Therefore, neither a tolerance nor a tolerance exemption is needed for the use of Rhodamine B as a dye in seed treatment pesticide products.

The final rule was published on December 27, 2001 (66 FR 66769) (FRL–6813–6). The Rhodamine B use pattern is now limited to use as a dye in seed treatment, and for a period of 3 years Rhodamine B can also be used as a dye in animal ear tag pesticide products. This 3–year time frame is needed to allow those pesticide ear tag products containing Rhodamine B to clear the channels of trade.

B. Future Actions

Rhodamine B's classification as a carcinogen remains unchanged. However, the Agency no longer considers List 1 classification for Rhodamine B for its use as a dye in seed treatment pesticide products to be appropriate. List 1 classifications are made according to hazard criteria only. However, the December 27, 2001 **Federal Register** limited the use of Rhodamine B to a specified use pattern. A List 4B inert ingredient is considered to be an inert ingredient for which the available toxicity (hazard) information when paired with the available exposure information indicates no reasonable expectation of adverse effects. Rhodamine B now meets the definition of a List 4B, and will be reclassified as such.

Those persons desiring to register products containing Rhodamine B as an inert ingredient for any uses other than as a dye in seed treatment would need to submit an extensive data set similar to that required in the 1993 Rhodamine B DCI. These data would be used by the Agency in a risk assessment on the proposed use. If, the risk assessment supports the required safety finding, then the use would be approved.

List of Subjects

Environmental protection, pesticides and pests.

Dated: February 26, 2002.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 02–5445 Filed 3–7–02; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–1072; FRL–6825–8]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

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 docket control number PF–1072, must be received on or before April 8, 2002.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–1072 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: James A. Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–5697; e-mail address: tompkins.jim@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 codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to, the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this action under docket control number PF–1072. 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–1072 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, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically.* You may submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters, and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–1072. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT.**

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

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

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

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

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

5. Provide specific examples to illustrate your concerns.

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

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

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic 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 support 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: February 21, 2002.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by E. I. du Pont Nemours and Company, and represents the view of the E. I. du Pont Nemours Company. EPA is publishing the petition summary verbatim without editing it 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.

E. I. du Pont Nemours and Company

PP 0F6120

EPA has received a pesticide petition (0F6120) from E. I. du Pont de Nemours and Company, DuPont Agricultural Products, Barley Mill Plaza, Wilmington, DE 19880–0038 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the herbicide chlorsulfuron: 2-Chloro-N-[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) aminocarbonyl] benzenesulfonamide in or on the raw agricultural commodities grass forage at 11 parts per million (ppm) and grass hay at 19 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 qualitative nature of residue in plants is adequately understood. Metabolism studies have been conducted in both wheat and barley and the metabolic profiles are consistent. In wheat, ^{14}C-triazine chlorsulfuron and ^{14}C-phenyl chlorsulfuron were applied foliarly to the field plots at the rates of 0.25 ounce active indregient/acre (oz ai/A) and 1.5 oz ai/A. Samples of wheat were harvested on the day of application (forage), 7 days later (late forage), and 19 days later (hay). At maturity, 82 days after treatment, the grain heads and straw were harvested. Chlorsulfuron showed systemic absorption and translocation. The deposited radioactivity on surfaces is small. Combustion analysis of the 0–day, 7–day, and 19–day 1x treatment resulted in total radioactive residue (TRRs) of approximately 1.155 ppm, 0.065 ppm, and 0.017 ppm ^{14}C-triazine chlorsulfuron equivalent, and 1.168 ppm, 0.102 ppm, and 0.024 ppm ^{14}C-phenyl chlorsulfuron equivalent, respectively. TRRs for the samples taken at maturity were 0.003 ppm for the straw, and at or below the limit of detection (0.001 ppm) for the grain. The primary metabolic pathway of chlorsulfuron in plants, involved hydroxylation of the intact parent molecule to yield 5-hydroxy chlorsulfuron, which subsequently underwent glucoside conjugation. The glucose conjugate of 5-hydroxy chlorsulfuron accounts for 49.5% and 25.6% TRR (0.032 and 0.004 ppm) in

