under PP 0F6071, Monsanto proposes that 40 CFR 180.364(a) header be amended to read as follows:

General. Tolerances are established for residues of glyphosate (N-(phosphonomethyl)glycine) per se resulting from the application of the isopropylamine salt of glyphosate, the ethanolamine salt of glyphosate, and the ammonium salt of glyphosate in or on the following RAC.

Monsanto also proposes that 40 CFR 180.364(a) be amended so that the headers for paragraphs (a)(2) and (a)(3) are deleted and the commodity tolerances listed in paragraphs (a)(2) and (a)(3) are reorganized into section (a) in alphabetical order under the header amended above.

Monsanto proposes that 40 CFR 180.364(d) be amended to read as follows:

Indirect or inadvertent residues. Tolerances are established for residues of glyphosate (N-(phosphonomethyl)glycine) per se resulting from the use of irrigation water containing residues of 0.5 ppm following applications on around aquatic sites at 0.1 ppm on the crop groupings citrus, cucurbits, forage grasses, forage legumes, fruiting vegetables, grain crops, leafy vegetables, nuts, pome fruits, root crop vegetables, seed and pod vegetables, stone fruits, and the individual commodities cottonseed, hops, and avocados. Where tolerances are established at higher levels from other uses of glyphosate in or on the subject crops, the higher tolerance should also apply to residues from the aquatic uses cited in this paragraph.

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 petitions. Additional data may be needed before EPA rules on these petitions.

A. Residue Chemistry

1. *Plant metabolism.* The qualitative nature of the residue in plants is adequately understood. Studies with a variety of plants including corn, cotton, soybeans, and wheat indicate that the uptake of glyphosate or its metabolite, aminomethylphosphonic acid (AMPA), from soil is limited. The material which is taken up is readily translocated. Foliarly applied glyphosate is absorbed and translocated throughout the trees or vines to the fruit of apples, coffee, dwarf citrus (calamondin), pears, and grapes. Metabolism via N-methylation yields N-methylated glycines and phosphonic acids. For the most part, the ratio of glyphosate to AMPA is 9 to 1 but can approach 1 to 1 in a few cases (e.g., soybeans and carrots). Much of the residue data for crops reflect a detectable residue of parent (0.05 - 0.15 ppm) along with residues below the level of detection (< 0.05 ppm) of AMPA. Only glyphosate parent is regulated in plant and animal commodities since the metabolite AMPA is not of toxicological concern.

2. *Analytical method.* Adequate enforcement methods are available for analysis of residues of glyphosate in or on plant commodities. These methods include gas liquid chromatography (GLC) (Method I in Pesticides Analytical Manual (PAM) II; the limit of detection is 0.05 ppm) and high performance liquid chromatography (HPLC) with fluorometric detection. The HPLC procedure has undergone successful Agency validation and was recommended for inclusion in PAM II. A gas chromatography/mass spectrometry (GC/MS) method for glyphosate in crops has also been validated by EPA's Analytical Chemistry Laboratory (ACL). The proposed revision in the tolerance regulation does not change the residue to be analyzed, which remains as glyphosate *per se*.

Adequate analytical methods are available for residue data collection and enforcement of proposed tolerances of glyphosate in or on alfalfa, hay; alfalfa, forage; corn, field, forage; and the stover and straw of cereal grains group.

3. *Magnitude of residues.* Adequate data concerning glyphosate residues on RAC have previously been submitted to the Agency. Accordingly, the available residue data for glyphosate support the proposed revision of the tolerance expression for glyphosate. As noted above, the proposed revision will permit glyphosate residues from the application of glyphosate in the form of its ethanolamine salt. In addition, any secondary residues occurring in liver, or kidney of cattle, goats, horses, and sheep, and liver and kidney of poultry will be covered by existing tolerances.

The submitted residue data adequately support the proposed tolerance on corn, field, forage at 3.0 ppm. The available crop residue data support the establishment of tolerances on alfalfa, hay at 400 ppm and alfalfa, forage at 175 ppm. The available data also support deletion of the current entries for alfalfa at 200 ppm and alfalfa, fresh at 0.2 ppm. The available crop field trial data support the establishment of tolerances in stover and straw of cereal grains group at 100 ppm. This tolerance is based on data from the three indicator crops corn, field, stover, wheat straw, and sorghum, stover that have previously been reviewed. Any secondary residues occurring in liver and kidney of cattle, goats, hogs, horses, and sheep, and liver and kidney of poultry will be covered by existing tolerances and the available data indicate that residues of glyphosate are not anticipated to occur in any other livestock commodities as a result of this action.

B. Toxicological Profile

EPA has previously evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children. The nature of the toxic effects caused by glyphosate are summarized below:

1. *Acute toxicity.* Several acute toxicology studies placing technical-grade glyphosate in Toxicity Category III and Toxicity Category IV. Technical glyphosate is not a dermal sensitizer.

2. A 21-day dermal toxicity study rabbits were exposed to glyphosate at levels of 0, 10, 1,000, or 5,000 milligrams/kilograms/day (mg/kg/day). The systemic no observed adverse effect level (NOAEL) was 1,000 mg/kg/day

and the lowest observed adverse effect level (LOAEL) was 5,000 mg/kg/day based on decreased food consumption in males. Although serum lactate dehydrogenase was decreased in both sexes at the high dose, this finding was not considered to be toxicologically significant.

3. *Genotoxicity*. Mutagenicity data included chromosomal aberration *in vitro* (no aberrations in Chinese hamster ovary (CHO) cells were caused with and without S9 activation); DNA repair in rat hepatocyte; *in vivo* bone marrow cytogenetic test in rats; rec-assay with *B. subtilis*; reverse mutation test with *S. typhimurium*; Ames test with *S. typhimurium*; and dominant-lethal mutagenicity test in mice. These genotoxicity studies are all negative.

4. *Reproductive and developmental toxicity*. Developmental toxicity studies were conducted with glyphosate in the rat and rabbit. In the rat study, test animals were given oral doses of 0, 300, 1,000, and 3,500 mg/kg/day with a developmental (fetal) NOAEL of 1,000 mg/kg/day based on an increase in number of litters and fetuses with delayed maturation of sternebrae and reduced body weight at 3,500 mg/kg/day, and a maternal NOAEL of 1,000 mg/kg/day based on clinical signs of toxicity and mortality at 3,500 mg/kg/day highest dose tested (HDT).

In the rabbit study, test animals were orally given doses of 0, 75, 175 and, 350 mg/kg/day of glyphosate. The maternal NOAEL is 175 mg/kg/day based on clinical signs of toxicity and mortality and the fetal NOAEL is 350 mg/kg/day HDT based on no developmental toxicity at any dose tested.

