degrade, decompose or depolymerize prior to, during, or after use.

5. TS-530 is not manufactured or imported from monomers and/or reactants that are not included on the TSCA substance inventory or manufactured under an applicable TSCA section 5 exemption.

6. TS-530 is not a water absorbing polymer with a number average molecular weight greater than or equal to 10,000.

7. TS-530 has an minimum-average molecular weight of 645,000 daltons. Substances with molecular weights greater than 400 generally are not absorbed through the intact skin, and substances with molecular weights greater than 1,000 generally are not absorbed through the GI tract. Chemicals not absorbed through the skin or GI tract generally are incapable of eliciting a toxic response.

8. TS-530 has a minimum-average molecular weight of 645,000 daltons. TS-530 meets the requirements for molecular weight distribution of oligomer contents of less than 5% with molecular weights less than 1,000 and less than 2% with molecular weights less than 500. Attached is a description of the molecular weight determination of TS-530.

Cabot Corporation believes that sufficient information has been submitted to assess the hazards of TS-530. No toxicology data are being submitted as the Agency does not generally require these data to rule on the exemption from the requirement of a tolerance for an inert ingredient. Because TS-530 conforms with the definition of a polymer and meets the criteria of a polymer under 40 CFR 723.250, Cabot Corporation believes there are no concerns for risks associated with toxicity.

C. Aggregate Exposure

1. *Dietary exposure.* TS-530 is not absorbed through the intact GI tract and is incapable of eliciting a toxic response.

2. *Drinking water.* TS-530 is not soluble in water and therefore there is no reason to expect human exposure to residues in water.

3. *Non-dietary exposure.* For most uses of TS-530 the primary route of exposure is dermal. TS-530 with a molecular weight significantly greater than 400 is not absorbed through the intact skin.

D. Cumulative Effects

Cabot Corporation believes that sufficient information has been submitted to assess the hazards of TS-530. Because TS-530 conforms with the definition of a polymer and meets the criteria of a polymer under 40 CFR 723.250, Cabot Corporation believes there are no concerns for risks associated with cumulative effects.

E. Safety Determination

1. *U.S. population.* Cabot Corporation believes that sufficient information has been submitted to assess the hazards of TS-530. Because TS-530 conforms with the definition of a polymer and meets the criteria of a polymer under 40 CFR 723.250, Cabot Corporation believes there are no concerns for risks associated with any potential exposure to adults.

2. *Infants and children.* Cabot Corporation believes that sufficient information has been submitted to assess the hazards of TS-530. Because TS-530 conforms with the definition of a polymer and meets the criteria of a polymer under 40 CFR 723.250, Cabot Corporation believes there are no concerns for risks associated with exposure to infants and children.

[FR Doc. 99–22049 Filed 8–24–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–882; FRL–6093–7]

Notice of Filing a Pesticide Petition to Establish a 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–882, must be received on or before September 24, 1999.

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'' section. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–882 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: James A. Tompkins, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–5697; and e-mail address: tompkins.james@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

Categories	NAICS	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 in the ''FOR FURTHER INFORMATION CONTACT'' section.

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–882. 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–882 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 5.1/6.1 or ASCII file format. All comments in electronic form must be identified by docket control number PF–882. 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 in the ''FOR FURTHER INFORMATION CONTACT'' section.

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

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

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

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

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

5. Provide specific examples to illustrate your concerns.

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

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

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data 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: August 10, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

1. Entek Corporation

PP 9F5095

EPA has received a pesticide petition (PP 9F5095) from Entek Corporation, 6835 Deerpath Road, Suite E, Elkridge, MD 21075 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180 by revising the existing tolerance regulation for glyphosate to allow application of glyphosate (in its acid form) on raw agricultural commodities (RAC). 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 nature of the residue in plants is adequately understood and consists of the parent, glyphosate and its metabolite, aminomethylphosphonic acid (AMPA). Only the glyphosate parent is to be regulated in plant and animal commodities since the metabolite AMPA is not of toxicological concern in food.

2. *Analytical method*. Glyphosate food residues can be measured using high pressure liquid chromatography

(HPLC) with fluorometric detection. This method is adequate for enforcement purposes; the methodology has been published in the pesticide analytical manual (PAM), Vol II. The method has a limit of detection (L0D) of 0.05 parts per million (ppm), which allows monitoring of food with residues at or above the tolerance levels.

3. *Magnitude of residues*. Adequate data concerning glyphosate residues on RAC has 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, this revision will permit glyphosate residues from the application of glyphosate in the acid form. 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.