wheat 7 and 19 days after ^{14}C-triazine chlorsulfuron treatment; and for 30.3% and 24.6% TRR (0.031 ppm and 0.006 ppm) 7 and 19 days after ^{14}C-phenyl chlorsulfuron treatment. In the 19–day triazine and phenyl labeled samples, 5-hydroxy chlorsulfuron was present at 0.001 ppm. After glucoside conjugation, the cleavage of the sulfonylurea linkage occurs to yield the corresponding sulfonamide conjugate and triazine. ^{14}C-triazine chlorsulfuron treated wheat contains 6.5% TRR (0.004 ppm) triazine amine in the 7–day sample. The glucose conjugate of 5-hydroxy chlorsulfonamide accounts for 8.6% TRR (0.009 ppm) in the 7–day sample and 10.4% TRR (0.002 ppm) in the 19–day sample from ^{14}C-phenyl chlorsulfuron treated wheat.

2. *Analytical method.* The analytical enforcement method exists for the determination of chlorsulfuron in cereal forage, hay, grain and straw and grass forage and hay. Samples are extracted in aqueous solution, acidified, purified and concentrated by reversed-phase solid-phase extraction. Extracts are analyzed by liquid chromatography/mass spectrometry employing electrospray ionization (ESI-LC/MS).

3. *Magnitude of residues.* It has been determined that the residue to be regulated is parent chlorsulfuron only. A study was conducted to determine the magnitude of residues of chlorsulfuron and its metabolite, 5-hydroxy chlorsulfuron in wheat forage, grain and straw following application of Glean FC herbicide, at the maximum label rate. Chlorsulfuron residues in wheat grain and straw were below 0.05 ppm, the limit of quantitation (LOQ) at all sites. Chlorsulfuron residues in wheat forage were below 0.05 ppm in all sites (PHI of 19 to 35 days) except one which had a residue level range of 0.31–0.60 ppm (PHI of 1 day).

Another study was conducted to determine the magnitude of residues of chlorsulfuron in wheat forage and hay at a 0 day PHI following application of chlorsulfuron at 0.5 oz a.i./A. The residues for wheat forage ranged between 0.66 and 5.0 ppm. The residues for wheat hay ranged between 0.56 and 12 ppm.

An additional study determined the magnitude and decline of residues of chlorsulfuron in pasture grass forage and hay following application of chlorsulfuron at 1.0 oz a.i./A. The application was made with the shortest time to harvest allowed by the label (0 day PHI). Applications were made, when the grass was at a forageable stage of growth. At a 0 day PHI, the residue levels in the grass forage were between 1.2 and 11 ppm. The residue levels in the grass hay at 0–day PHI were between 1.0 and 19 ppm.

In a greenhouse rotational crop study, wheat, sugar beets and rape plants were grown on soil, which had been treated with ^{14}C-chlorsulfuron at 1.0 oz/A and field-aged for periods of 4 and 12 months. In all crops planted 4 months following chlorsulfuron treatment, intact ^{14}C-chlorsulfuron, if present at all, was less than 0.2 parts per billion (ppb).

B. Toxicological Profile

1. *Acute toxicity.* Based on EPA criteria, technical chlorsulfuron is in toxicity Category IV for oral and inhalation routes of exposure, and for dermal irritation. Chlorsulfuron is in toxicity Category III for eye irritation, and the dermal route of exposure. It is not a skin sensitizer.

Acute oral toxicity in rats: LD_{50} = 5,545 milligrams/kilogram (mg/kg) (M), 6,293 mg/kg (F) mg/kg (F)

Acute dermal toxicity in rabbits: LD_{50} > 3,400 mg/kg

Acute inhalation toxicity in rats: LC_{50} > 5.9 mg/L

Primary eye irritation in rabbits: Moderate effects reversed within 72 hours

Primary dermal irritation in rabbits: non-irritant

2. *Genotoxicty.* Technical chlorsulfuron has shown no genotoxic or mutagenic activity in the following *in vitro* and *in vivo* tests:

In vitro Mutagenicity Ames Assay: Negative

In vitro Mutagenicity CHO/HPRT Assay: Negative

In vitro Cytogenetic Study: Negative

In vitro DNA Repair Study: Negative

In vitro UDS: Negative

In vivo Dominant Lethal Mutagenicity: Negative

3. *Reproductive and developmental toxicity.* In a multigeneration reproduction study in rats fed 0, 100, 500, or 2,500 ppm chlorsulfuron, the only observed effect on reproduction endpoints was slightly decreased fertility indices in rats from the 2,500 ppm group. Mean number of pups per litter, gestation, lactation, and viability indices, litter survival, and mean weanling body weights and weight gains were not adversely influenced by chlorsulfuron. No gross or histopathological abnormalities were observed in weanling rats. The no observed adverse effect level (NOAEL) based on decreased fertility indices was 500 ppm. The NOAEL based on systemic toxicity was 100 ppm.

