(trifluoromethyl)-1,3,4-thiadiazol-2-yl]oxy]acetamide.

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

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are available for public inspection in the Public Information and Records Intregrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Rm. 119, CM #2, Arlington, VA 22202 (703–305–5805). Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of Information Office (A-101), 401 M St., SW., Washington, D.C. 20460. Such requests should: (1) Identify the product name and registration number and (2) specify the data or information desired.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: May 13, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 98–14161 Filed 5–28–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–806; FRL–5791–2]

Monsanto Company; Pesticide Tolerance Petitions Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–806, must be received on or before June 29, 1998.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

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

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: James A. Tompkins, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW, Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 239, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–5697; e-mail: tompkins.james@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: 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.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–806] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

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

List of Subjects

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

Dated: May 14, 1998.

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. Monsanto Company

PP 8F4937

EPA has received a pesticide petition (PP 8F4937) from Monsanto Company, 700 14th St., NW., Suite 1100, Washington, DC 20005. proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of

halosulfuron-methyl: methyl 5-[(4,6-dimethoxy-2-pyrimidinyl)amino] carbonyl aminosulfonyl-3-chloro-1-methyl-1H-pyrazole-4-carboxylate in or on the raw agricultural commodity undelinted cotton seed & cotton gin by-products at 0.05 parts per million (ppm), rice grain at 0.05 ppm, rice straw at 0.20 ppm, tree nut group (Group 14) nutmeat at 0.05 ppm and hulls at 0.20 ppm, pistachio, nutmeat at 0.05 ppm, pistachio, hulls at 0.2 ppm.

In addition, Monsanto proposes the establishment of tolerances for halosulfuron methyl (as parent only) in or on the following raw agricultural commodities:

Corn, field: grain at 0.05 ppm, forage at 0.2 ppm, and fodder at 0.8 ppm.

Grain, sorghum (milo): grain at 0.05 ppm, forage at 0.05 ppm, and fodder/stover at 0.10 ppm.

Monsanto also proposes removing 40 CFR 180.479 (b) which reads as follows:

Indirect or inadvertent tolerances. Tolerances are established for indirect or inadvertent residues of the herbicide halosulfuron-methyl and its metabolites determined as 3-chloro-1-methyl-5-sulfamoylpyrazole-4-carboxylic acid and expressed as parent equivalents, in or on the following raw agricultural commodities when present therein as a result of the application of halosulfuron-methyl to growing crops.

Soybean, forage at 0.5 ppm, soybean, hay at 0.5 ppm, soybean, seed at 0.5 ppm, wheat, forage at 0.1 ppm, wheat, grain at 0.1 ppm. and wheat, straw at 0.2 ppm.

EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of halosulfuron-methyl as well as the nature of the residues in plants is adequately understood for purposes of these tolerances. Metabolism studies were conducted in three crops, viz.; field corn, sugarcane and soybeans. Metabolism depends on the mode of application. Preemergent applications result in rapid soil degradation of halosulfuron-methyl followed by crop uptake of the resulting pyrazole moiety. The pyrimidine ring binds tightly to soil and is eventually converted to carbon dioxide by microbial degradation. In postemergent applications, little metabolism and translocation take place resulting in unmetabolized parent compound as the major residue on the directly treated foliar surfaces. Very low residue levels of the metabolite 3-chloro-1-methyl-5-sulfamoylpyrazole-4-carboxylic acid (3-CSA) are found in the grain.

2. *Analytical method*. A practical analytical method, gas chromatography with an electron- capture detector which detects and measures total residues (halosulfuron-methyl and metabolites) is available for enforcement purposes with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances. This enforcement method has been submitted to the Food and Drug Administration for publication in the Pesticide Analytical Manual, Vol. II (PAM II). It has undergone independent laboratory validation and validation at the Beltsville laboratory. The Analytical Chemistry section of the EPA concluded that the method is adequate for enforcement. Analytical method is also available for analyzing meat by-products which also underwent successful independent laboratory and Beltsville laboratory validations.

3. *Magnitude of residues*. In the tree nut residue study, there were no quantifiable residues found in nut meats using an analytical method with limit of quantitation (LOQ) of 0.05 ppm. Residues ranging from <0.05 to 0.154 ppm were found in almond hulls when treated at 1.4 times the recommended rate. There were no detectable residues found in cotton undelinted seed as well as from the resulting processed commodities even at treatment rates of more than 5 times the maximum recommended rate per season. No quantifiable residues were found in cotton gin byproducts. The residues in the rice grain and rice processed fractions were below the limit of detection of 0.02 ppm at all locations. 5 of the 18 sites showed residues in rice straw ranging from 0.06 to 0.17 ppm while 13 sites had non-quantifiable residues (<0.05 ppm). Results of the aquatic sediment dissipation study showed that the parent and major metabolite residues dissipated rapidly in both soil and water phases with DT_{50} values of 1.3 and 1.87 days and DT_{90} of 6.48 and 12 days from 2 sites, respectively. The half-life of halosulfuron-methyl in the paddy water phase is calculated to be 0.87 days following direct application to water. The vertical mobility is not a major route of dissipation. The residues (parent and metabolites that are hydrolyzable to 3-CSA) dissipated rapidly in the upper soil layer but showed no indication of significant downward movement into the lower soil layers.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicological studies placing the technical-grade halosulfuron-methyl in Toxicity Category III. A 90-day feeding study in rats resulted in a lowest-observed-effect-level (LOEL) of 497 milligrams/kilograms/day (mg/kg/day) in males and 640 mg/kg/day in females, and a no-observed-effect-level (NOEL) of 116 mg/kg/day in males and 147 mg/kg/day in females.

2. *Genotoxicty*. Bacterial/mammalian microsomal mutagenicity assays were performed and found not to be mutagenic. Two mutagenicity studies were performed to test gene mutation and found to produce no chromosomal aberrations or gene mutations in cultured Chinese hamster ovary cells. An in *vivo* mouse micronucleus assay did not cause a significant increase in the frequency of micronucleated polychromatic erythrocytes in bone marrow cells. A mutagenicity study was performed on rats and found not to induce unscheduled DNA synthesis in primary rat hepatocytes.