Two studies evaluating the reproductive effects of glyphosate were conducted in the rat. In a 3–generation study, rats were fed dosage levels of 0, 3, 10, and 30 mg/kg/day of glyphosate. The NOAEL for systemic and reproductive/developmental parameters is 30 mg/kg/day based on no-adverse effects noted at any dose level.

In a 2–generation reproduction study, rats were fed dosage levels of 0,100, 500, and 1,500 mg/kg/day of glyphosate. The NOAEL for systemic and developmental parameters is 500 mg/kg/day based on body weight effects, clinical signs of toxicity in adult males and decreased pup body weights and a reproductive NOAEL of 1,500 mg/kg/day HDT.

5. *Subchronic toxicity*. Subchronic (90–day) feeding studies were conducted with the rat, mouse, and dog. In the rat study, the test animals were fed dosage levels of 0, 1,000, 5,000, and 20,000 ppm of glyphosate. The NOAEL is 20,000 ppm based on no-effects at the HDT. In the mouse study, the test animals were fed dosage levels of 0, 5,000, 10,000, and 50,000 ppm of glyphosate. The NOAEL is 10,000 ppm based on body weight effects at the HDT.

In the dog study, the test animals were given glyphosate, via capsule, at doses of 0, 200, 600, and 2,000 mg/kg/day. The NOAEL is 2,000 mg/kg/day based on no-effects at the HDT.

6. *Chronic toxicity*. In a 12–month oral study, dogs were given glyphosate, via capsule, at doses of 0, 20, 100, and 500 mg/kg/day. The NOAEL is 500 mg/kg/day based on no-adverse effects at any dose level.

In a 26–month chronic feeding/oncogenicity study, rats were fed glyphosate at dosage levels of 0, 3, 10, and 31 mg/kg/day (males) and 0, 3, 11, and 34 mg/kg/day (females). The NOAEL is 31 mg/kg/day (males) and 34 mg/kg/day (females) based on no carcinogenic or other adverse effects at any dose level. Because a maximum tolerated dose (MTD) was not reached, this study was classified as supplemental for carcinogenicity.

In a 24–month chronic feeding/oncogenicity study, rats were fed glyphosate at dosage levels of 0, 89, 362, and 940 mg/kg/day (males) and 0, 113, 457, and 1,183 mg/kg/day (females). The systemic NOAEL is 362 mg/kg/day based on body weight effects in the female and eye effects in males. There was no carcinogenic response at any dose level.

In a mouse oncogenicity study, mice were fed glyphosate at dosage levels of 0, 150, 750, and 4,500 mg/kg/day with a NOAEL of 750 mg/kg/day based on body weight effects and microscopic liver changes at the HDT. There was no carcinogenic effect at the HDT of 4,500 mg/kg/day. Glyphosate is classified as a Group E (evidence of non-carcinogenicity for humans). This classification is based on the following findings:

i. There were no treatment related tumor findings in three state-of-the-art long-term bioassays.

ii. Glyphosate was tested up to the limit dose in the rat and up to levels higher than the limit dose in mice.

iii. There is no evidence of genotoxicity for glyphosate.

7. *Animal metabolism*. The qualitative nature of the residue in animals is adequately understood. Studies with lactating goats and laying hens fed a mixture of glyphosate and AMPA indicate that the primary route of elimination was by excretion (urine and feces). These results are consistent with metabolism studies in rats, rabbits, and cows. The terminal residues in eggs, milk, and animal tissues are glyphosate and its metabolite AMPA; there was no evidence of further metabolism. The terminal residue to be regulated in livestock is glyphosate *per se*.

8. *Metabolite toxicology*. Only glyphosate parent is to be regulated in plant and animal commodities since the metabolite AMPA is not of toxicological concern in food.

9. *Endocrine disruption*. The toxicity studies required by EPA for the registration of pesticides measure numerous endpoints with sufficient sensitivity to detect potential endocrine-modulating activity. No effects have been identified in subchronic, chronic, or developmental toxicity studies to indicate any endocrine-modulating activity by glyphosate. In addition, negative results were obtained when glyphosate was tested in a dominant-lethal mutation assay. While this assay was designed as a genetic toxicity test, agents that can affect male reproduction function will also cause effects in this assay. More importantly, the multi-generation reproduction study in rodents is a complex study design which measures a broad range of endpoints in the reproductive system and in developing offspring that are sensitive to alterations by chemical agents. Glyphosate has been tested in two separate multi-generation studies and each time the results demonstrated that glyphosate is not a reproductive toxin.

C. Aggregate Exposure

1. *Dietary exposure*. Tolerances have been established (40 CFR 180.364) for the residues of (n-(phosphonomethyl)glycine resulting from the application of the isopropylamine salt of glyphosate and/or the monoammonium salt of glyphosate, in or on a variety of plant and animal RACs including kidney of cattle, goats, hogs, horses, and sheep at 4.0 ppm; liver of cattle, goats, hogs, horses, and sheep at 0.5 ppm; and liver and kidney of poultry at 0.5 ppm based on animal feeding studies and worst-case livestock diets. The RAC corn, field, forage is not consumed by humans. Thus, the only possible exposure from this increased tolerance would be secondary residues in animal commodities which may occur from this use through the feeding of corn forage to livestock.

The petition proposes to expand this residue definition to include application of the ethanolamine salt of glyphosate. Risk assessments were conducted by EPA to assess dietary exposures from glyphosate as follows:

i. *Food—Acute exposure and risk*. Acute dietary risk assessments are

performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1 day or single exposure. An acute dietary risk assessment was not performed because no endpoints attributable to single dose were identified in the oral studies including rat and rabbit developmental studies. There are no data requirements for acute and subchronic neurotoxicity studies and no evidence of neurotoxicity in any of the toxicity studies at very high doses. The Agency has concluded with reasonable certainty that glyphosate dose not elicit an acute toxicological response, and that an acute dietary risk assessment is not needed.

ii. *Chronic exposure and risk*. The chronic dietary exposure analysis was conduced using the reference dose (RfD) of 2.0 mg/kg/day based on the maternal NOAEL of 175 mg/kg/day from a developmental study and an uncertainty factor of 100 (applicable to all population groups) the Dietary Exposure Evaluation Model (DEEM) analysis assumed tolerance levels residues and 100% of the crop treated. These assumptions resulted in the following theoretical maximum residue contributions (TMRC) and percentage RfDs for certain population subgroups. The TMRC for the U.S. population (48 contiguous States) was 0.029960 or 1.5% of the RfD, 0.026051 or 1.3% of the RfD for nursing infants (less than on 1 year old), 0.065430 or 3.3% of the RfD for non-nursing infants less than 1 year old; 0.064388 or 3.2% of the RfD for children (1–6 years old); 0.043017 or 2.2% of the RfD for children (7–12 years old); 0.030928 or 1.5% of the RfD for females (13+/nursing); 0.030241 or 1.5% of the RfD for non-Hispanic whites; and 0.030206 or 1.5% of the RfD for non-Hispanic blacks. These exposure levels will be unaffected by the proposed amendment to the tolerance regulation.