In studies conducted to evaluate potential developmental toxicity, chlorsulfuron was neither teratogenic nor uniquely toxic to the conceptus (i.e., not considered a developmental toxin). In the rat study, chlorsulfuron was administered by gavage to rats on days 7–16 of gestation at daily dose levels of 0, 55, 165, 500, or 1,500 mg/kg. There was evidence of maternal toxicity (spontaneous death, weight loss, reductions of feed consumption) at the two highest dose levels. The remaining groups showed no evidence of any effects on maternal body weights, feed consumption or clinical signs. No effects were seen in any experimental group on mean nidations, live fetuses per litter, *in utero* survival or on mean corpora lutea counts. Fetal toxicity was evident as a depression in fetal weights only at the highest dose tested (HDT). Treatment with chlorsulfuron did not result in any significant increase in fetal alterations (malformations or variations). Maternal toxicity was observed at daily dose levels greater than or equal to 500 mg/kg. Fetal toxicity was seen only at a level of 1,500 mg/kg, a maternally toxic dose. The NOAEL was 165 mg/kg/day for the dam and 500 mg/kg/day for the conceptus. In the rabbit developmental toxicity study, chlorsulfuron was administered by gavage to rabbits on days 7–19 of gestation at daily dose levels of 0, 25, 75, 200, or 400 mg/kg. Since no overt maternal or fetal toxicity was evident, a supplementary study was conducted in which chlorsulfuron was administered at daily dose levels of 0, 400, and 1,000 mg/kg. Maternal toxicity, evident at the highest level, 1,000 mg/kg/day, consisted of a significant incidence of mortality and abortions; a significant increase in the incidence of females with clinical signs and significantly decreased mean maternal body weight changes. In addition, mean maternal weight gains for days 7–29 were also significantly reduced. At 400 mg/kg/day, the only evidence of maternal toxicity was a significant reduction in mean maternal adjusted body weight gains on days 7–29. No other maternal toxic effects were seen at any dose level. There was no evidence of fetal toxicity seen in either study. Therefore, under the conditions of these studies, the NOAEL was 200 mg/kg/day for the dam and > 1,000 mg/kg/day for the conceptus.

4. *Subchronic toxicity.* In a ten-dose oral subacute test, chlorsulfuron was administered orally to male rats at a repeated dose level of 2,200 mg/kg/day for 10 days over a 2–week period. No test compound-related gross or histologic changes were observed.

The rat was the most sensitive species to subchronic exposure of chlorsulfuron. Male and female rats were fed diets for 98 days that contained

0, 100, 500, or 2,500 ppm chlorsulfuron. Male rats fed diets at 500 or 2,500 ppm exhibited decreased urine pH and decreased plasma creatinine. Rats in the 500 and 2,500 ppm groups also exhibited decreased monocyte counts. These findings show that the NOAEL for chlorsulfuron was 100 ppm for male and female rats (98–day dietary). In the mouse study, groups of male and female mice were fed chlorsulfuron at levels of 0, 500, 2,500, 5,000, or 7,500 ppm. No meaningful differences in weight gain, food consumption, or food efficiency existed between control and treated mice fed chlorsulfuron. Male mice fed 5,000 or 7,500 ppm had lower erythrocyte count and higher mean corpuscular volumes and mean corpuscular hemoglobin values than control males. Female mice fed 5,000 or 7,500 ppm, had fewer neutrophilic granulocytes and more lymphocytes than control females. No hematologic effects were seen in mice fed 500 or 2,500 ppm chlorsulfuron. Gross pathologic findings in mice at all feeding levels and microscopic findings in mice fed 7,500 ppm were considered to be spontaneous or the result of intercurrent disease. No effects attributable to the feeding of chlorsulfuron were observed in mice fed 500 or 2,500 ppm chlorsulfuron. Therefore, the NOAEL for male and female mice is 2,500 ppm (90–day dietary).