3. *Reproductive and developmental toxicity*. A developmental toxicity study in rats resulted in a developmental LOEL of 750 mg/kg/day, based on decreases in mean litter size and fetal body weight, and increases in resorptions, resorptions/dam, post-implantation loss and in fetal and litter incidences of soft tissue and skeletal variations, and a developmental NOEL of 250 mg/kg/day. Maternal LOEL was 750 mg/kg/day based on increased incidence of clinical observations, reduced body weight gains, and reduced food consumption and food efficiency. The maternal NOEL was 250 mg/kg/day.

A developmental toxicity study in rabbits resulted in a developmental LOEL of 150 mg/kg/day, based on decreased mean litter size and increases in resorptions, resorptions/dam and post-implantation loss, and a developmental NOEL of 50 mg/kg/day. The maternal LOEL was 150 mg/kg/day based on reduced body weight gain and reduced food consumption and food efficiency. The maternal NOEL was 50 mg/kg/day.

A dietary 2-generation reproduction study in rats resulted in parental toxicity at 223.2 mg/kg/day in males and 261.4 mg/kg/day in females in the form of decreased body weights, decreased body weight gains, and reduced food consumption during the premating period. Very slight effects were noted in body weight of the offspring at this dose. This effect was

considered to be developmental toxicity (developmental delay) rather than a reproductive effect. No effects were noted on reproductive or other developmental toxicity parameters. The systemic/ developmental toxicity LOEL was 223.2 mg/kg/day in males and 261.4 mg/kg/day in females; the systemic/ developmental toxicity NOEL was 50.4 mg/kg/day in males and 58.7 mg/kg/day in females. The reproductive LOEL was greater than 223.2 mg/kg/day in males and 261.4 mg/kg/day in females; the reproductive NOEL was equal to or greater than 223.2 mg/kg/day in males and 261.4 mg/kg/day in females.

4. *Subchronic toxicity*. A 21-day dermal toxicity study in rats resulted in a NOEL of 100 mg/kg/day in males and greater than 1,000 mg/kg/day in females. The only treatment-related effect was a decrease in body weight gain of the 1,000 mg/kg/day group in males.

5. *Chronic toxicity*. A 1-year chronic oral study in dogs resulted in a LOEL of 40 mg/kg/day based on decreased weight gain and a NOEL of 10 mg/kg/day for systemic toxicity. A 78-week carcinogenicity study was performed on mice. Males in the 971.6 mg/kg/day group had decreased body weight gains and an increased incidence of microconcretion/mineralization in the testis and epididymis. No treatment-related effects were noted in females. Based on these results, a LOEL of 971.9 mg/kg/day was established in males and NOELs of 410 mg/kg/day in males and 1,214.6 mg/kg/day in females were established. The study showed no evidence of carcinogenicity. A combined chronic toxicity/ carcinogenicity study in rats resulted in a LOEL of 225.2 mg/kg/day in males and 138.6 mg/kg/day in females based on decreased body weight gains, and a NOEL of 108.3 mg/kg/day in males and 56.3 mg/kg/day in females. The study showed no evidence of carcinogenicity.

6. *Animal metabolism*. EPA stated that the nature of the residue in ruminants was determined to be adequately understood. In the tissues and milk of goats, the major extractable residue was the unmetabolized parent compound. Based on the low residues of the parent compound in corn grain and the low transfer of residues in the metabolism study, tolerances on poultry products were not required. In the rat metabolism study, parent compound was absorbed rapidly but incompletely. Excretion was relatively rapid at all doses tested with majority of radioactivity eliminated in the urine and feces by 72 hours. Fecal elimination of parent was apparently the result of unabsorbed parent.

7. *Metabolite toxicology*. The toxicology studies listed below were conducted with the 3-CSA metabolite. Based on the toxicological data of the 3-CSA metabolite, EPA concluded that it has lower toxicity compared to the parent compound and that it should not be included in the tolerance expression. The residue of concern is the parent compound only.

i. A 90-day rat feeding study resulted in a LOEL in males of >20,000 ppm and a NOEL of 20,000 ppm (1,400 mg/kg/day). In females, the LEL is 10,000 ppm (772.8 mg/kg/day) based on decreased body weight gains and a NOEL of 1,000 ppm (75.8 mg/kg/day).

ii. A developmental toxicity resulted in a LOEL for maternal toxicity of >1,000 mg/kg/day based on the absence of systemic toxicity, a NOEL of 1,000 mg/kg/day. The developmental LOEL is >1,000 mg/kg/day and the NOEL is 1,000 mg/kg/day.

iii. The microbial reverse gene mutation did not produce any mutagenic effect while the mammalian cell gene mutation/chinese hamster ovary cells did not show a clear evidence of mutagenic effect in the Chinese hamster ovary cells.

iv. The mouse micronucleus assay did not show any clastogenic or aneugenic effect.

8. *Endocrine disruption*. No specific tests have been conducted with halosulfuron-methyl to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there were no significant findings in other relevant toxicity tests, i.e., teratology and multi-generation reproduction studies, which would suggest that halosulfuron-methyl produces effects characteristic of the disruption of the estrogenic hormone.

C. Aggregate Exposure

1. *Dietary exposure*— i. *Food*. For purposes of assessing the potential dietary exposure from food under existing tolerances, aggregate exposure based on the Theoretical Maximum Residue Contribution (TMRC) which is an estimate of the level of residues consumed daily if each food item contained pesticide residues equal to the tolerance. The calculated TMRC value was 0.0005 mg/kg body weight/day for the general US population which will utilize only 0.51% of the Reference Dose (RfD) for established tolerances for halosulfuron-methyl and its metabolites in/on raw agricultural commodities of field corn, grain sorghum (milo) and secondary tolerances in meat and meat byproducts (cattle, goats, hogs, horses, and sheep). TMRC is obtained by multiplying the tolerance levels for each commodity by the average daily consumption of the food forms of that commodity eaten by the U.S. population and various population subgroups. In conducting this exposure assessment, conservative assumptions were made, e.g., 100% of all commodities will contain halosulfuron-methyl residues and those residues would be at the level of their respective tolerances. This results in a large overestimate of human exposure. Monsanto conducted another dietary exposure analysis to include food from crops in subsequent petitions including this petition. This analysis added dietary exposure from the following raw agricultural commodities using the proposed tolerance levels of each commodity, viz.; sweet corn (kernel + cobs with husks removed at 0.05 ppm, forage at 0.2 ppm, fodder/stover at 0.8 ppm), pop corn (grain at 0.05 ppm, fodder/stover at 0.8 ppm), sugarcane (cane at 0.05 ppm), tree nut crop grouping (nut meat at 0.05 ppm, hulls at 0.2 ppm), pistachio nuts (nutmeat at 0.05 ppm, hulls at 0.2 ppm), cotton (undelinted seed at 0.05 ppm, gin byproduct at 0.2 ppm) and rice (grain at 0.05 ppm and straw at 0.2 ppm). Food consumption data from the USDA Nationwide Food consumption survey for 1989-1992 and the EXPOSURE-1 software by TAS, Inc. were used in the calculation. Even with the same conservative assumptions, the potential dietary exposure to halosulfuron-methyl from consumption of products for which it is currently labeled and proposed resulted in a TMRC of 0.00064 mg/kg body weight/day and represents only 0.6% of the RfD for the general U.S. population. Field corn and sorghum forage and fodder are fed to animals, thus exposure of humans to residues from these commodities might result if such residues are transferred to meat, milk, poultry or eggs. However, based on the results of animal metabolism and the amount of halosulfuron-methyl expected in animal feeds, Monsanto concludes that there is no reasonable expectation that residues of halosulfuron-methyl will exceed existing tolerances in meat. The regulation of animal commodities and poultry products are not required.