iii. *Chronic risk-carcinogenic*. Glyphosate has been classified as a *Group E* chemical evidence of carcinogenicity in two acceptable animal species.

iv. *Drinking water*. Generic Expected Environmental Concentration (GENEEC) and Screening Concentration and Ground Water (SCI-GROW) models were run by EPA to produce maximum estimates of glyphosate concentrations in surface and ground water, respectively. The drinking water exposure for glyphosate from the ground water screening model, SCI-GROW, yields a peak and chronic Estimated Environmental Concentration (EEC) of 0.0011 parts per billion (ppb) in ground water. The GENEEC values represent upper-bound estimates of the concentrations that might be found in surface water due to glyphosate use. Thus, the GENEEC model predicts that glyphosate surface water concentrations range from a peak of 1.64 ppb to a 56–day average of 0.19 ppb. The model estimates are compared directly to drinking water level of comparison (DWLOC) (chronic). The DWLOC (chronic) is the theoretical concentration of glyphosate in drinking water so that the aggregate chronic exposure (food + water + residential) will occupy no more than 100% of the RfD. This assessment does not take into account expected reductions in any glyphosate concentrations in water arising from water treatment of surface water prior to releasing it for drinking purposes. The Agency's default body weights and consumption values used to calculate DWLOCs are as follows: 70 kg/2L (adult male), 60 kg/2L (adult female), and 10 kg/1L (child).

v. *Acute exposure and risk*. An acute dietary endpoint and dose was not identified in the toxicology data base. Adequate rat and rabbit developmental studies did not provide a dose or endpoint that could be used for acute dietary risk purposes. Additionally, there were no data requirements for acute or subchronic rat neurotoxicity studies since there was no evidence of neurotoxicity in any of the toxicology studies at very high doses.

vi. *Chronic exposure and risk*. The DWLOC (chronic, non-cancer) risk is calculated by multiplying the allowed chronic water exposure (mg/kg/day) x body weight (kg) divided by the consumption (L) x 103 µg/mg. The DWLOCs are 69,000 µg/L for the U.S. population in 48 contiguous States, males (13+), non-Hispanic whites, and non-Hispanic blacks; and 19,000 for non-nursing infants (less than 1 year old) and children (1–6 years). Although the GENEEC and SCI-GROW models are known to produce worst-case estimates, the resulting average concentrations of glyphosate in the surface and ground water are more than 10,000-fold less than the DWLOC (chronic). Therefore, taking into account present uses and uses proposed in this action, Monsanto concludes with reasonable certainty that no harm will result from chronic aggregate exposure to glyphosate.

2. *Non-dietary exposure*. Glyphosate is currently registered for use on the following residential non-food sites: Around ornamentals, shade trees, shrubs, walk, driveways, flower beds, and home lawns. Based on the registered uses of glyphosate, the potential for residential exposures exists. However, based on the low acute toxicity and lack of other toxicological concerns, glyphosate does not meet the Agency's criteria for residential data requirements and a residential exposure assessment is not required since there are no toxicological endpoints selected for either dermal or inhalation exposure. Exposures from residential uses are not expected to pose undue risks or harm to public health.

i. *Acute exposure and risk*. There are no acute toxicological concerns for glyphosate. Glyphosate has been the subject of numerous incident reports, primarily for eye and skin irritation injuries, in California. Some glyphosate end-use products are in Toxicity Categories I and II for eye and dermal irritation. The Reregistration Eligibility Decision Document for Glyphosate (September 1993) indicated that the Agency is not adding additional personal protective equipment (PPE) requirements to labels of end-use products, but that it continues to recommend the PPE and precautionary statements required for end-use products in Toxicity Categories I and II.

ii. *Chronic exposure and risk*. Although there are registered residential uses for glyphosate, glyphosate does not meet the Agency's criteria for residential data requirements, due to the lack of toxicological concerns. Incidental acute and/or chronic dietary exposures from residential uses of glyphosate are not expected to pose undue risks to the general population, including infants and children.

iii. *Short- and intermediate-term exposure and risk*. EPA identified no toxicological concerns for short-intermediate- and long-term dermal or inhalation routes of exposures for glyphosate. The Agency has concluded that exposures from residential uses of glyphosate are not expected to pose undue risks.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information''concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.''

EPA does not have, at this time, available data to determine whether glyphosate has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, glyphosate does not produce a toxic metabolite that is also produced by other substances. For the purposes of

this tolerance action, therefore, EPA should assume that glyphosate does not have a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the final rule for Bifenthrin Pesticide Tolerances November 26, 1997, (62 FR 62961) (FRL–5754–7).

E. Safety Determination

1. *U.S. population*—i. *Acute risk*. There was no acute dietary endpoint identified, therefore, there are no acute toxicological concerns for glyphosate.

ii. *Chronic risk*. Using the TMRC exposure assumptions described in this unit, EPA has concluded that aggregate exposure to glyphosate from food will utilize 1.5% of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is non- nursing infants (less than 1 year) and children (1–6 years old) as discussed below. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to glyphosate in drinking water and from non-dietary, non-occupational exposure, the aggregate exposure will not exceed 100% of the RfD. EPA has previously concluded that there is a reasonable certainty that no harm will result from aggregate exposure to glyphosate residues at this level. The safety determination is unaffected by the proposed change in the tolerance regulation.

iii. *Short- and intermediate-term risk*. Short-and intermediate-term dermal and inhalation risk is not a concern due to the lack of significant toxicological effects observed with glyphosate under these exposure scenarios. Short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure.

iv. *Aggregate cancer risk for U.S. population*. Glyphosate has been classified as a Group E chemical, with no evidence of carcinogenicity for humans in two acceptable animal studies.

v. *Determination of safety*. Based on these risk assessments, Monsanto concludes that there is a reasonable certainty that no harm will result from aggregate exposure to glyphosate residues.

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

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a margin of exposure (MOE) analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined interspecies and intraspecies variability) and not the additional tenfold MOE/uncertainty factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor.

i. *Prenatal and postnatal sensitivity*. The oral prenatal and prenatal data demonstrated no indication of increased sensitivity of rats or rabbits to *in utero* and postnatal exposure to glyphosate.

ii. *Conclusion*. There is a complete toxicity data base for glyphosate and exposure data are complete or are estimated based on data that reasonably accounts for potential exposures. Based on these data, there is no indication that the developing fetus or neonate is more sensitive than adult animals. No developmental neurotoxicity studies have been required at this time. A developmental neurotoxicity data requirement is an upper tier study and is required only if effects observed in the acute and 90–day neurotoxicity studies indicate concerns for frank neuropathy or alterations seen in fetal nervous system in the developmental or reproductive toxicology studies. The Agency has concluded that reliable data support the use of the standard 100-fold uncertainty factor for glyphosate, and that a tenfold (10x) uncertainty factor is not needed to protect the safety of infants and children.

iii. *Acute risk*. There are no acute toxicological endpoints for glyphosate. The Agency has concluded that establishment of the proposed tolerances would not pose an unacceptable aggregate risk.