B. Toxicological Profile

1. *Acute toxicity*. Glyphosate is practically non-toxic by the oral route of exposure since the acute oral LD_{50} for glyphosate is greater than 5,000 milligrams/kilograms (mg/kg).

2. *Genotoxicty*. Glyphosate was evaluated in the standard battery of mutagenicity screening tests and all assays were negative. The individual genotoxicity studies are: *in vitro* chromosomal aberration (no aberrations in Chinese hamster ovary cells were caused, with or without S9 activation); DNA repair in rat hepatocytes; *in vivo* bone marrow cytogenic test in rats; rec-assay with *B. subtilis*; reverse mutation test with *S. typhimurium*; Ames Test with *S. typhimurium*; and the dominant-lethal mutagenicity test in mice.

3. *Reproductive and developmental toxicity*. Developmental toxicity studies were conducted with glyphosate in the rat and mouse. In the rat study, test animals were orally given doses of 0, 300, 1,000 and 3,500 mg/kg/day of glyphosate. The maternal no-observable adverse effect level (NOAEL) is 1,000 mg/kg/day based on clinical signs of toxicity, body weight effects and mortality and the fetal NOAEL is 1,000 mg/kg/day based on reduced body weights and delayed sternebrae maturation at the highest dose tested (HDT), 3,500 mg/kg/day.

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 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 animals and decreased pup body weights and a reproductive NOAEL of 1,500 mg/kg/day.

4. *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.

5. *Chronic toxicity/carcinogenicity*. 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.

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.

6. *Animal metabolism*. The nature of the residue in animals is adequately understood and consists of the parent, glyphosate and its metabolite AMPA.

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

8. *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 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—Food*. Dietary exposure was estimated using the Theoretical Maximum Residue Contribution (TMRC) from all present tolerances. The TMRC is obtained by multiplying the tolerance level residue for each food commodity by consumption data which estimates the amount of those products eaten by various population subgroups. In conducting this exposure assessment, very conservative assumptions are made: 100% of these crops will contain glyphosate residues and the residues will be at the tolerance level.

2. *Drinking water*. In examining aggregate exposure, the Food Quality Protection Act (FQPA) directs EPA to consider available information concerning exposures from the pesticide residue via drinking water. The lifetime health advisory and maximum contaminant level (MCL) for glyphosate are both 700 parts per billion (ppb) in the EPA Office of Drinking Water's ''Drinking Water Health Advisory - Pesticides.'' The MCL represents the level at which no known or anticipated adverse health effects will occur allowing for an adequate margin of safety and is based on the reference dose (RfD). Environmental fate data for glyphosate shows little potential for the chemical to migrate to drinking water. In addition, glyphosate is not highly mobile and not persistent in soil or water.

Because the Agency lacks sufficient water-related exposure data to complete a comprehensive water risk assessment for many pesticides, EPA has commenced and nearly completed a process to identify a reasonable yet conservative boundary figure for the potential contribution of water-related exposures to the aggregate risk posed by a pesticide.

In developing a boundary figure, EPA estimated residue levels in water for a number of specific pesticides using various data sources. The Agency then applied the estimated residue levels, in conjunction with appropriate toxicological endpoints RfDs or acute dietary NOAELs and assumptions about body weight and consumption, to calculate, for each pesticide, the increment of aggregate risk contributed by consumption of contaminated water. While EPA has not yet pinpointed the appropriate bounding figure for consumption of contaminated water, the ranges the Agency is continuing to examine are all below the level that would cause glyphosate to exceed the RfD if the change in the tolerance expression being considered in this document is granted. The Agency has therefore concluded that the potential exposures associated with glyphosate in water, even at higher levels the Agency may consider a conservative upper bound, would not prevent the Agency from determining that there is a reasonable certainty of no harm if the proposed tolerance revision is granted.

3. *Non-dietary exposure*. Glyphosate is registered for use on several non-food sites such as around ornamentals, shade trees, shrubs, walks, driveways, flowerbeds, home lawns, farmstead including building foundations, along and in fences, in dry ditches and canals, along ditch banks, farm roads, shelter belts, forestry, Christmas trees, and industrial sites and other non-crop or industrial areas such as airports, lumber yards, manufacturing sites, utility substations, parking areas, petroleum tank farms and pumping stations. Margins of Exposure (MOEs) are determined for non-dietary exposure based on toxicological endpoints and measured or estimated exposures. Since glyphosate is not a carcinogen, the 21–day dermal study lacked any observable effects at the limit dose; and no adverse effects were observed in developmental toxicity studies in rats up to 1,000 mg/kg/day and rabbits up to 175 mg/kg/day, no toxicological endpoints are applicable. Because available data indicate no evidence of significant toxicity via the dermal or inhalation routes, MOE's were not calculated and risk assessments are not required for non-occupational residential uses.