ii. *Drinking water*. There is no Maximum Contaminant Level (MCL) established for residues of halosulfuron-methyl. It is not listed for MCL development or drinking water monitoring under the Safe Drinking Water Act nor is it a target of EPA's National Survey of Wells for Pesticides.

Monsanto is not aware of any halosulfuron-methyl detections in any wells, ponds, lakes or streams resulting from its use in the United States. A Lifetime Health Advisory Level (HAL), calculated using EPA procedures, may be used as a preliminary acceptable level in drinking water. The calculated level is 700 ppb which assumes a 20% relative contribution from water and which is sufficient to provide ample margins of safety. In addition, EPA has concluded that potential levels of halosulfuron-methyl or metabolites in soil and water do not appear to have significant toxicological effects on humans or animals and presents a negligible risk.

The EPA has expressed concern regarding potential groundwater contamination by the sulfonylurea (SU) class of chemistry in general and has required generic label warnings for halosulfuron-methyl; however, results of the field dissipation and lysimeter studies and a recently completed aquatic sediment study with halosulfuron-methyl should mitigate the concern for this chemical in particular.

Based on the very low level of mammalian toxicity, lack of other toxicological concerns and low use rates, Monsanto believes that there is reasonable certainty that no harm will result from exposure to halosulfuron-methyl via drinking water sources.

iii. *Non-dietary exposure*. Halosulfuron-methyl is labeled for use on commercial and residential turf and other non-crop sites which could have minimal opportunity for exposure. The agricultural uses including the proposed uses in tree nut crop group, pistachio nuts, cotton and rice will not increase the non-occupational exposure appreciably, if at all. Any exposure to halosulfuron-methyl resulting from turf use will result from dermal exposure during application and will be limited because of low use rates. In the 21-day dermal study, no treatment related adverse effects were observed and the NOAEL was determined to be greater than the highest dose tested, >1,000 mg/kg. Halosulfuron-methyl is non-volatile with a vapor pressure of $<1 \times 10^{-7}$ mm Hg, hence, inhalation exposure during and after application will not add significantly to aggregate exposure. Based on the physical and chemical characteristics, low use rates, low acute toxicity and lack of other toxicological concerns, Monsanto believes that the risk posed by non-occupational exposure to halosulfuron-methyl is minimal.

D. Cumulative Effects

Halosulfuron-methyl belongs to the sulfonyl urea class of chemistry. The mode of action of halosulfuron-methyl is the inhibition of the plant enzyme aceto lactase synthetase (ALS), which is essential for the production of required amino acid in plants. Although other registered sulfonyl ureas may have similar herbicidal mode of action, there is no information available to suggest that these compounds exhibit a similar toxicity profile in the mammalian system that would be cumulative with halosulfuron-methyl. Thus, consideration of a common mechanism of toxicity is not appropriate at this time. Monsanto is considering only the potential risks of halosulfuron-methyl in its aggregate exposure assessment.

E. Safety Determination

1. *U.S. population—Chronic dietary exposure*. As stated above, the EPA's calculated aggregate chronic exposure to halosulfuron-methyl from the established tolerances for field corn and grain sorghum raw agricultural commodities utilizes only 0.51% of the RfD using very conservative assumptions. Monsanto's subsequent calculation to include the proposed tolerances on sweet corn, pop corn, sugarcane, tree nut crop grouping, pistachio nuts, rice and cotton estimates that it will utilize only 0.6% of the RfD for the entire U.S. population. 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. Toxicology data indicating low potential for mammalian toxicity and lack of other toxicity concerns plus the conservative assumptions used in this calculation support the conclusion that there is a ''reasonable certainty of no harm'' to the U.S. population in general from aggregate exposure to halosulfuron-methyl residues from all anticipated dietary exposures and all other non-occupational exposures.

2. *Acute dietary exposure*. The detailed DRES acute exposure analysis evaluates individual food consumption and estimates the distribution of single day exposures through the diet for the US population and certain subgroups. Since the toxicological effect to which high end exposure is compared is developmental toxicity, EPA determined that the DRES subgroup of concern is females (13+ years) which approximates women of child-bearing age. The appropriate NOEL to use to assess safety in acute exposure is 50 mg/kg body weight/day from a developmental toxicity study in rabbits.

For shorter term risk, the Margin of Exposure (MOE), a measure of how closely the high end exposure comes to the NOEL and is calculated as a ratio of the NOEL to the exposure (NOEL/exposure = MOE). For toxicological endpoints established based upon animal studies ,the agency is generally not concerned unless the MOE is below 100. In this analysis, tolerance levels were used to calculate the exposure of the highest exposed individual (females, 13+ year subgroup). High end exposure for this subgroup resulted in an MOE in excess of 30,000. Therefore, the acute dietary exposure to halosulfuron-methyl does not represent a risk concern. Monsanto has calculated the MOE for all tolerances (established and proposed) which resulted in an MOE of 31,623 for the entire U.S. population. Monsanto's calculation used the individual food consumption data from the 1989-1992 USDA Food Consumption Surveys and the EXPOSURE-4 software by TAS, Inc. Therefore, Monsanto concludes that there is a reasonable certainty that no harm will result from acute aggregate exposure to halosulfuron-methyl residues.

3. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of halosulfuron-methyl, Monsanto considered 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 the potential for adverse effects on the developing organism resulting from exposure during prenatal development to the female parent. Reproduction studies provide information relating to effects from exposure to the chemical on the reproductive capability of both (mating) parents and on off spring from pre-natal and post-natal exposure to the pesticide as well as systemic toxicity.

In a developmental toxicity study in the rat, the NOEL for both maternal and developmental toxicity was considered to be 250 mg/kg/day. In a developmental toxicity study in rabbits, a NOEL for both developmental and maternal toxicity was considered to be 50 mg/kg/day. A dietary 2-generation reproduction study in rats resulted in parental toxicity at 223.2 mg/kg/day in males and 261.4 mg/kg/day in females in the form of decreased body weights, decreased body weight gains, and reduced food consumption during the premating period. Very slight effects were noted in body weight of the offspring at this dose. This effect was

considered to be developmental toxicity (developmental delay) rather than a reproductive effect. No effects were noted on reproductive or other developmental toxicity parameters. The systemic/developmental toxicity NOEL was 50.4 mg/kg/day in males and 58.7 mg/kg/day in females. The reproductive NOEL was equal to or greater than 223.2 mg/kg/day in males and 261.4 mg/kg/day in females. In all cases, the reproductive and developmental NOELs were greater than the NOEL on which the RfD was based, thus allowing for an additional margin of safety and indicating that halosulfuron-methyl does not pose any increased risk to infants or children.

4. *Chronic analysis*. Using the conservative dietary exposure assumptions described above, the TMRC for the most exposed subgroups is 0.00117 mg/kg body weight/day for nonnursing infants (less than 1-year old) and 0.001008 mg/kg body weight/day for children (1 to 6 years old), and that this aggregate exposure to residues of halosulfuron-methyl utilizes only 1.170 and 1.008% of the RfD, respectively when existing tolerances are considered. Monsanto's subsequent analysis included contribution from the proposed tolerances in sugarcane, sweet corn/popcorn, tree nut crop grouping, pistachio nuts, rice and cotton. The TMRC utilized only 1.7 and 1.3% of the RfD, respectively.

FFDCA section 408 provides that EPA may apply an additional safety factor (up to 10) in the case of threshold effects for infants and children to account for pre- and post-natal toxicity and the completeness of the data base. Based on current toxicological data requirements, the data base relative to pre- and post-natal effects in children is complete. Further, the NOEL of 10 mg/kg/day from the 1-year feeding study in dogs, which was used to calculate the RfD (discussed above), is already lower than the NOELs from the reproductive and developmental studies with halosulfuron-methyl by a factor of at least 25- and 5-fold, respectively. An additional safety factor is not warranted and the RfD of 0.1 mg/kg/day is appropriate for assessing aggregate risk to infants and children.

Therefore, based on complete and reliable toxicity data and the conservative exposure assessment, Monsanto concludes that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to halosulfuron-methyl residues.

F. International Tolerances

Maximum residue levels have not been established for residues of halosulfuron-methyl on corn, sorghum, sugarcane, sweet corn, pop corn, tree nuts, pistachio nuts, rice or cotton or any other food or feed crop by the Codex Alimentarius Commission.

2. Norvartis Crop Protection Inc.

PP 3F4225

EPA has received a pesticide petition (PP 3F4225) from Norvartis Crop Protection INC., P.O. Box 18300, Greensboro, NC 27419, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 extending time limited tolerances for residues of Triasulfuron in or on the raw agricultural commodity grass, forage at 7.0 ppm, grass, hay at 2.0 ppm and kidney of cattle, goats, hogs, horses, and sheep at 0.5 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue in plants is understood. The metabolism of triasulfuron in wheat proceeds by hydroxylation of the phenyl ring and hydrolytic cleavage of the urea bridge. The residue of regulatory concern is parent triasulfuron. Because the metabolism work in wheat can be translated to grasses, parent compound is the residue of regulatory concern for grasses.

2. *Analytical method*. Triasulfuron in grass was analyzed by Analytical Method AG-500B which the validated tolerance enforcement method. According to Method AG-500B, triasulfuron is extracted with a mixture of methanol and phosphoric acid. The extract is diluted with water. Triasulfuron residues are partitioned into dichloromethane and cleaned up on a BondElut CN solid phase extraction column. Residues are determined by column-switching HPLC utilizing a Lichrosorb CN column followed by a Zorbax ODS column, with UV detection at 232 nm.

3. *Magnitude of residues*. A total of 16 field trials have been conducted in 16 States. Seven sites tested bromegrass or fescue, 5 used bluegrass, and 4 used bermudagrass. A total of 69.6% of U.S. pastureland was represented by these trials. Two post broadcast spray applications were made 60-days apart at a rate of 12 grams active ingredient/A/application. Time-limited tolerances were previously established at 7 ppm in grass, forage and 2 ppm in grass, hay pending the submission of additional residue trials. These additional field trials which are included in the numbers above did not show residues exceeding the current tolerances in either grass, forage (0-day PHI) or grass, hay (30-days PHI). The feeding of either substrate to beef or dairy cattle will not result in existing tolerances in animal commodities being exceeded.

B. Toxicological Profile

1. *Acute toxicity*. Triasulfuron has a low order of acute toxicity. The rat oral LD_{50} is > 5,000 milligrams/kilogram (mg/kg), the acute rabbit dermal LD_{50} is > 2,000 mg/kg and the rat inhalation LC_{50} is > 5.2 mg/L. Triasulfuron is slightly irritating to the eye but not irritating to skin. It is not a skin sensitizer in guinea pigs. The commercial formulation of triasulfuron (75WP) has a similar acute toxicity profile. Both the technical material and the 75WP formulation require a Category III CAUTION Signal Word on the label.

2. *Genotoxicty*. Assays for genotoxicity were comprised of tests evaluating the potential of triasulfuron to induce point mutations (*Salmonella typhimurium*, *Saccharomyces cerevisiae* and mouse lymphoma L5178Y/TK/+/- cells), chromosome aberrations (micronucleus test in Chinese hamsters) and the ability to induce either unscheduled DNA synthesis in rat hepatocytes and human fibroblasts. The results indicate that triasulfuron is not mutagenic or clastogenic and does not induce unscheduled DNA synthesis.