3. *Chronic risk*. Using the exposure assumptions described in this unit, EPA has concluded that aggregate exposure to glyphosate from food utilizing present tolerances will utilize 3.0% of the RfD for infants and children. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. These dietary exposure levels are unaffected by the proposed tolerances on alfalfa, hay and alfalfa, forage, because these commodities are only consumed by livestock, and the existing tolerances in liver and kidney fractions of cattle, goats, horses, sheep, and poultry are considered sufficient to account for any additional dietary burden these animals may encounter. Although there is a low likelihood potential exposure to glyphosate in drinking water and from non-dietary, non-occupational exposure, EPA has previously concluded that the aggregate exposure is not expected to exceed 100% of the RfD. The safety determination is unaffected by the proposed change in the tolerance regulation.

4. *Short- or intermediate-term risk*. Short-term and intermediate- term dermal and inhalation risk is not a concern due to the lack of significant toxicological effects observed with glyphosate under these exposure scenarios.

5. *Determination of safety*. Based on these risk assessments, EPA has previously concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to glyphosate residues at these levels.

F. International Tolerances

No Codex Maximum Residue Levels (MRLs) exist for alfalfa, hay, alfalfa, forage; or corn, field, forage. A MRL exists for straw and fodder (dry) of cereal grains (Code as 0091) at 100 ppm. Therefore, the proposed tolerance for stover and straw of cereal grains group at 100 ppm will harmonize to United States regulations with those in place internationally. Codex MRLs have been established in or on many RACs. These petitions propose no additional numerical changes; therefore, the

agreement between United States tolerances and Codex MRLs are not affected by other proposals in this action.

2. Rohm and Haas Company

9F6033

EPA has received a pesticide petition (9F6033) from Rohm and Haas Company, 100 Independence Mall West, Philadelphia, PA proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of methoxyfenozide benzoic acid, 3-methoxy-2-methyl-,2-(3,5-dimethylbenzoyl)-2-(1,1- dimethylethyl) hydrazide in or on the RACs grapes, raisins, and fruiting vegetables (except cucurbits) at 1.0, 1.5, and 2.0 ppm respectively. 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 methoxyfenozide in plants (grapes, apples, cotton, and rice) is adequately understood for the purposes of these tolerances. The metabolism of methoxyfenozide in all crops was similar and involves cleavage of the methoxyl side chain to the free phenol, RH-117236 or oxidation of the alkyl substituents of the aromatic rings primarily at the benzylic positions. In all crops, parent compound comprised the majority of the total dosage. None of the metabolites were in excess of 10% of the total dosage.

2. *Analytical method*. A high performance liquid chromatographic (HPLC) analytical method using ultraviolet (UV) or mass selective (MS) detection has been validated for grapes, raisins, grape juice, wine, peppers, tomatoes, and tomato processed fractions (juice, puree, paste). The method involves extraction by blending with solvents, purification of the extracts by liquid-liquid partitions and final purification of the residues using basic alumina column chromatography and solid phase extraction. The LOQ is 0.02 ppm for all matrices.

3. *Magnitude of residues*—i. *Grapes*. Residue studies showed a range of residues in grapes from 0.20–0.86 ppm, and support a tolerance of 1.0 ppm in grapes and 1.5 ppm in raisins. Residues did not concentrate in red and white wine or in clarified and unclarified juice.

ii. *Fruiting vegetables*. Residue studies showed a range of residues in peppers from 0.032–1.03 ppm and in tomatoes (cherry and non-cherry) from 0.05–1.86, supporting a tolerance of 2.0 ppm for the crop group. No concentration of residues was seen in the tomato processed fractions juice, puree or paste.

B. Toxicological Profile

1. *Acute toxicity*. Methoxyfenozide has low acute toxicity. Methoxyfenozide was practically non-toxic by ingestion of a single oral dose in rats and mice (LD_{50} > 5,000 mg/kg) and was practically non-toxic by dermal application (LD_{50} > 5,000 mg/kg). Methoxyfenozide was not significantly toxic to rats after a 4–hour inhalation exposure with an LC_{50} value of > 4.3 milligrams per liter (mg/L) (highest attainable concentration), is not considered to be a primary eye irritant or a skin, irritant, and is not a dermal sensitizer. An acute neurotoxicity study in rats did not produce any neurotoxic or neuropathologic effects with a NOAEL > 2,000 mg/kg.

2. *Genotoxicty*. Methoxyfenozide tested negative (non-mutagenic, non-genotoxic) in a battery of *in vitro* and *in vivo* assays, which included an Ames assay with and without metabolic activation, a CHO/HGPRT assay, an *in vitro* chromosome aberration assay in CHO cells with and without a metabolic activation, an *in vivo* micronucleus assay in mouse bone marrow cells.

3. *Reproductive and developmental toxicity*—i. NOAELs for developmental and maternal toxicity to methoxyfenozide were established at 1,000 mg/kg/day HDT in both the rat and rabbit. No signs of developmental toxicity were exhibited.

ii. In a 2–generation reproduction study in the rat, the reproductive/developmental toxicity NOAELs of 1,552 mg/kg/day was 100-fold higher than the parental (systemic) toxicity NOAEL of 200 ppm (15.5 mg/kg/day).

4. *Subchronic toxicity*—i. The NOAEL in a 90–day rat feeding study was 1,000 ppm (69.3 mg/kg/day for males, 72.4 mg/kg/day for females). The LOAEL was 5,000 ppm (353 mg/kg/day for males, 379 mg/kg/day for females). Increased liver weight and liver histopathology were observed at the LOAEL of 5,000 ppm. Methoxyfenozide did not produce neurotoxic or neuropathologic effects when administered in the diets of rats for 3 months at concentrations up to and including the limit dose of 20,000 ppm (NOAEL = 1,318 mg/kg/day for males, 1,577 mg/kg/day for females).

ii. In a 90–day feeding study with mice, the NOAEL was 2,500 ppm (428 and 589 mg/kg/day for males and females, respectively). The LOAEL was 7,000 ppm (1,149 and 1,742 mg/kg/day for males and females, respectively). Decreases in body weight gain were noted in both sexes of mice at the LOAEL of 7,000 ppm.

iii. A 90–day dog feeding study gave a NOAEL of 3,000 ppm, the HDT (198 and 209 mg/kg/day for males and females, respectively). Extension of treatment of the low dose animals for 6 weeks at 15,000 ppm (422 and 460 mg/kg/day for males and females, respectively) produced no signs of systemic toxicity.