D. Cumulative Effects

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 it in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on common mechanism of toxicity, glyphosate does not produce a toxic metabolite which is common to other substances.

For the purposes of this tolerance action, EPA has assumed that glyphosate does not have a common mechanism of toxicity with other substances. A condition of the registrations associated with these tolerances will be that registrants will provide common mechanism data in a timely manner when and if the Agency asks for it. After EPA develops methodologies for more fully applying common mechanism of toxicity issues to risk assessments, the Agency will develop a process to reexamine those tolerance decisions made earlier.

E. Safety Determination

1. *U.S. population*—i. *Acute dietary exposure*. Based upon available data, glyphosate does not pose any acute dietary risk and an acute dietary risk assessment is not required

ii. *Chronic dietary exposure*. Using the TAS Exposure I software and 1977-78 food consumption data, a chronic dietary exposure estimate has been performed based on 100% of the crops treated and all residues at tolerance levels. Based on this assessment, the total dietary exposure from glyphosate residues is 1.4% of the RfD for the overall U.S. population and 3.1% of the RfD for non-nursing infants, the most highly exposed population subgroup. As noted above, an additional risk assessment for residential uses was not required because there is no evidence of significant toxicity via dermal or inhalation exposure routes. Even though an appropriate bounding figure for consumption of contaminated water has not been determined, the ranges being examined are all below the level that would cause glyphosate to exceed the RfD. Generally, there is no concern for exposures below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicology data and the conservative exposure assessment employed, there is a reasonable certainty that no harm will occur from aggregate exposure to glyphosate.

2. *Infants and children*. FFDCA section 408 provides than an additional tenfold MOE (safety) for infants and children in the case of threshold effects may be needed to account for prenatal, and postnatal toxicity and the completeness of the data base unless it is determined that a different MOE (safety) will be safe for infants and children. Reliable data support using the standard MOE (usually 100x for combined interspecies and intraspecies variability), without the additional tenfold MOE, when a complete data base under current guidelines exists and when the nature of the findings from these studies do not raise concerns regarding the adequacy of the standard MOE.

The toxicological data base for evaluating prenatal and postnatal toxicity for glyphosate is considered to be complete at this time. Risk to infants and children for glyphosate was determined by using two developmental toxicity studies in rats and rabbits and a 2-generation reproduction study in rats. The developmental toxicity studies evaluate the potential for adverse effects on the developing organism resulting from exposure during prenatal development. The reproduction study provides information relating to effects from exposure to the chemical on the reproductive capability of both (mating) parents and on systemic toxicity, in addition to information on prenatal development. The results of these studies indicate that glyphosate does not produce birth defects and is not a reproductive toxin.

In the rabbit, no developmental toxicity was observed at the HDT where significant maternal toxicity occurred (death and clinical signs at 350 mg/kg/day). Because no developmental toxicity was observed at any dose level, the developmental toxicity NOAEL is considered to be 350 mg/kg/day. In the rat developmental toxicity study, severe

maternal (systemic) and developmental toxicity was noted at 3,500 mg/kg/day, the HDT. It is important to note that the HDT in this study was 3.5 times higher than the limit dose currently required by EPA guidelines. The maternal and developmental (pup) NOAEL was 1,000 mg/kg/day. No effects on reproductive parameters were observed.

In the rat 2-generation study, prenatal toxicity was observed at 1,500 mg/kg day as soft stools, decreased food consumption, and body weight gain; therefore, the systemic NOAEL is considered to be 500 mg/kg/day. Developmental (pup) toxicity was only exhibited at 1,500 mg/kg/day as decreased body weight gain of the F1a, F2a, and F2b male, and female pups during the second and third weeks of lactation. Glyphosate did not affect the ability of rats to mate, conceive, carry or deliver normal offspring at any dose level.

The RfD is based on the NOAEL for maternal toxicity in the rabbit developmental toxicity study. No developmental effects were noted in the study; effects were noted only at doses 20-fold higher than the NOAEL used for the RfD. No prenatal or postnatal effects were seen in any study in the absence of maternal toxicity. In the rat reproduction study, developmental effects were noted at doses 5 times higher than the NOAEL used for the RfD. The Agency does not believe the effects seen in these studies are of such concern to require an additional safety factor. Accordingly, the Agency believes the RfD has an adequate margin of protection for infants and children. The dietary exposure from current uses of glyphosate range from 1% of the RfD for nursing infants (less than 1–year old) to 3% for non-nursing infants and children 1 to 6 years old. Therefore, there is reasonable certainty that no harm will occur to infants and children from aggregate exposure to glyphosate.