3. *Reproductive and developmental toxicity*. The developmental and teratogenic potential of triasulfuron was investigated in rats and rabbits. The results indicate that triasulfuron was maternally toxic in the rat at doses of > 300 mg/kg/day. Developmental toxicity in the form of delayed skeletal maturation was observed only at the highest dose tested (HDT) of 900 mg/kg/day. The corresponding maternal and developmental NOELs were established at doses of 100 and 300 mg/kg/day, respectively in the rat. In the rabbit, maternal toxicity was observed at the HDT of 240 mg/kg/day; no evidence of developmental toxicity was present at 240 mg/kg/day. The maternal developmental NOELs were 120 and 240 mg/kg/day, respectively. No evidence of teratogenicity was observed at the HDT in either the rat or rabbit.

There was no effect of triasulfuron on reproductive performance in a 2 generation rat reproduction study conducted at doses of 1, 50 and 250 mg/kg/day. Maternal and fetal toxicity as indicated by decreased body weight gain was noted at the HDT of 250 mg/kg/day. The maternal and developmental NOEL was 50 mg/kg/day.

4. *Subchronic toxicity*. The subchronic toxicity of triasulfuron was evaluated in the rat and dog at high doses. Triasulfuron was poorly tolerated in the rat at doses of > 516 mg/kg/day as indicated by increased mortality, decreased body weight gain and kidney damage due to the presence of triasulfuron-containing calculi present in the urogenital tract. The NOEL in the rat was 10 mg/kg/day. Triasulfuron was not well tolerated by the dog at doses of 10,000 ppm (250 mg/kg/day) as indicated by body weight reduction, anemia, and effects on the spleen, liver and kidney. The NOEL was 1,000 ppm (33 mg/kg/day).

5. *Chronic toxicity*. The chronic toxicity of triasulfuron was investigated in long term studies in the rat, mouse and dog. Target organs included the liver, kidney and blood. NOELs were established at dose levels of 32.1, 1.2, and 129 mg/kg/day, respectively. The mouse is the most sensitive species with a NOEL = 1.2 mg/kg/day. The carcinogenicity studies on triasulfuron showed no evidence of an oncogenic response in either mouse or rat. The chemical is classified in category E.

6. *Animal metabolism*. The metabolism of triasulfuron has been well characterized in standard FIFRA rat, goat and poultry metabolism studies. Parent triasulfuron accounts for the majority of the excreted dose in these species. Cleavage of the sulfonylurea bridge occurs at a low rate but it is more prevalent in goats and hens than in rats. Hydroxylation of the phenyl ring, which constitutes the major metabolic pathway elucidated in wheat, also was found in the rat. None of the metabolites identified in these studies are considered to be toxicologically different than parent.

7. *Metabolite toxicology*. The metabolism of triasulfuron has been well characterized in rat, goat and poultry metabolism studies. None of the metabolites identified in these studies are considered to be toxicologically different than parent.

8. *Endocrine disruption*. Triasulfuron does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There was no effect of triasulfuron on reproductive performance in a 2-generation rat reproduction study conducted at doses of 1, 50 and 250 mg/kg/day. Although residues of triasulfuron have been found in raw agricultural commodities, there is no evidence that triasulfuron bioaccumulates in the environment.

C. Aggregate Exposure

1. *Food*. Novartis has estimated the aggregate exposure to triasulfuron based on the established and time-limited tolerances for triasulfuron (40 CFR 180.459). The theoretical maximum residue contribution to diet is obtained by multiplying the tolerance level residue for all these raw agricultural commodities by the consumption data which estimates the amount of these products consumed by various population subgroups. Because some of these raw agricultural commodities (e.g. wheat and barley forage and fodder, grass forage and hay) are fed to animals, the transfer of residues to animal commodities has been calculated based on a conservatively constructed cattle diet. In addition, Novartis has conservatively assumed that 100% of the raw agricultural commodities contain residues of triasulfuron at tolerance levels.

2. *Drinking water*. Another potential source of exposure of the general population to residues of pesticides are residues in drinking water. The potential for triasulfuron to enter surface or groundwater sources of drinking water is limited because of the low use rate. The Maximum Contaminant Level Guideline (MCLG) calculated for triasulfuron according to EPA's procedures is 84 ppb, a value that is substantially greater than levels that are likely to be found in the environment under proposed conditions of use.

3. *Non-dietary exposure*. Novartis has evaluated the estimated non-occupational exposure to triasulfuron and concludes that the potential for non-occupational exposure to the general population is unlikely since triasulfuron is not planned to be used in or around the home, including home lawns.

D. Cumulative Effects

Novartis also has considered the potential for cumulative effects of triasulfuron and other chemicals belonging to this class that may have a common mechanism of toxicity. Novartis concluded that consideration of a common mechanism of toxicity is not appropriate at this time since there is no data to establish whether a common mechanism exists.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above, based on the completeness and reliability of the toxicity data, Novartis has concluded that aggregate exposure to triasulfuron will utilize a maximum of 4.63% of the RfD 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. Therefore, Novartis concludes that there is a reasonable certainty that no harm will result from aggregate exposure to triasulfuron or residues of triasulfuron that may appear in raw agricultural commodities.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of triasulfuron, Novartis has considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat on triasulfuron. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from chemical exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to a chemical on the reproductive capability of mating animals and data on systemic toxicity.

Developmental toxicity in the form of delayed skeletal maturation was observed in the rat only at the HDT of 900 mg/kg/day. The corresponding maternal and developmental NOELs were established at doses of 100 and 300 mg/kg/day, respectively in the rat. In the rabbit, maternal toxicity was observed at the HDT of 240 mg/kg/day; no evidence of developmental toxicity was present at 240 mg/kg/day.

There was no effect of triasulfuron on reproductive performance in a 2 generation rat reproduction study conducted at doses of 1, 50 and 250 mg/kg/day. Maternal and fetal toxicity as indicated by decreased body weight gain was noted at the HDT 250 mg/kg/day. The maternal and developmental NOELs were 50 mg/kg/day.

Section 408 of the FFDCA provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. Further, for triasulfuron,

the NOEL of 1.2 mg/kg/day from the mouse oncogenicity study, which was used to calculate the RfD of 0.01 mg/kg/day, was approximately 50 times lower than the developmental NOEL level from the rat multigeneration reproduction study. There is no evidence to suggest that developing organisms are more sensitive to the effects of triasulfuron than are adults.