Methoxyfenozide did not produce toxicity in the rat when administered dermally for 4 weeks at doses up to and including the limit dose of 1,000 mg/kg/day. These findings correlate with the low dermal penetration observed with ^{14}C-methoxyfenozide, formulated as the wettable powder (i.e., after 24 hours 1–3% of the administered dose was systemically absorbed).

5. *Chronic toxicity*—i. The NOAEL in a 1 year feeding study in dogs was 300 ppm (9.8 and 12.6 mg/kg/day for male and females, respectively). The LOAEL was 3,000 ppm (106 and 111 mg/kg/day for male and females, respectively) based on minimal hematological effects.

ii. An 18–month mouse carcinogenicity study showed no signs of carcinogenicity at dosage levels up to and including 7,000 ppm (1,020 and 1,354 mg/kg/day for male and females, respectively), the HDT.

iii. In a combined rat chronic/oncogenicity study, the NOAEL for chronic toxicity was 200 ppm (10.2 and 11.9 mg/kg/day for males and females, respectively) and the LOAEL was 8,000 ppm (411 and 491 mg/kg/day for males and females, respectively). No carcinogenicity was observed at the dosage levels up to 20,000 ppm (1,045 and 1,248 mg/kg/day for males and females, respectively).

6. *Animal metabolism*. In toxicokinetic and metabolism studies in the rat, methoxyfenozide was rapidly absorbed following oral exposure with peak plasma levels occurring within 0.5 hours of administration. Methoxyfenozide does not bioaccumulate in that the compound is rapidly and almost completely eliminated within 24 hours. Methoxyfenozide was extensively metabolized in rats. Including parent compound, 32 metabolites, of which 26 were identified, were isolated from the rat urine and feces. The primary pathway of methoxyfenozide metabolism involves demethylation of the A-ring methoxyl moiety to form the corresponding A-ring phenol, RH-

117,236, which is readily conjugated with glucuronic acid to RH-1518. Hydroxylation on the B-ring methyl moieties is also an important metabolic pathway.

7. *Metabolite toxicology*. Common metabolic pathways for methoxyfenozide have been identified in both plants (grape, apple, rice, and cotton) and animals (rat, goat, hen). Extensive degradation and elimination of polar metabolites occurs in animals such that residues are unlikely to accumulate in humans or animals exposed to these residues through the diet. The rapid metabolism and excretion of methoxyfenozide in part accounts for the compound's overall low toxicity profile in animals.2 The main metabolite of methoxyfenozide in plants and animals, the A-ring phenol, RH-117,236, produced no toxicity in mice (LD_{50} > 5,000 mg/kg) and was negative when tested in the Ames mutagenic assay. Other metabolites of methoxyfenozide (e.g., glucuronides) would be expected to produce minimal to no toxicity given structure activity considerations.

8. *Endocrine disruption*. Based on structure-activity information as well as the lack of developmental and reproductive toxicity, methoxyfenozide is unlikely to exhibit estrogenic activity. No indicators of estrogenic or other endocrine effects were observed in mammalian chronic studies or in mammalian and avian reproduction studies. Methoxyfenozide is within a class of chemistry (diacylhydrazines) that is not known to bind to mammalian steroid receptors. Overall, the weight of evidence provides no indication that methoxyfenozide has endocrine activity in vertebrates.

C. Aggregate Exposure

1. *Dietary exposure*. Tolerances are proposed for the residues of methoxyfenozide in or grapes, raisins, and fruiting vegetables. Risk assessments were conducted by Rohm and Haas to assess dietary exposures and risks from methoxyfenozide as follows:

i. *Food—a. Acute exposure and risk*. No acute endpoint of concern was identified for methoxyfenozide and no acute risk assessment is required.

b. *Chronic exposure and risk*. For chronic dietary risk assessment, the proposed tolerance values and anticipated (average) residues are used and the assumption that 100% of all grapes and fruiting vegetables (in addition to cotton, and pome fruit) will contain residues of methoxyfenozide at the tolerance or anticipated residue levels. The RfD used for the chronic dietary analysis is 0.1 mg/kg/day based on the NOAEL of 9.8-10.0 mg/kg/day from the rat and dogs chronic studies. Potential chronic exposures were estimated using NOVIGEN'S (DEEM Version 6.74) which uses USDA food consumption data from the 1994-1996 survey. With the proposed tolerances and anticipated residue levels for methoxyfenozide, the percentage of the RfD utilized is as follows:

Groups	Tolerance Levels, Total %RfD	Anticipated Residues, Total %RfD
U.S. Population - 48 contiguous States	6.8	0.6
Hispanics	7.5	0.6
Non-Hispanic/non-white/non-black	6.9	0.7
Nursing Infants > 1 year old	5.2	0.8
Non-Nursing Infants > 1 year old	14.7	2.0
Children 1–6 years old	20.2	1.9
Children 7-12 years old	9.3	0.8

The chronic dietary risks from these uses do not exceed EPA's level of concern.

ii. *Drinking water*. Submitted environmental fate studies suggest that methoxyfenozide is moderately persistent and mobile, and could potentially leach to ground water and runoff to surface water under certain environmental conditions. However, in terrestrial field dissipation and orchard dissipation studies, residues of methoxyfenozide showed minimal mobility and remained associated with the upper layers of soil. Foliar interception (up to 70% of the total dosage applied) by target crops reduces the ground level residues of methoxyfenozide.

Acute and chronic exposures to methoxyfenozide in drinking water were estimated using the GEENEC V1.2 and SCI-GROW models, as directed in OPP's Interim Approach for Addressing Drinking Water Exposure. GEENEC is a highly conservative model used to estimate residue concentrations in surface water. SCI-GROW is an equally conservative model used to estimate residue concentrations in shallow, highly vulnerable ground water (i.e., sites with sandy soils and depth to ground water of 10 to 20 feet). As indicated in EPA's drinking water exposure guidance, a very small percentage of people in the United States would derive their drinking water from such sources. GEENEC (56–day average) and SCI-GROW water exposure values for methoxyfenozide utilize 1% or less of the RfD for adults and children.

There is no established Maximum Concentration Level (MCL) for residues of methoxyfenozide in drinking water. No drinking water health advisory levels have been established for methoxyfenozide. There is no entry for methoxyfenozide in the ''Pesticides in Groundwater Database'' (EPA 734-12-92-001, September 1992).

iii. *Chronic exposure and risk*. There are insufficient water-related exposure data to complete a comprehensive drinking water assessment for methoxyfenozide at this time. However, in order to mitigate the potential for methoxyfenozide to leach into ground water or runoff to surface water, precautionary language has been incorporated into the proposed product label. Also, to the best of our knowledge, previous experience at EPA with more persistent and mobile pesticides for which there were available data to perform quantitative risk assessments demonstrated that drinking water exposure was typically a small percentage of the total dietary exposure. This observation holds even for pesticides detected in wells and drinking water at levels nearing or exceeding established MCLs. Considering the precautionary language on the label and our knowledge of previous experience with persistent chemicals, no risk from residues of methoxyfenozide in drinking water is anticipated.