F. International Tolerances

Codex MRLs have been established for residues of glyphosate in or on several raw agricultural commodities.

2. Novartis Crop Protection, Inc.

PP 4F4336 and PP 5F4469

EPA has received pesticide petitions (PP 4F4336 and PP 5F4469) from Novartis Crop Protection, Inc., P.O. Box 18300, Greensboro, NC 27419 proposing, pursuant to section 408(d) of FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR 180.481 by extending time-limited tolerances for residues of prosulfuron, 1-(4-methoxy-6-methyl-triazin-2-yl)-3-[2-(3,3,3-trifluoropropyl)-phenylsulfonyl]-urea in or on the RAC cereal grains group (except rice and wild rice) grain at 0.01 ppm; cereal grains group (except rice and wild rice) forage at 0.10 ppm; cereal grains group (except rice and wild rice) fodder at 0.01 ppm; cereal grains group (except rice and wild rice) straw at 0.02 ppm; cereal grains group (except rice and wild rice) hay at 0.20 ppm; milk at 0.01 ppm; and meat, fat, kidney, liver and meat byproducts of cattle, goats, hogs, horses, and sheep at 0.05 ppm until December 31, 2001. 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 prosulfuron in corn is well understood. Significant pathways involve oxidation of the phenyl ring to give 5-hydroxy prosulfuron, which is followed by sugar conjugation. Hydrolytic cleavage of the sulfonylurea bridge occurs for both prosulfuron and 5-hydroxy prosulfuron yielding the corresponding sulfonamide and triazine amine moieties. The sulfonamide metabolites are subsequently conjugated with sugars. Demethylation of the triazine amine results in the formation of the corresponding hydroxy triazine, which is further hydrolyzed at the amino group to form the dihydroxy triazine.

2. *Analytical method*. Novartis Crop Protection, Inc has submitted practical analytical methods for the detection and measurement of residue levels of prosulfuron in or on RAC and processed commodities of cereal grains, and for meat, milk and eggs. The LOD for prosulfuron is 0.8 ng injected and the limit of quantitation (LOQ) is 0.01 ppm for crop commodities, processed fractions and milk, and 0.05 ppm for meat and eggs. The method is based on commodity-specific cleanup procedures followed by determination by high performance liquid chromatography with ultraviolet (UV) detection.

3. *Magnitude of residues*. Complete, full geography residue programs, including processing, have been conducted on corn, wheat and grain sorghum. A three-level dairy animal feeding study to determine the transfer of residues of prosulfuron from animal feed commodities to meat and milk has also been conducted.

B. Toxicological Profile

1. *Acute toxicity*. Studies conducted with the technical material of prosulfuron:

i. Rat acute oral toxicity study with a LD_{50} of 949 mg/kg for males and 546 mg/kg for females.

ii. Rabbit acute dermal toxicity study with a $LD_{50} > 2,000$ mg/kg.

iii. Rat acute inhalation toxicity study with a $LC_{50} > 5.4$ milligrams per liter (mg/L).

iv. Rabbit eye irritation study showing slight irritation (Category III).

v. Rabbit dermal irritation study showing no irritation (Category IV).

vi. Guinea pig dermal sensitization study with the Buehler's method showing negative findings Guinea pig dermal sensitization study with the maximization method showing negative findings.

2. *Genotoxicty*. No genotoxic activity is expected of prosulfuron under *in vivo* or physiological conditions. The compound has been tested in a bacterial reverse gene mutation assay with and without metabolic activation using different *S. typhimurium* and *E. coli* stains; in a mammalian gene mutation study using V79 cells; in an *in vitro* mammalian cytogenetic test using Chinese hamster ovary cells with and without metabolic activation; in a micronucleus test in mice; and in a DNA-repair using freshly isolated rat liver hepatocytes. All these tests were negative for mutagenicity.

3. *Reproductive and developmental toxicity*. FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base. While the final review of some of additional developmental data has not yet been completed, based on the current toxicological data requirements, the data base on prosulfuron relative to prenatal and postnatal effects for children is considered to be complete.

In assessing the potential for additional sensitivity of infants and children to residues of prosulfuron, Novartis considered data from teratogenicity studies in the rat and the rabbit and a 2-generation reproduction studies in the rat. The teratogenicity studies are designed to evaluate adverse effects on the developing embryo as a result of chemical exposure during the period of organogenesis. Reproduction studies provide information on effects from chemical exposure on the reproductive capability of mating animals and systemic and developmental toxicity from *in-utero* exposure.