Using the conservative exposure assumptions described above and the chronic toxicity NOEL of 1.2 mg/kg/day (RfD of 0.01 mg/kg/day), Novartis has determined that the % of the RfD that will be utilized by aggregate exposure to residues of triasulfuron is 3.98% for nursing infants less than 1-year old, 15.43% for non-nursing infants, 10.91% for children 1 to 6-years old and 7.34% for children 7 to 12-years old. Therefore, based on the completeness and reliability of the toxicity data 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 triasulfuron residues.

F. International Tolerances

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRL's) established for residues of triasulfuron in or on raw agricultural commodities.

3. Zeneca Ag Products

PP 8F4954

EPA has received a pesticide petition (PP 8F4954) from Zeneca Ag Products, 1800 Concord Pike, Wilmington, DE 19850-5458 proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the herbicide, 2-[4-(methylsulfonyl)-2-nitrobenzoyl]-1,3-cyclohexanedione, in or on the raw agricultural commodities field corn, field corn fodder and field corn forage at 0.01 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue of 2-[4-(methylsulfonyl)-2-nitrobenzoyl]-1,3-cyclohexanedione, (hereafter referred to by the trade name ZA1296) in plants is adequately understood. ZA1296 is rapidly and completely metabolized in corn. No single extract or component accounted for greater than 0.01 ppm in grain. Numerous components were characterised in forage and fodder, including the metabolite 2-amino-4-methylsulfonyl benzoic acid (AMBA) and its conjugates and 4-methylsulfonyl-2-nitrobenzoic acid (MNBA). In addition to ZA1296, MNBA was included in crop residue analysis.

2. *Analytical method*. The proposed analytical method involves extraction, partition, clean-up and separation of ZA1296 and MNBA, oxidation of ZA1296, reduction, clean-up and detection of residues by reversed-phase HPLC using fluorescence detection. The limit of quantitation for ZA1296 and the metabolite MNBA is 0.01 ppm.

3. *Magnitude of residues*. Twenty residue trials were conducted in the US (EPA regions I, II, V and VI). The proposed use of ZA1296 does not result in residues (LOQ of 0.01 ppm) of ZA1296 or the metabolite MNBA in field corn grain, forage or fodder.

B. Toxicological Profile

1. *Acute toxicity*. A battery of acute toxicity tests were conducted which place ZA1296 in acute oral toxicity category IV, acute dermal toxicity category III, acute inhalation toxicity category IV, primary eye irritation category III, and primary dermal irritation category IV. ZA1296 is not a skin sensitizer. ZA1296 is not a neurotoxin in males and females at 2,000 mg/kg (limit test).

2. *Genotoxicty*. ZA296 was found to be negative for mutagenicity in a battery of mutagenicity tests (*in vitro*) Ames Testing, Mouse Lymphoma, Human Lymphocytes and *in vivo* Mouse Micronucleus).

3. *Reproductive and developmental toxicity*—i. *Developmental toxicity (rabbit)*. New Zealand white rabbits were dosed orally by gavage with 0, 100, 250 or 500 mg/kg/day ZA1296 on days 8-20 of gestation. The top dose level in this study was set on the basis of significant maternal toxicity seen at higher dose levels in a preliminary study. At dose levels of 250 and 500 mg/kg/day there was a low incidence of whole litter losses. ZA1296 was not associated with significant maternal toxicity or evidence of teratogenicity. Dose levels of 100 mg/kg/day or more were associated with changes in the ossification of the fetal skeleton but not with structural malformation. The changes in ossification are transient in nature and considered not to be of toxicological significance in terms of post natal development. A developmental NOAEL of 100 mg/kg/day was established in this study.

ii. *Developmental toxicity (rat)*. Rats were dosed orally by gavage with 0, 100, 300 or 1,000 mg/kg/day ZA1296 on days 7-16 of gestation. Maternal toxicity, as evidenced by reductions in body weight and food consumption, was seen at dose levels of 100, 300 or 1,000 mg/kg/day ZA1296. Administration of ZA1296 at dose levels of up to 1,000 mg/kg/day produced no evidence of teratogenicity. An increased incidence of minor skeletal defects and skeletal variants and increases in mean manus and pes scores were seen at all dose levels and were indicative of reduced ossification or a disturbance in the normal pattern of ossification. The changes in ossification are transient in nature and are considered not to be of toxicological significance in terms of post-natal development. Fetal weight was reduced at 1,000 mg/kg/day. A developmental NOAEL of 300 mg/kg/day was established in this study.

iii. *Reproductive toxicity (rat)*. In a 3-generation study rats were fed diets containing 0, 2.5, 10 or 2500 ppm ZA1296. Dietary administration of ZA1296 had no effect on mating performance but was found to result in reduced pup survival at a dose of 2,500 ppm in all 3-generations and at 100 ppm in the second generation only. These findings were not present in recovery subgroups removed from treated diet in the third generation. There was also a reduction in the number of pups per litter, and effects on body weights and in the eye and kidney. In the third generation, there were no effects in the eyes or kidneys of offspring from animals which were returned to control diet 4 weeks prior to mating and effects on litter size were less marked than in the continuous treatment group. A NOEL of 2.5 ppm ZA1296 (0.3 mg/kg/day) was established in this study. In light of the mechanism of toxicity, investigations into the effects seen in this study in pups are considered not to be relevant to human risk assessment.

iv. *Reproductive toxicity (mouse)*. In a 2-generation study mice were fed diets containing 0, 10, 50, 350, 1,500 or 7.000 ppm ZA1296. There were no adverse effect of ZA1296 on the reproductive performance of the mouse, on fertility and fecundity of the F0 and F1 adult animals or on survival of their offspring. The body weights of the offspring were reduced at 1,500 and 7,000 ppm ZA1296. A NOEL of 350 ppm ZA1296 (71 mg/kg/day) was established in this study.