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

dietary residential exposure is anticipated.

D. Cumulative Effects

The methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way are not available at this time. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decision on specific classes of chemicals will be heavily dependent on chemical-specific data, much of which may not be presently available.

Although at present the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides for which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed).

At this time, no data are available to determine whether methoxyfenozide benzoic acid, 3-methoxy-2-methyl-, 2-(3,5-dimethylbenzoyl)-2-(1,1-dimethylethyl)hydrazide has a common mechanism of toxicity with other substances. Thus, it is not appropriate to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, methoxyfenozide benzoic acid, 3-methoxy-2-methyl-,2-(3,5-dimethylbenzoyl)-2-(1,1-dimethylethyl) hydrazide does not produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, methoxyfenozide benzoic acid, 3-methoxy-2-methyl-,2-(3,5-dimethylbenzoyl)-2-(1,1-dimethylethyl) hydrazide is assumed not to have a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*—i. *Acute exposure and risk*. Since no acute endpoint of concern has been identified for methoxyfenozide, no acute risk assessment is required.

ii. *Chronic exposure and risk*. Using the conservative exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of methoxyfenozide from the proposed tolerances is 6.8% (tolerance levels) and 0.6% (anticipated residues) for the U.S. population. Aggregate exposure (food and water) are not expected to exceed 100%. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Rohm and Haas concludes that there is a reasonable certainty that no harm will result from aggregate exposure to methoxyfenozide residues to the U.S. population.

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

ii. *Developmental toxicity studies*—a. *Rats*. In a developmental toxicity study in rats, the maternal (systemic) NOAEL was 1,000 mg/kg/day HDT. The developmental (pup) NOAEL was > 1,000 mg/kg/day HDT

b. *Rabbits*. In a developmental toxicity study in rats, the maternal (systemic) NOAEL was 1,000 mg/kg/day HDT. The developmental (pup) NOAEL was > 1,000 mg/kg/day HDT.

iii. *Reproductive toxicity study—Rats*. In a multi–generation reproductive toxicity study in rats, the parental (systemic) NOAEL was 15.5 mg/kg/day, based on liver effects at the LOAEL of 153 mg/kg/day. The reproductive (pup) NOAEL was 1,552 mg/kg/day HDT. No adverse reproductive effects were observed.

iv. *Prenatal and postnatal sensitivity*—a. *Prenatal sensitivity*. The developmental NOAELs of > 1,000 mg/kg/day HDT from the developmental toxicity studies in rats and rabbits demonstrate that there is no developmental (prenatal) toxicity present for methoxyfenozide. Additionally, these developmental NOAELs are greater than 100-fold higher than the NOAEL of 9.8–10.0 mg/kg/day from the rat and dogs chronic studies which are the basis of the RfD.

b. *Postnatal sensitivity*. In the reproductive toxicity study in rats, the reproductive NOAEL (1,552 mg/kg/day) is about 100-fold higher than the parental NOAEL (15.5 mg/kg/day). These developmental and reproductive studies indicate that methoxyfenozide does not have additional prenatal and postnatal sensitivity for infants and children in comparison to other exposed groups.

v. *Acute exposure and risk*. No acute endpoint was identified for methoxyfenozide, and therefore, no acute risk assessment is required.

vi. *Chronic exposure and risk*. For chronic dietary risk assessment, tolerances and anticipated residue values are used and the assumption that 100% of all grapes and fruiting vegetables (in addition to cotton and pome fruit) will contain residues at the tolerance or anticipated residue levels. The percentage RfD utilized from the proposed tolerances and anticipated residues is calculated using the DEEM (Version 6.74, licensed by Novigen Sciences Inc.) which uses USDA food consumption data from the 1994-1996 survey.

With the proposed tolerances and anticipated residues for methoxyfenozide, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of methoxyfenozide is 20.2% (tolerance levels) and 1.9% (anticipated residues) for children 1-6 years old. Aggregate exposure (food and water) are not expected to exceed 100%. Rohm and Haas concludes that there is a reasonable certainty that no harm will result from aggregate exposure to methoxyfenozide residues to non-nursing infants.

F. International Tolerances

There are currently no CODEX, Canadian or Mexican MRLs established for methoxyfenozide in grapes, raisins, or fruiting vegetables, so no harmonization issues are required for this action.

3. Rohm and Haas Company

9F6062

EPA has received a pesticide petition (9F6062) from Rohm and Haas

Company, 100 Independence Mall West, Philadelphia, PA, proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of methoxyfenozide benzoic acid, 3-methoxy-2-methyl-, 2-(3,5-dimethylbenzoyl)-2-(1,1-dimethylethyl) hydrazide in or on the RACs, leafy green vegetables, leaf petioles, head and stem brassica, and leafy brassica greens at 25.0, 10.0, 6.5, and 20.0 ppm respectively. 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 methoxyfenozide in plants (grapes, apples, cotton, and rice) is adequately understood for the purposes of these tolerances. The metabolism of methoxyfenozide in all crops was similar and involves cleavage of the methoxyl side chain to the free phenol, RH-117236, or oxidation of the alkyl substituents of the aromatic rings. In all crops, parent compound comprised the majority of the total dosage. None of the metabolites were in excess of 10% of the total dosage.

2. *Analytical method*. A high performance liquid chromatographic (HPLC) analytical method using ultraviolet (UV) or mass selective (MS) detection has been validated for vegetable crops. The method involves extraction by blending with solvents, purification of the extracts by liquid-liquid partitions and final purification of the residues using basic alumina column chromatography and solid phase extraction. The LOQ is 0.02 ppm for all matrices.

3. *Magnitude of residues*. The crop field trial data submitted with the petition support the proposed tolerances for residues of methoxyfenozide in leafy and cole crop vegetables.

B. Toxicological Profile

1. *Acute toxicity*. Methoxyfenozide has low acute toxicity. Methoxyfenozide was practically non-toxic by ingestion of a single oral dose in rats and mice (LD_{50} > 5,000 mg/kg) and was practically non-toxic by dermal application (LD_{50} > 5,000 mg/kg). Methoxyfenozide was not significantly toxic to rats after a 4–hour inhalation exposure with an LC_{50} value of > 4.3 mg/L (highest attainable concentration), is not considered to be a primary eye irritant or a skin irritant, and is not a dermal sensitizer. An acute neurotoxicity study in rats did not produce any neurotoxic or neuropathologic effects with a NOAEL > 2,000 mg/kg.

2. *Genotoxicty*. Methoxyfenozide tested negative (non-mutagenic, non-genotoxic) in a battery of *in vitro* and *in vivo* assays, which included an Ames assay with and without metabolic activation, a CHO/HGPRT assay, an *in vitro* chromosome aberration assay in CHO cells with and without a metabolic activation, an *in vivo* micronucleus assay in mouse bone marrow cells.