In the rat teratology study, prosulfuron was administered at dose levels of 0, 5, 50, 200 and 400 mg/kg during days 6 to 15 of gestation. Evidence of maternal toxicity (decreased body weight gain and reduced food consumption) and developmental toxicity (increased incidence of skeletal variations that was not significantly different from the historical control) was found at the maximum tolerated dose of 400 mg/kg. There was no evidence of teratogenicity at any dose, and the maternal and developmental no observed adverse effect levels (NOAELs) were established at 200 mg/kg.

Prosulfuron was administered to rabbits at dose levels of 0, 1, 10, and 100 mg/kg during days 7 to 19 of pregnancy. Maternal toxicity (decreased body weight gain and reduced food consumption) was observed in the 100 mg/kg dose group. There was no evidence of teratogenicity at any dose.

Since a range-finding rabbit teratology study had seen additional clinical findings and fetotoxicity at maternally toxic doses (≧ 150 mg/kg) but not in the definitive study at up to 100 mg/kg, a second rabbit teratology study was conducted at doses of 0, 20, 100, and 200 mg/kg/day. Maternal toxicity was observed at 200 mg/kg. The developmental NOAEL was 100 mg/kg and the maternal NOAEL was 20 mg/kg in this study. There was no evidence of teratogenicity at any dose.

Prosulfuron was administered to rats at dose levels of 0, 0.67, 13.3, 136, or 278 (males), and 0, 0.76, 15.3, 152, or 311 mg/kg/day (females) throughout two consecutive generations. The reproductive and systemic NOAELs of 13.3 mg/kg/day were based on decreased mean body weights and body weight gain observed at 136 mg/kg/day for both pups and parental animals. No treatment- related effects on reproductive performance (i.e., to produce, deliver or raise litters), litter sizes, viability of pups and necropsy findings in parental animals and offspring were noted up to the highest dose level.

Based on all these teratology and reproduction studies, the lowest NOAEL for developmental toxicity is estimated to be 13.3 mg/kg while the lowest NOAEL in the subchronic and chronic studies is 1.84 mg/kg/day (from the dog chronic study). Therefore, no additional sensitivity for infants and children to prosulfuron is suggested by the data base.

4. *Subchronic toxicity*. The liver was identified as a target organ at high doses in the rat, mouse and dog as indicated by slightly increased liver enzymes and liver weights. No histomorphologic correlates of liver damage was noted in the 90–day studies except in the mouse study where centrilobular hypertrophy was found in males at feeding levels ≧ 1,750 ppm and in females at levels ≧ 3,500 ppm. Effects of prosulfuron on the hematopoietic system (anemia) were noted in the dog at feeding levels ≧ 1,500 ppm and myocardial lesions were found in the 3,000 ppm animals in the 90–day study. Myocardial vacuolative degeneration was observed in male mice fed prosulfuron for 90 days at levels ≧ 3,500 ppm and in females at 7,000 ppm.

In general, NOAELs for target organ effects were established at doses that were much higher than overall study NOAELs, which were based on other indicators of toxicity such body weight gain.

5. *Chronic toxicity*. Prosulfuron was fed to dogs at dosages of 0, 0.33, 1.95, 18.6 or 41.0 mg/kg/day (males) and 0, 0.31, 1.84, 20.2, or 48.8 mg/kg/day (females) for 1–year. The NOAEL was 1.84 mg/kg/day based on hematologic and clinical chemistry effects and incidence of lipofuscin accumulation in the liver at 18.6 mg/kg/day.

In an 18–month mouse carcinogenicity study, prosulfuron was administered at dose levels of 0, 1.71, 81.4, 410 or 832 mg/kg/day (males), and 0, 2.11, 100, 508 or 1,062 mg/kg/day (females). There was no evidence of carcinogenic effects up to 1,062 mg/kg/day, the HDT. The NOAEL was 1.71 mg/kg/day in males, and 100 mg/kg/day in females based on increased incidence/severity of centrilobular hepatocellular hypertrophy. A 2–year chronic feeding/carcinogenicity study in rats fed dosages of 0, 0.4, 7.9, 79.9 or 160.9 (males), and 0, 0.5, 9.2, 95.7 or 205.8 mg/kg/day (females) was conducted. There was uncertain evidence of carcinogenicity with slight increases in the incidence of mammary gland adenocarcinomas in females at 95.7 and 205.8 mg/kg/day, slight increase in incidence of benign testicular interstitial cell tumors at 79.9 and 160.9 mg/kg/day (significant trend only). A systemic NOAEL of 7.9 mg/kg/day was based on decreased body weight and body weight gain, hematopoietic effects (males), and possibly increased serum GGT and decreased liver, kidney and adrenal weights (females) at 79.9 mg/kg/day.