4. *Subchronic toxicity*—i. *21-day dermal (rabbits)*. Rabbits were repeatedly dosed with ZA1296 at 0, 10,

500 or 1,000 mg/kg/day for 21 days. The NOEL for sub-acute dermal toxicity was >1,000 mg/kg/day (limit dose).

ii. *90-day rodent (rat).* In a first study male and female rats were dosed with 0, 1, 125, 1,250 or 12,500 ppm ZA1296 in the diet for 90-days. The NOEL was determined to be 1ppm for males and females (0.09 and 0.1 mg/kg/day, respectively) based on reduced bodyweight and increased liver weight in males and females at 125 ppm and increased kidney weight and ocular keratitis in males at 125 ppm. 125 ppm (13 mg/kg/day) was a NOEL for the ocular keratitis in females. In a second study in male rats dosed with ZA1296 at 0, 10, 20 or 150 ppm ZA1296 in the diet for 90-days, a NOEL of 20 ppm (1.7 mg/kg/day) was determined for reduced bodyweight. At the 10 ppm dose level ocular keratitis and increased liver and kidney weights were observed. In a third study in male and female rats dosed with ZA1296 at 0, 2.5, 5.0, 7.5 or 150 ppm in the diet for 90-days, NOELs of 5 ppm (0.41 mg/kg/day) for ocular keratitis and increased kidney weight and 7.5 ppm (0.63 mg/kg/day) for reduced bodyweight were determined in males. NOELs of 7.5 ppm (0.71 mg/kg/day) for reduced bodyweight and increased liver weight and 150 ppm (14 mg/kg/day) for increased kidney weight were determined in females. At 2.5 ppm in males increased liver weight was observed. In light of investigations into the mechanism of toxicity, these changes are all considered not to be relevant to human risk assessment.

iii. *90-day rodent (mouse).* Mice were dosed 0, 50, 350 or 7,000 ppm in the diet for 90-days. In females no clear toxic effects were observed at 7,000 ppm (1,500 mg/kg/day). In males 7,000 ppm (1,200 mg/kg/day) was associated with a reduced growth rate and food utilization. In males and females 350 ppm (62 and 80 mg/kg/day, respectively) produced no effects which were considered to be toxicologically significant.

iv. *90-day non-rodent (dog).* Beagle dogs were dosed with ZA1296 at 0, 100, 600 or 1,000 mg/kg/day as a daily oral dose by capsule, for a period of 90-days. The NOEL in the dog over 90-days was 100 mg/kg/day. Minimal toxicity was observed at 600 and 1,000 mg/kg/day, evident as reduced bodyweights in males and a microcytic polycythemia in both sexes. Mesothelial proliferation of the atrium of the heart was evident in 2 male dogs at 1,000 mg/kg/day.

v. *90-day neurotoxicity (rat).* Rats were dosed with ZA1296 at 0, 2.5, 100 or 5,000 ppm in the diet for 90-days. The NOAEL for subchronic neurotoxicity was determined to be 5,000 ppm (400 and 460 mg/kg/day for males and females, respectively) based on the absence of changes indicative of neurotoxicity.

5. *Chronic toxicity*—i. *1-year non-rodent (dog).* Beagle dogs were dosed with ZA1296 at 0, 10, 100 or 600 mg/kg/day as a daily oral dose by capsule, for a period of 1-year. The NOEL in this study was 100 mg/kg/day. At 600 mg/kg/day males showed a significant reduction in bodyweight and both sexes showed a slight microcytic polycythemia, indicating that a maximum tolerated dose had been achieved. Minimal ocular keratitis was observed in 1 male and 1 female at 600 mg/kg/day.

ii. *1-year rodent (mouse).* Mice were dosed with ZA1296 at 0, 10, 50, 350 or 7,000 ppm in the diet for 1 year. The NOEL in males and females was 350 ppm (56 and 72 mg/kg/day, respectively). At 7,000 ppm (limit dose) bodyweight was reduced in males, and there was an increased incidence of eosinophilic change in the gall bladder of females.

iii. *Combined rodent chronic toxicity/oncogenicity (rat).* Rats were dosed with ZA1296 at 0, 7.5, 100 or 2,500 ppm in the diet for up to 2 years. In addition rats were fed diet containing 1 or 2.5 ppm ZA1296 for up to 2-years to determine the chronic ocular toxicity. Oral administration of 7.5, 100 or 2,500 ppm ZA1296 for at least 2-years caused ocular keratitis, reduced bodyweights, increased liver and kidney weights, and an increased incidence of common spontaneous lesions in the Alderley Park rat. In light of investigations into the mechanism of toxicity, these changes are all considered not to be related to human risk assessment. Satellite groups of rats fed 1 and 2.5 ppm ZA1296 showed that dietary levels of 2.5 ppm in males and 7.5 ppm in females were without ocular effect. ZA1296 was considered not to be carcinogenic in the rat in this study. A NOEL of 7.5 ppm ZA1296 was established for females.

iv. *Oncogenicity in the rodent (mouse).* Mice were fed diets containing 0, 10, 350 or 7,000 ppm ZA1296 for up to 80-weeks. Oral administration of 7,000 ppm (900-1,100 mg/kg/day) ZA1296 (limit dose) for at least 80-weeks produced no evidence of carcinogenicity in male or female mice.

6. *Animal metabolism*. The absorption, distribution, metabolism and excretion of ZA1296 has been thoroughly investigated in rats and studied in mice. In both species ZA1296 is well absorbed following an oral dose. Elimination of ZA1296 is rapid in both species, with most of the ZA1296 eliminated, in the urine, unchanged with only minor amounts of the urinary and fecal metabolites, including MNBA and AMBA, detected. In poultry ZA1296 is excreted generally unchanged. In ruminants ZA1296 is extensively metabolised and excreted. AMBA dosed to ruminants is readily absorbed and excreted, generally unchanged. AMBA is not accumulated in edible tissues or milk.

7. *Metabolite toxicology*. In acute oral toxicity studies in male and female rats both MNBA and AMBA had an oral LD_{50} of >5,000 mg/kg. In the Ames assay, both MNBA and AMBA were found to be negative for mutagenicity in the absence and presence of metabolic activation.

8. *Endocrine disruption*. EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect.'' EPA is currently working with interested shareholders, including other government agencies, public interest groups, industry, and research scientists, to develop a screening and testing program and a priority setting scheme to implement this program. Congress has allowed 3-years from the passage of FQPA (August 3, 1999) to implement this program. When this program is implemented, EPA may require further testing of ZA1296 and end-use product formulations for endocrine disrupter effects.