5. *Chronic toxicity.* In a long-term feeding study with chlorsulfuron, male and female mice were fed diets of 0, 100, 500, or 5,000 ppm chlorsulfuron. Mean body weights and weight gains of mice in the 5,000 ppm treatment groups were decreased when compared to those of their respective control groups. The NOAEL for chronic (2–year dietary) exposure of chlorsulfuron in mice was 500 ppm for male and female mice. No behavioral, clinical, hematological, gross pathological or histological abnormalities were observed, that could be related to the dietary administration of chlorsulfuron. Chlorsulfuron was not oncogenic when administered to male and female mice for 2 years at levels of 100, 500, or 5,000 ppm. In a long-term feeding study, male and female rats were fed diets containing 0, 100, 500, or 2,500 ppm chlorsulfuron. Mild to moderate reduction in mean body weights and weight gains in male rats from the 500 and 2,500 ppm treatment groups was observed. No other behavioral, nutritional, clinical, or hematological abnormalities that could be attributed to chlorsulfuron treatment were observed during the feeding study. The NOAEL (2–year dietary) in male and female rats was 100 ppm (5 mg/kg). Chlorsulfuron was not an oncogen in rats.

In a 1–year chronic study with dogs, male and female dogs were fed dietary levels of 0, 100, 2,000, or 7,500 ppm chlorsulfuron. There were slight body weight decreases and hematological changes in females in the 7,500 ppm treatment group. Therefore, the NOAEL (1–year dietary) is 2,000 ppm.

6. *Animal metabolism.* Due to its rapid elimination, metabolism of chlorsulfuron in animals is minimal. O-Demethylation and cleavage of the sulfonylurea linkage were observed.

Rats were dosed with ^{14}C-phenyl labeled chlorsulfuron. Chlorsulfuron and its metabolites were excreted rapidly from the rats. An average of 85% of the recovered radioactivity was excreted in the urine and 12% in the feces. Less than 1% of any of the various doses was retained in the body organs. Most (85%) of the excreted radioactivity was present, as intact chlorsulfuron with minor amounts of 2-chlorobenzene-sulfonamide and two polar metabolites.

Results from a metabolism study with two radioactive forms of chlorsulfuron (^{14}C-triazine and ^{14}C-phenyl) in lactating goats show that chlorsulfuron is readily excreted unchanged in urine and feces of the goat. The target dose for each test goat was 45 mg/goat/day, which is equivalent to a daily dietary intake of 25 ppm, assuming daily food consumption of 1.8 kg. The results of this study indicate that chlorsulfuron is readily excreted unchanged in urine and feces of the goat. A majority of the cumulative dose was excreted in the urine (69–75%) and feces (5.9–7.6%).

Additional radioactivity was recovered in the cage wash and accounted for 3.8–6.7% of the dose. O-desmethylchlorsulfuron was identified in the feces indicating there is O-dealkylation of chlorsulfuron most likely by gut microflora. The appearance of 4-methoxy-6-methyl-1,3,5-triazin-2-amine and 2-chlorobenzenesulfonamide indicates hydrolysis of the amide linkage in the sulfonylurea bridge. Neither of these metabolites was present in the urine or feces, suggesting they are further metabolized before being excreted. Total milk residues reached steady-state after 24 hours, indicating bioaccumulation of residues in milk is unlikely. The highest tissue residues were found in the kidney and liver, because urinary and fecal excretion are the primary routes of elimination for chlorsulfuron. It is unlikely that chlorsulfuron or any of its metabolites will bioaccumulate in the tissues or milk of the lactating goat.

The poultry metabolism study was conducted at 1 ppm ^{14}C-chlorsulfuron in feed for up to 14 days in laying hens. After 14 days, 85–99% of the total radioactivity was accounted for in the hen excreta, with the majority being ^{14}C-chlorsulfuron. These data are consistent with previous research; demonstrating no accumulation of chlorsulfuron residues in animal tissues and minimal metabolism of the chlorsulfuron molecule in the rat and goat.