6. *Carcinogenicity*. The HED RfD/Peer Review Committee (PRC) classified this chemical as a Class D oncogen based on the conclusion that there was uncertain evidence of carcinogenicity. No relevant treatment-related oncogenic potential was observed in rats and mice. The slightly increased incidence of testicular interstitial cell tumors in male rats at ≧ 79.9 mg/kg/day and the slightly increased incidence of mammary gland adenocarcinoma in females at ≧ 95.7 mg/kg/day was not significant when the increased survival in these groups was considered. Furthermore, the overall incidence of mammary tumors was essentially identical in all groups including the control. There was no evidence of carcinogenicity in mice and dosages in both studies were sufficient for identifying a cancer risk. In the absence of carcinogenicity, it is appropriate that a RfD approach be used for quantitation of human risks.

7. *Animal metabolism*. Prosulfuron is rapidly absorbed from the gastrointestinal tract of rats and is rapidly excreted. Approximately 90% of the administered dose is excreted during the first 48 hours, predominately via urine. Tissue residues are low. Prosulfuron is metabolized primarily via hydroxylation at side chain and phenyl ring positions and O-demethylation of the triazyl methoxy group. Minor pathways include unsaturation of the trifluoropropyl side chain, hydrolysis of the phenylsulfonylurea bridge and oxidative/hydrolytic cleavage of the triazine ring system.

In the goat, the orally administered prosulfuron is quickly eliminated primarily via the urine as prosulfuron. The metabolism of prosulfuron in the goat follows a similar pathway as observed in the rat although not as extensive. Accordingly, the biotransformation is limited to oxidation of the triazinyl methyl, O-demethylation of the triazinyl methoxy group and hydrolytic cleavage of the sulfonylurea bridge. The majority of the residues were accounted for as prosulfuron, the triazine amine, which results from bridge hydrolysis (CGA-150829) and the triazinyl hydroxymethyl metabolite (CGA-273437). In the hen, metabolism is similar to that observed in the rat and goat. The major residues found in edible tissues and eggs were prosulfuron, and the triazine amine (CGA-150829) and the sulfonamide (CGA-159902) which results from hydrolysis of the sulfonylurea bridge. In conclusion, the major metabolic pathways in the rat, goat, and hen are similar.

7. *Metabolite toxicology*. Metabolic pathways of prosulfuron in plants and animals are comparable and no (detectable) residues are found in or on crops. All relevant plant metabolites are observed in the animals and are thus toxicologically covered. The remaining plant metabolites are toxicologically insignificant. Therefore, no analytical method for metabolite determination is necessary for routine residue monitoring. For enforcement purposes

(e.g., high overdose or application too close to harvest) parent prosulfuron is the appropriate compound for analytical monitoring.

8. *Endocrine disruption*. Prosulfuron does not belong to a class of chemicals known for having significant adverse effects on the endocrine system. Developmental toxicity studies in rats and rabbits and reproduction study in rats gave no indication that prosulfuron might have any effects on endocrine function related to development and reproduction. The subchronic and chronic studies also showed no evidence of a long-term effect related to the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. Acute and Chronic dietary exposure assessments were conducted for prosulfuron using tolerance values published in 40 CFR part 180.481. In both assessments it was assumed that 100% of all corn and cereal grains were treated with prosulfuron (100% market share). The exposure analyses was conducted using food consumption data from USDA's 1994-96 Continuing Survey of Intake by Individuals (CSFII) and Novigen Sciences, Inc. Dietary Exposure Evaluation Model (DEEMTM). Chronic exposure was compared to a RfD of 0.02 mg/kg based on a NOAEL of 1.84 mg/kg in a dog feeding study and a 100-fold uncertainty factor. This exposure analysis showed that the U.S. population had an exposure of less than 1% of the chronic RfD. The most sensitive sub-population was children (1-6 years old) with a chronic exposure of 2.4%.

Acute exposure was compared to an acute RfD of 0.1 mg/kg, which was based on a NOAEL of 10 mg/kg from an acute neurotoxicity study in the rat and a 100-fold uncertainty factor. The most sensitive sub-population was all infants with an exposure of 2.2% of the acute RfD. The U.S. population showed an exposure of 1.5% of the RfD.