3. *Reproductive and developmental toxicity*—i. NOAELs for developmental and maternal toxicity to methoxyfenozide were established at 1,000 mg/kg/day HDT in both the rat and rabbit. No signs of developmental toxicity were exhibited.

ii. In a 2-generation reproduction study in the rat, the reproductive/developmental toxicity NOAEL of 1,552 mg/kg/day was 100-fold higher than the parental (systemic) toxicity NOAEL of 200 ppm (15.5 mg/kg/day).

4. *Subchronic toxicity*—i. The NOAEL in a 90–day rat feeding study was 1,000 ppm (69.3 mg/kg/day for males, 72.4 mg/kg/day for females). The LOAEL was 5,000 ppm (353 mg/kg/day for males, 379 mg/kg/day for females). Increased liver weight and liver histopathology were observed at the LOAEL of 5,000 ppm. Methoxyfenozide did not produce neurotoxic or neuropathologic effects when administered in the diets of rats for 3 months at concentrations up to and including the limit dose of 20,000 ppm (NOAEL = 1,318 mg/kg/day for males, 1,577 mg/kg/day for females).

ii. In a 90–day feeding study with mice, the NOAEL was 2,500 ppm (428 and 589 mg/kg/day for males and females, respectively). The LOAEL was 7,000 ppm (1,149 and 1,742 mg/kg/day for males and females, respectively). Decreases in body weight gain were noted in both sexes of mice at the LOAEL of 7,000 ppm.

iii. A 90–day dog feeding study gave a NOAEL of 3,000 ppm, the HDT (198 and 209 mg/kg/day for males and females, respectively). Extension of treatment of the low dose animals for 6 weeks at 15,000 ppm (422 and 460 mg/kg/day for males and females, respectively) produced no signs of systemic toxicity.

Methoxyfenozide did not produce toxicity in the rat when administered dermally for 4 weeks at doses up to and including the limit dose of 1,000 mg/kg/day. These findings correlate with the low dermal penetration observed with ^{14}C-methoxyfenozide, formulated as the wettable powder (i.e., after 24 hours 1–3% of the administered dose was systemically absorbed).

5. *Chronic toxicity*—i. The NOAEL in a 1 year feeding study in dogs was 300 ppm (9.8 and 12.6 mg/kg/day for male and females, respectively). The LOAEL was 3,000 ppm (106 and 111 mg/kg/day for male and females, respectively) based on minimal hematological effects.

ii. An 18–month mouse carcinogenicity study showed no signs of carcinogenicity at dosage levels up to and including 7,000 ppm (1,020 and 1,354 mg/kg/day for male and females, respectively), the HDT.

iii. In a combined rat chronic/oncogenicity study, the NOAEL for chronic toxicity was 200 ppm (10.2 and 11.9 mg/kg/day for males and females, respectively) and the LOAEL was 8,000 ppm (411 and 491 mg/kg/day for males and females, respectively). No carcinogenicity was observed at the dosage levels up to 20,000 ppm (1,045 and 1,248 mg/kg/day for males and females, respectively).

6. *Animal metabolism*. In toxicokinetic and metabolism studies in the rat, methoxyfenozide was rapidly absorbed following oral exposure with peak plasma levels occurring within 0.5 hour of administration. Methoxyfenozide does not bioaccumulate in that the compound is rapidly and almost completely eliminated within 24 hours. Methoxyfenozide was extensively metabolized in rats. Including parent compound, 32 metabolites, of which 26 were identified, were isolated from the rat urine and feces. The primary pathway of methoxyfenozide metabolism involves demethylation of the A-ring methoxyl moiety to form the corresponding A-ring phenol, RH-117,236, which is readily conjugated with glucuronic acid to RH-1518. Hydroxylation on the B-ring methyl moieties is also an important metabolic pathway.

7. *Metabolite toxicology*. Common metabolic pathways for methoxyfenozide have been identified in both plants (grape, apple, rice, and cotton) and animals (rat, goat, hen). Extensive degradation and elimination of polar metabolites occurs in animals such that residues are unlikely to accumulate in humans or animals exposed to these residues through the diet. The rapid metabolism and excretion of methoxyfenozide in part accounts for the compound's overall low toxicity profile in animals. The main metabolite of methoxyfenozide in plants and animals, the A-ring phenol, RH-117,236, produced no toxicity in mice (LD_{50} > 5,000 mg/kg) and was

negative when tested in the Ames mutagenic assay. Other metabolites of methoxyfenozide (e.g., glucuronides) would be expected to produce minimal to no toxicity given structure activity considerations.

8. *Endocrine disruption*. Based on structure-activity information as well as the lack of developmental and reproductive toxicity, methoxyfenozide is unlikely to exhibit estrogenic activity. No indicators of estrogenic or other endocrine effects were observed in mammalian chronic studies or in mammalian and avian reproduction studies. Methoxyfenozide is within a class of chemistry (diacylhydrazines) that is not known to bind to mammalian steroid receptors. Overall, the weight of evidence provides no indication that methoxyfenozide has endocrine activity in vertebrates.

C. Aggregate Exposure

1. *Dietary exposure*. Tolerances are proposed for the residues of methoxyfenozide in or grapes, raisins, and fruiting vegetables. Risk assessments were conducted by Rohm and Haas to assess dietary exposures and risks from methoxyfenozide as follows:

i. *Food—a. Acute exposure and risk*. No acute endpoint of concern was identified for methoxyfenozide and no acute risk assessment is required.

b. *Chronic exposure and risk*. For chronic dietary risk assessment, the proposed tolerance values and anticipated (average) residues are used and the assumption that 100% of all leafy and cole crop vegetable crops (in addition to cotton, pome fruit, grapes, and fruiting vegetables) will contain residues of methoxyfenozide at the tolerance levels. The RfD used for the chronic dietary analysis is 0.1 mg/kg/day based on the NOAEL of 9.8–10.0 mg/kg/day from the rat and dogs chronic studies. Potential chronic exposures were estimated using NOVIGEN'S (DEEM Version 6.74) which uses USDA food consumption data from the 1994-1996 survey. With the proposed tolerances for methoxyfenozide, the percentage of the RfD utilized is as follows:

Population Subgroup	Tolerance Levels, Total %RfD
U.S. Population - 48 contiguous States	16.4
Non-Hispanic/non-white/non-black	22.4
Nursing Infants < 1 year old	5.4
Non-Nursing Infants < 1 year old	23.1
Children 1–6 years old	29.9
Children 7–12 years old	18.1
Females 13+ (nursing)	16.7

The chronic dietary risks from these uses do not exceed EPA's level of concern.

ii. *Drinking water*. Submitted environmental fate studies suggest that methoxyfenozide is moderately persistent and mobile, and could potentially leach to ground water and runoff to surface water under certain environmental conditions. However, in terrestrial field dissipation and orchard dissipation studies, residues of methoxyfenozide showed minimal mobility and remained associated with the upper layers of soil. Foliar interception (up to 70% of the total dosage applied) by target crops reduces the ground level residues of methoxyfenozide.