Dairy cattle were fed chlorsulfuron at dietary levels of 2, 10, and 50 ppm for 28 days. The chlorsulfuron residue levels in milk rose within 3 days to steady-state plateaus, remaining constant during fortified feeding, and decreased to below the analytical detection limit of 0.010 ppm within 3 days of terminating the fortified feeding. Average steady-state residue levels in the milk during fortified feeding, were 0.064 ppm for cows fed at the 50 ppm dietary rate and 0.013 ppm for cows fed at the 10 ppm dietary rate. No more than 0.2% of the ingested chlorsulfuron appeared as residues in the milk. Chlorsulfuron was rapidly eliminated from the animal in the urine and feces. Average concentrations of chlorsulfuron in urine and feces were 24 ppm and 0.6 ppm, respectively, for cows fed chlorsulfuron at the 50 ppm dietary level. Chlorsulfuron was detected in the kidney 0.25 ppm, liver 0.024 ppm, and lean muscle < 0.010 ppm of the cow fed at the 50 ppm dietary level, but was undetected (< 0.01 ppm in subcutaneous fat. Chlorsulfuron residues in all analyzed tissue decreased to< 0.010 ppm for all cows within 8–days of returning to a diet without chlorsulfuron. Addition of the proposed grass tolerances will not significantly increase the dietary burden for cattle since tolerances already exist for cereal feed commodities. The total dietary burden of chlorsulfuron for cattle will remain less than 50 ppm.

7. *Metabolite toxicology.* There is no evidence that the metabolites of chlorsulfuron as identified in either the plant, or animal metabolism studies are of any toxicological significance.

8. *Endocrine disruption.* Chronic, lifespan, and multigenerational bioassays in mammals and acute, and subchronic studies on aquatic organisms and wildlife did not reveal endocrine effects. Any endocrine related effects, would have been detected in this definitive array of required tests. The probability of any such effect due to agricultural uses of chlorsulfuron is negligible.

C. Aggregate Exposure

1. *Dietary exposure.* Since pasture grasses are cattle feed commodities, rather than food commodities, addition of grass forage and hay tolerances, will not contribute directly to dietary exposure.

i. *Food.* A dietary exposure assessment for chlorsulfuron was conducted using the Dietary Exposure Evaluation Model Versions 6.79 (Acute Module) and 6.76 (Chronic Module) of DEEM. Dietary exposure to chlorsulfuron, was based upon the following food commodities: Barley, oat, wheat, milk, and meat. For this assessment, it was assumed that 100% of the crop was treated with chlorsulfuron. Based on a comparison with the use profile for most other herbicides, this is an extremely conservative estimate. Chlorsulfuron is not an acute toxicant, however, for completeness an acute dietary risk assessment was conducted. The predicted acute exposure for the U.S. population subgroup was 0.0039 milligrams/kilograms bodyweight/day (mg/kg bwt/day) at the 95th percentile. The population subgroup with the highest predicted level of acute exposure at the 95th percentile was the children, age 1–6 years old subgroup with an exposure of 0.0084 mg/kg bwt/day. Based on a NOAEL of 165 mg/kg bwt/day from the repeated dose developmental toxicity study, and a 100–fold safety factor, the acute reference dose (aRfD) would be 1.65 mg/kg bwt/day. For the U.S. population, the predicted exposure at the 95th percentile is equivalent to 0.24% of the aRfD. For the population subgroup with the highest level of exposure (children 1–6 years old), the exposure at the 95th percentile would be equivalent to 0.51% of the aRfD. Because the predicted exposures expressed as percentages of the aRfD, are well below 100%, there is reasonable certainty that no acute effects would result from dietary exposure to chlorsulfuron.

The predicted chronic exposure for the U.S. population subgroup was 0.0013 mg/kg bwt/day. The population subgroup with the highest predicted level of chronic exposure was the children, age 1–6 year subgroup with an exposure of 0.0038 mg/kg bwt/day. Based on a chronic NOAEL of 5 mg/kg bwt/day and a 100–fold safety factor, the chronic reference dose (cRfD) would be 0.05 mg/kg bwt/day. For the U.S. population, the predicted exposure is equivalent to 2.5% of the cRfD. For the population subgroup with the highest level of exposure (children, 1–6 years old), the exposure would be equivalent to 7.7% of the cRfD. Because the predicted exposures, expressed as percentages of the cRfD, are well below 100%, there is reasonable certainty, that no chronic effects would result from dietary exposure to chlorsulfuron.