9. *Reference dose*. As required by the Food Quality and Protection Act of 1996, the mechanism of toxicity of ZA1296 has been thoroughly investigated in studies (FQPA) in the rat, mouse and man. These data clearly demonstrate that the response to ZA1296 administration in man is very similar to that seen in the mouse which should therefore, be used in preference to the rat when assessing the safety of ZA1296 to humans. The proposed reference dose (RfD) for use in the assessment of risk from chronic exposure is 0.56 mg/kg/day and is derived from the 1 year chronic toxicity study in the mouse with a NOEL of 56 mg/kg/day and a 100-fold uncertainty factor.

C. Aggregate Exposure

1. *Dietary exposure*. The potential dietary exposure to ZA1296 was estimated from tolerance levels and 100% crop treated. No tolerances are proposed for meat, milk and eggs. The total dietary exposure for the U.S. population and the most highly exposed

subgroup in the population, non-nursing infants, is 0.000011 mg/kg/day and 0.000027 mg/kg/day, respectively.

2. *Drinking water*. Drinking water estimated concentrations (DWEC) were calculated using EPA models for groundwater and surface water - SCI-GROW, GENEEC and PRZM/EXAMS. Chronic Drinking Water Levels of Concern (DWLOC) were calculated according to the EPA SOP and compared to the DWEC. Estimated average contributions of ZA1296 in surface and groundwater are less than the levels of concern for ZA1296 in drinking water as a contribution to chronic aggregate exposure.

3. *Non-dietary exposure*. Zeneca has not estimated non-occupational exposure for ZA1296 since the only pending registration for ZA1296 is limited to commercial crop production use. ZA1296 products are not labelled for any residential uses therefore, eliminating the potential for residential exposure. The potential for non-occupational exposure to the general population is considered to be insignificant.

D. Cumulative Effects

Zeneca also considered the potential for cumulative effects of ZA1296 and other substances that have a common mechanism of toxicity. Zeneca has concluded that consideration of a common mechanism of toxicity is not appropriate at this time since there is no indication that toxic effects produced by ZA1296 would be cumulative with those of any other chemical compounds. Triketone chemistry is new and ZA1296 has a novel mode of action compared to currently registered active ingredients.

E. Safety Determination

1. *U.S. population*. Dietary and occupational exposure will be the major routes of exposure to the U.S. population and ample margins of safety have been demonstrated for both situations. The total dietary exposure for the U.S. population is 0.000011 mg/kg/day. This utilizes only 0.002% of the RfD. The MOE for occupational exposure is >5,500. Based on the completeness and reliability of the toxicity data and the conservative exposure assessments, there is reasonable certainty that no harm will result from the aggregate exposure of residues of ZA1296 including all anticipated dietary exposure.

2. *Infants and children*. The total dietary exposure for the most highly exposed subgroup in the population, non-nursing infants, is 0.000027 mg/kg/day. This utilizes only 0.0048% of the RfD. There are no residential uses of ZA1296 and the estimated average contributions of ZA1296 in surface and groundwater are less than the levels of concern for ZA1296 in drinking water as a contribution to chronic aggregate exposure. Based on the completeness and reliability of the toxicity data and the conservative exposure assessments, there is reasonable certainty that no harm will result from the aggregate exposure of residues of ZA1296 including all anticipated dietary exposure.

F. International Tolerances

A maximum residue level has not been established for ZA1296 by the Codex Alimentarius Commission.

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BILLING CODE 6560–50–F

FEDERAL COMMUNICATIONS COMMISSION

[CC Docket No. 91–141; DA 98–839]

Local Competition Survey

AGENCY: Federal Communications Commission.

ACTION: Notice.

SUMMARY: On May 8, 1998, the Common Carrier Bureau issued a Public Notice to solicit comment on how the Commission can collect sufficient information about local competition to achieve the regulatory flexibility, pro-competition, and universal service objectives of the Telecommunications Act of 1996 (1996 Act) while minimizing filing burdens on respondents. The Public Notice seeks comment on what information should be collected as well as on such issues as whether periodic data collection should be mandatory and which telecommunications carriers should provide information.

DATES: Comments to the Public Notice are due on or before June 7, 1998. Reply comments are due on or before June 22, 1998.

ADDRESSES: Comments and reply comments should be sent to the Office of the Secretary, Federal Communications Commission, 1919 M Street, N.W., Suite 222, Washington, D.C. 20554, with a copy to Ms. Terry Conway of the Common Carrier Bureau, Federal Communications Commission, 2033 M Street, N.W., Suite 500, Washington, D.C. 20554. Parties should also file one copy of any documents filed in this docket with the Commission's copy contractor, International Transcription Services, Inc. (ITS), 1231 20th St., NW, Washington, DC 20036, (202) 857–3800.

FOR FURTHER INFORMATION CONTACT: Thomas J. Beers, Deputy Chief of the Industry Analysis Division, Common Carrier Bureau, at (202) 418–0952, or Ellen Burton, Industry Analysis Division, Common Carrier Bureau, at (202) 418–0958. Users of TTY equipment may call (202) 418–0484.

SUPPLEMENTARY INFORMATION: This is a summary of the Bureau's Public Notice released May 8, 1998 (DA 98–839). The full text of this Public Notice is available for inspection and copying during normal business hours in the FCC Reference Center, Room 239, 1919 M Street, Washington, D.C. 20554. The complete text also may be purchased from the Commission's copy contractor, International Transcription Service, Inc., (202) 857–3800, 1231 20th St., NW, Washington, DC 20036.

Summary of the Public Notice

The Commission requires timely and reliable information on the pace and extent of development of competition for local telecommunications services in different geographic markets to evaluate the effectiveness of decisions taken to implement the pro-competition provisions and to achieve the universal service goals of the Telecommunication Act of 1996 (47 U.S.C. Section 151 *et seq.*). The Commission also requires such information to identify services and geographic markets where local competition has developed sufficiently to allow the Commission to exercise its regulatory forbearance authority (47 U.S.C. Section 160(a)).

The Commission has previously concluded (*Expanded Interconnection with Local Telephone Company Facilities*, Memorandum Opinion and Order, 59 FR 38922 (August 1, 1994), CC Docket No. 91–141, 9 FCC Rcd 5154, 5177 (1994)) that an information collection program is necessary to monitor the state of local competition in diverse areas of the country so that the Commission might make its regulatory requirements more flexible as competition develops in particular areas. The Commission delegated authority to the Chief, Common Carrier Bureau, to formulate the detailed elements of a reporting program, to decide which service providers must provide information, and to specify the format and timing of reports.

I. Background

3. Only a limited amount of information on the state of local competition can be derived from sources currently reported to the