Acute and chronic exposures to methoxyfenozide in drinking water were estimated using the GEENEC V1.2 and SCI-GROW models, as directed in OPP's Interim Approach for Addressing Drinking Water Exposure. GEENEC is a highly conservative model used to estimate residue concentrations in surface water. SCI-GROW is an equally conservative model used to estimate residue concentrations in shallow, highly vulnerable ground water (i.e., sites with sandy soils and depth to ground water of 10 to 20 feet). As indicated in EPA's drinking water exposure guidance, a very small percentage of people in the United States would derive their drinking water from such sources. GEENEC (56–day average) and SCI-GROW water exposure values for methoxyfenozide utilize 1% or less of the RfD for adults and children.

There is no established Maximum Concentration Level (MCL) for residues of methoxyfenozide in drinking water. No drinking water health advisory levels have been established for methoxyfenozide. There is no entry for methoxyfenozide in the ''Pesticides in Groundwater Database'' (EPA 734-12-92-001, September 1992).

iii. *Chronic exposure and risk*. There are insufficient water-related exposure data to complete a comprehensive drinking water assessment for methoxyfenozide at this time. However, in order to mitigate the potential for methoxyfenozide to leach into ground water or runoff to surface water, precautionary language has been incorporated into the proposed product label. Also, to the best of our knowledge, previous experience at EPA with more persistent and mobile pesticides for which there were available data to perform quantitative risk assessments demonstrated that drinking water exposure was typically a small percentage of the total dietary exposure. This observation holds even for pesticides detected in wells and drinking water at levels nearing or exceeding established MCLs. Considering the precautionary language on the label and our knowledge of previous experience with persistent chemicals, no risk from residues of methoxyfenozide in drinking water is anticipated.

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

D. Cumulative Effects

The methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way are not available at this time. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on

chemical-specific data, much of which may not be presently available.

Although at present the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides for which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed).

At this time, no data are available to determine whether methoxyfenozide benzoic acid, 3-methoxy-2-methyl-, 2-(3,5-dimethylbenzoyl)-2-(1,1-dimethylethyl) hydrazide has a common mechanism of toxicity with other substances. Thus, it is not appropriate to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, methoxyfenozide benzoic acid, 3-methoxy-2-methyl-,2-(3,5-dimethylbenzoyl)-2-(1,1-dimethylethyl) hydrazide does not produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, methoxyfenozide benzoic acid, 3-methoxy-2-methyl-,2-(3,5-dimethylbenzoyl)-2-(1,1-dimethylethyl) hydrazide is assumed not to have a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*—i. *Acute exposure and risk*. Since no acute endpoint of concern has been identified for methoxyfenozide, no acute risk assessment is required.

ii. *Chronic exposure and risk*. Using the conservative exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of methoxyfenozide from the proposed tolerances is 16.4% for the U.S. population. Aggregate exposure (food and water) are not expected to exceed 100%. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Rohm and Haas concludes that there is a reasonable certainty that no harm will result from aggregate exposure to methoxyfenozide residues to the U.S. population.

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

ii. *Developmental toxicity studies*—a. *Rats*. In a developmental toxicity study in rats, the maternal (systemic) NOAEL was 1,000 mg/kg/day HDT. The developmental (pup) NOAEL was > 1,000 mg/kg/day HDT.

b. *Rabbits*. In a developmental toxicity study in rats, the maternal (systemic) NOAEL was 1,000 mg/kg/day HDT. The developmental (pup) NOAEL was > 1,000 mg/kg/day.

iii. *Reproductive toxicity study rats*. In a multigeneration reproductive toxicity study in rats, the parental (systemic) NOAEL was 15.5 mg/kg/day, based on liver effects at the LOAEL of 153 mg/kg/day. The reproductive (pup) NOAEL was 1,552 mg/kg/day HDT. No adverse reproductive effects were observed.

iv. Prenatal and postnatal sensitivity—a. *Prenatal sensitivity*. The developmental NOAELs of > 1,000 mg/kg/day HDT from the developmental toxicity studies in rats and rabbits demonstrate that there is no developmental (prenatal) toxicity present for methoxyfenozide. Additionally, these developmental NOAELs are greater than 100-fold higher than the NOAEL of 9.8-10.0 mg/kg/day from the rat and dogs chronic studies which are the basis of the RfD.

b. *Postnatal sensitivity*. In the reproductive toxicity study in rats, the reproductive NOAEL (1,552 mg/kg/day) is about 100-fold higher than the parental NOAEL (15.5 mg/kg/day). These developmental and reproductive studies indicate that methoxyfenozide does not have additional prenatal and postnatal sensitivity for infants and children in comparison to other exposed groups.

3. *Acute exposure and risk*. No acute endpoint was identified for methoxyfenozide, and therefore, no acute risk assessment is required.

4. *Chronic exposure and risk*. For chronic dietary risk assessment, tolerances and anticipated residue values are used and the assumption that 100% of all leafy and cole crop vegetables (in addition to cotton, pome fruit, grapes, and fruiting vegetables) will contain residues at the tolerance levels. The percentage RfD utilized from the proposed tolerances is calculated using the DEEM (Version 6.74, licensed by Novigen Sciences Inc.) which uses USDA food consumption data from the 1994-1996 survey.

With the proposed tolerances for methoxyfenozide, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of methoxyfenozide is 29.9% for children 1–6 years old. Aggregate exposure (food and water) are not expected to exceed 100%. Rohm and Haas concludes that there is a reasonable certainty that no harm will result from aggregate exposure to methoxyfenozide residues to non-nursing infants.

F. International Tolerances

There are currently no CODEX, Canadian or Mexican maximum residue levels (MRLs) established for methoxyfenozide in leafy or cole crop vegetables so no harmonization issues are required for this action.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–6512–7]

Announcement and Publication of a Standard Letter To Be Sent to Parties Requesting a Prospective Purchaser Agreement (PPA); a Checklist of Information Generally Required Before a PPA Can Be Negotiated; and a Revised Model PPA Announced by EPA on October 1, 1999

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: To further promote the reuse of CERCLA sites, EPA is streamlining the process for evaluating and negotiating Prospective Purchaser Agreements (PPAs). On October 1, 1999, EPA issued a standard letter to be sent to parties requesting PPAs (Attachment A); a proposed checklist of information needed by EPA to evaluate requests (Attachment B); and a revised Model PPA (Attachment C).

The full text of these three documents follow.

FOR FURTHER INFORMATION CONTACT: For information on the letter and checklist, contact David Gordon in the Office of