ii. *Drinking water.* Surface water exposure was estimated using the Generic Expected Environmental Concentration (GENEEC) model, a screening level model for determining concentrations of pesticides in surface water. GENEEC uses the soil/water partition coefficient, hydrolysis half life, and maximum label rate to estimate surface water concentration. In addition, the model contains a number of conservative underlying assumptions. Therefore, the drinking water concentrations derived from GENEEC for surface water are likely to be overestimated. Ground water exposures were estimated, using SCI-GROW and predicted levels were below those predicted by GENEEC; so GENEEC estimates were used below. EPA uses drinking water levels of comparison (DWLOCs) as a surrogate measure to capture risk associated with exposure to pesticides in drinking water. A DWLOC is the concentration of a pesticide in drinking water, that would be acceptable as an upper limit in light of total aggregate exposure to that pesticide from food, water, and residential uses. A DWLOC will vary, depending on the residue level in foods, the toxicity endpoint, and with drinking water consumption patterns and body weights for specific subpopulations.

iii. *Acute exposure and risk.* The acute DWLOCs are 58 ppm for the U.S. population and 16 ppm for the subpopulation with the highest exposure (infants < 1 year old). The estimated maximum concentration of chlorsulfuron in surface water (7.4 ppb derived from GENEEC, is much lower than the acute DWLOCs. Therefore, one can conclude with reasonable certainty that residues of chlorsulfuron in drinking water do not contribute significantly to the aggregate acute human health risk.

iv. *Chronic exposure and risk.* The chronic DWLOCs are 1.7 ppm for the U.S. population and 0.5 ppm for the subpopulation with the highest exposure (children 1–6 years old). These DWLOCs values are significantly higher than the GENEEC 56–day estimated environmental concentration of 7.3 ppb for chlorsulfuron in surface water. Therefore, one can conclude with reasonable certainty that residues of chlorsulfuron in drinking water, do not contribute significantly to the aggregate chronic human health risk.

v. *Acute risk.* Using the exposure assumptions described above, DuPont concluded with reasonable certainty that the aggregate exposure to chlorsulfuron to food will utilize less than 1% of the aRfD for all population subgroups. EPA generally has no concern for exposures below 100% of the aRfD because the aRfD represents the level at or below which a single day's aggregate exposure will not pose appreciable risks to human health. Despite the theoretical potential for exposure to chlorsulfuron in drinking water, the aggregate exposure (food + water) will not exceed the Agency's level of concern.

vi. *Chronic risk.* Using the exposure assumptions described above, DuPont has concluded that aggregate exposure to chlorsulfuron from food will utilize less than 8% of the cRfD for all population subgroups. EPA generally has no concern for exposures below 100% of the cRfD because the cRfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the theoretical potential for exposure to chlorsulfuron in drinking water, the aggregate exposure will not exceed 100% of the cRfD.

2. *Non-dietary exposure.* Chlorsulfuron is not registered for any use that could result in non-occupational or non-dietary exposure to the general population.

D. Cumulative Effects

Chlorsulfuron belongs to the sulfonylurea class of crop protection chemicals. While other structurally similar compounds in this class are registered herbicides, the herbicidal activity of sulfonylureas is due to the inhibition of acetolactate synthase (ALS), an enzyme found only in plants. This enzyme is part of the biosynthesis pathway leading to the formation of branched chain amino acids. Animals lack ALS and this biosynthetic pathway. This lack of ALS contributes to the relatively low toxicity of sulfonylurea herbicides in animals. There is no reliable information that would indicate or suggest that chlorsulfuron has any toxic effects on mammals that would be cumulative with those of any other chemical.

E. Safety Determination

1. *U.S. population.* The proposed analytical methods involve extraction, purification and concentration by reversed-phase solid-phase extraction. Extracts are analyzed by liquid chromatography/mass spectrometry

employing electrospray ionization (ESI-LC/MS).

Based on data and information submitted by DuPont, EPA previously determined that the establishment of tolerances of chlorsulfuron on wheat, barley, oats, milk and meat would protect the public health, including the health of infants and children. Establishment of a new tolerance of 11 ppm for chlorsulfuron on grass, forage and 19 ppm on grass, hay will not adversely impact public health. The proposed new tolerances are for feed commodities and will not directly impact human dietary intake. The proposed use on grass will only pose a small incremental increase in potential dietary burden for cattle. It has been determined that the existing meat and milk tolerances will accommodate this proposed new use on pasture grasses.

Based on the completeness and reliability of the toxicology database and using the conservative assumptions presented earlier, EPA has established a RfD of 0.05 mg/kg/day. This was based on the NOAEL for the chronic rat study, females (5.0 mg/kg/day) and a 100-fold safety factor. It has been concluded that the aggregate exposure was less than 8% of the RfD. Generally, exposures below 100% of the RfD are of no concern because it represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, there is reasonable certainty that no harm will result from aggregate exposures to chlorsulfuron residues.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of chlorsulfuron, data from the previously discussed developmental and multigeneration reproductive toxicity studies were considered.