These results show that there is more than a reasonable certainty of no harm, through exposure to prosulfuron residues in the diet.

ii. *Drinking water—estimated surface drinking water concentrations*. The GENEEC estimated surface water concentrations of prosulfuron were 1.86 ppb on the Peak Day-0 and 1.40 ppb on the average 56–day. According to the EPA ''OPP's Interim Approach for Addressing Drinking Water Exposure,'' the average 56–day value is divided by three when correcting for overestimation of the GENEEC model. This resulted in a corrected potential drinking water exposure via surface water of 1.40 ppb / 3 =0.4667 ppb.

These concentrations were not adjusted for the estimated market share, regional soil characteristics or percentage of use area. The Peak Day-0 estimate, 1.86 ppb, was used in the acute exposure analysis and the corrected 56–day drinking water concentration of 0.4667 ppb was used in the chronic exposure analysis.

iii. *Estimated ground water concentrations*. The SCI-GROW estimated ground water concentration for the prosulfuron uses of 0.406585 ppb contributed little to the overall exposure. The estimated concentrations were not adjusted for the estimated market share or percentage of use area.

2. *Drinking water levels of concern*—

i. *Acute exposure*. The estimated ground and surface water concentrations of prosulfuron contributed little to the potential acute human exposure. The acute drinking water levels of concern ($DWLOC_{acute}$) for prosulfuron were based on the acute RfD, a MOE, the 99.9th percentile of the acute dietary exposure for U.S. population subgroups and the body weight - daily water consumption of each respective subgroup. The acute RfD of prosulfuron was 10 mg/kg/day based on the findings from the acute neurotoxicity rat study. The lowest MOE for any pesticide is 100 and this was used as a conservative approach. The dietary exposure estimates for subgroups of the U.S. population included the U.S. population all seasons, all infants (<1–year), nursing infants (< 1–year), non-nursing infants (< 1–year), children (1–6 years) and children (7-12 years). The dietary exposure estimates for all the subgroups were less than 0.0023 mg/kg/bodyweight/day. The calculated $DWLOC_{acute}$ values for these respective subgroups were 3447, 987, 980 and 978 ppb.

The estimated ground water concentration (0.406585 ppb) and the peak day-0 surface water concentration (1.86 ppb) of prosulfuron did not exceed the $DWLOC_{acute}$ values. Therefore, there is reasonable certainty that the residues of prosulfuron in the drinking water would not result in unacceptable levels of acute aggregate human health risk, and that such exposure would not exceed the exposure allowable by the risk cup.

ii. *Chronic exposure*. The estimated ground and surface water concentrations of prosulfuron contributed little to the potential chronic human exposure. The chronic (non-cancer) drinking water levels of concern ($DWLOC_{chronic}$) for prosulfuron were based on the chronic RfD, any estimated residential exposure, the chronic dietary exposure for select U.S. population subgroups and the body weight - daily water consumption of each respective subgroup. The chronic RfD for prosulfuron was 0.02 mg/kg/bwt/day based on the findings of a chronic dog study. There was no estimated residential exposure from the prosulfuron uses. The potential chronic dietary exposure estimates were calculated for the same subgroups selected for the acute exposure analysis. These potential chronic dietary values included 0.00172 mg/kg/bwt/day for the United States all seasons and < 0.0005 for the remaining subgroups (non-nursing infants < 1–year, children 1–6 years, and children 7–12 years). The calculated $DWLOC_{chronic}$ values for the respective subgroups were 694, 198, 195 and 197 ppb.

The GENEEC estimated concentration of prosulfuron in surface water at the average 56–day was 1.40 ppb. According to EPA ''OPP's Interim Approach for Addressing Drinking Water Exposure,'' the average 56–day value is divided by three when correcting for overestimation of the GENEEC model. This resulted in a corrected surface drinking water concentration of 1.40 ppb/3 = 0.4667 ppb.

The estimated ground water concentration (0.406585 ppb) and the corrected average 56–day surface water concentration (0.4667 ppb) of prosulfuron did not exceed the $DWLOC_{chronic}$ values. Therefore, there is reasonable certainty that the residues of prosulfuron in the drinking water would not result in unacceptable levels of chronic aggregate human health risk, and that such exposure would not exceed the exposure allowable by the risk cup.

3. *Non-dietary exposure*. Non-dietary exposure to prosulfuron is considered negligible as the chemical is registered for agricultural use only. For workers handling this chemical, acceptable MOE (in the range of thousands) have been obtained for both acute and chronic scenarios.

D. Cumulative Effects

Consideration of a common mechanism of toxicity is not appropriate at this time since there is no information to indicate that toxic effects produced by prosulfuron would be cumulative with those of any other types of chemicals. Furthermore, the triazine containing sulfonyl-urea is a new type of herbicide and no compound in this general chemical class currently has a significant market share. Consequently, it is considered appropriate to only

include the potential exposure to prosulfuron in an aggregate risk assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data base for prosulfuron, Novartis has calculated aggregate exposure levels for this chemical. The calculation shows that less than 1% of the RfD will be utilized for the U.S. population based on chronic toxicity endpoints. 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. Novartis concludes that there is a reasonable certainty that no harm will result from aggregate exposure to prosulfuron residue.

2. *Infants and children*. No adverse effects on the ability to produce, deliver or rear offspring was observed in a 2-generation study in rats. Likewise, teratogenicity studies in rats and rabbits did not reveal any teratogenic, embryotoxic or fetotoxic potential of prosulfuron. The lowest observed adverse effect level (LOAEL) for developmental toxicity was established in the rat reproduction study at 13.3 mg/kg, which is higher than the chronic NOAEL of 1.84 mg/kg, on which the RfD is based.

Using the same conservative exposure assumptions as employed for the determination in the general population, Novartis has calculated that the percent of the RfD that will be utilized by aggregate exposure to residues of prosulfuron is only 2.4% for children (1–6 years old), the most impacted sub-population. Therefore, based on the completeness and reliability of the toxicity data base and the conservative exposure assessment, Novartis concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to prosulfuron residues.

F. International Tolerances

No Codex MRLs have been established for residues of prosulfuron.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–50861; FRL–6092–5]

Issuance of Experimental Use Permits

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has granted experimental use permits (EUPs) to the following pesticide applicants. An EUP permits use of a pesticide for experimental or research purposes only in accordance with the limitations in the permit.

FOR FURTHER INFORMATION CONTACT: By mail: Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

In person or by telephone: Contact the designated person at the following address at the office location, telephone number, or e-mail address cited in each experimental use permit: 1921 Jefferson Davis Highway, Arlington, VA.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does This Notice Apply to Me?

This notice is directed to the public in general. Although this action may be of particular interest to those persons who conduct or sponsor research on pesticides, the Agency has not attempted to describe all the specific entities that may be affected by this action. If you have any questions regarding the information in this notice, consult the designated contact person listed for the individual EUP.

B. How Can I Get Additional Information or Copies of This Document or Other Documents?

You may obtain electronic copies of this document from the EPA Internet Home Page at http://www.epa.gov/. 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/.

II. EUPs

EPA has issued the following EUPs:

241–EUP–141. Extension/Amendment. American Cyanamid Company, P.O. Box 400 Princeton, NJ 08543–0400. This experimental use permit allows the use of 1,600 pounds of the termiticide 4-bromo-2-(4-chlorophenyl)-1-(ethoxymethyl)-5-(trifluoromethyl)-1-pyrrole-3-carbonitrile, chlorfenapyr on 463 sites (structures) to evaluate the control of termites and other wood boring insects. The program is authorized only in the States of Alabama, Arizona, Arkansas, California, Connecticut, Delaware, District of Columbia, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Kentucky, Louisiana, Maryland, Massachusetts, Michigan, Mississippi, Missouri, Nebraska, New Jersey, New York, North Carolina, Ohio, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Virginia, and Washington. The experimental use permit is effective from June 30, 1999 to November 30, 2000. (Ann Sibold; Rm. 220, Crystal Mall #2; telephone: 703–305–6502; e-mail address: sibold.ann@epa.gov)

62719–EUP–44. Issuance. Dow AgroSciences LLC, 9330 Zionsville Road, Indianapolis, Indiana 46268–1054. This experimental use permit allows the use of 3,379,758 pounds of the soil fumigants 1,3-dichloropropene and chloropicrin from the product InLine on 15,000 acres of soil, treated using drip irrigation systems only, to be planted in cauliflower, cucumbers, eggplant, lettuce, melons, onions, peppers, pineapples, squash, strawberries, and tomatoes to evaluate the control of nematodes, symphylans, and certain soil-borne diseases. The program is authorized only in the States of Alabama, Arizona, California, Colorado, Florida, Georgia, Hawaii, Idaho, Oregon, and Washington. The experimental use permit is effective from June 25, 1999 to June 25, 2002. (Mary L. Waller; Rm. 249, Crystal Mall #2; telephone: (703) 308–9354; e-mail address: waller.mary@epa.gov)

Persons wishing to review these EUPs are referred to the designated contact person. Inquiries concerning these permits should be directed to the persons cited above. It is suggested that interested persons call before visiting the EPA office, so that the appropriate file may be made available for inspection purposes from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Experimental use permits.

Dated: August 10, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 99–21549 Filed 8–24–99; 8:45 am]

BILLING CODE 6560–50–F