Developmental studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during pre-natal development. Reproduction studies provide information relating to reproductive and other effects on adults and offspring from pre-natal and post-natal exposures to the pesticide. The studies with chlorsulfuron demonstrated no evidence of developmental toxicity at exposures below those causing maternal toxicity. This indicates that developing animals are not more sensitive to the effects of chlorsulfuron administration than adults.

FFDCA section 408 provides that EPA may apply an additional uncertainty 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 current toxicological data requirements, the data base for chlorsulfuron relative to pre-natal and post-natal effects for children is complete. In addition, the NOAEL of 5.0 mg/kg/day in the chronic rat study (and upon which the RfD is based) is much lower than the NOAELs defined in the reproduction and developmental toxicology studies. The sub-population with the highest level of exposure was children (1-6 years old), where exposure was approximately 7.7% of the RfD. Based on these conservative analyses, there is reasonable certainty that no harm will result to infants and children from aggregate exposures to chlorsulfuron.

F. International Tolerances

There are no Codex MRLs established for chlorsulfuron.

[FR Doc. 02–5446 Filed 3–7–02; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[FRL–7154–9]

Notice of Availability and Request for Public Comment: Proposed National Pollutant Discharge Elimination System (NPDES) General Permit for Discharges of Storm Water Discharges From Construction Activities in Indian Country Within the State of Wisconsin

AGENCY: Environmental Protection Agency, Region 5 (EPA).

ACTION: Extension of public comment period.

SUMMARY: Today's notice announces an extension of the public comment period regarding EPA's proposed National Pollutant Discharge Elimination System (NPDES) general permit for storm water discharges from construction activities in Indian country within the State of Wisconsin. The general permit is proposed to cover discharges within Indian country, including the following areas: Bad River Indian Reservation, Forest County Potawatomi Indian Reservation, Ho-Chunk Nation Indian Reservation, Lac Courte Oreilles Indian Reservation, Lac Du Flambeau Indian Reservation, Menominee Indian Reservation, Oneida Indian Reservation, Red Cliff Indian Reservation, Sokaogon (Mole Lake) Indian Reservation, St. Croix Indian Reservation, and the Stockbridge-Munsee Indian Reservation.

EPA published the proposed general permit in the **Federal Register** on December 21, 2001 (66 FR 65957–65961). The purpose of this notice is to correct a procedural oversight during the original notice and comment period. A public meeting will be held followed by a public hearing. The date and location is listed below:

Date: April 4, 2002.

Location: Bay Beach Wildlife Sanctuary, 1660 East Shore Drive, Green Bay, WI.

Time: 2 p.m. to 4 p.m. (Public meeting). 6 p.m. to 8 p.m. (Public Hearing).

DATES: Comment period on the proposed permit must be received by April 12, 2002. EPA will accept comments submitted in writing or transmitted electronically.

ADDRESSES: Comments on the draft permit may be sent to: Brian Bell, NPDES Programs Branch (WN–16J), U.S. Environmental Protection Agency, Region 5, 77 West Jackson Boulevard, Chicago, IL 60604. Comments may also be transmitted electronically to *bell.brianc@epa.gov.*

FOR FURTHER INFORMATION CONTACT: Brian Bell, at the above address or, via telephone at 312–886–0981.

Dated: February 22, 2002.

Thomas Poy,

Acting Director, Water Division, Region 5.

[FR Doc. 02–5602 Filed 3–7–02; 8:45 am]

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FEDERAL COMMUNICATIONS COMMISSION

[IB Docket 95–59; DA 02–248]

The Preemption of Local Zoning Regulation of Satellite Earth Stations

AGENCY: Federal Communications Commission.

ACTION: Notice.

SUMMARY: In this document , the International Bureau (''Bureau'') announces the list of the petitioners that did not respond to the October 2001 public notice, as set forth in the attached Appendix A. These parties may file a supplemental notice of their intent to pursue their respective petitions for reconsideration within 30 days after publication of this Public Notice in the **Federal Register**. The Commission intends to dismiss those petitions for reconsideration from parties that do not indicate intent to pursue their respective petitions for reconsideration. To ensure that each party who filed a petition for reconsideration to the 1996 Antenna Order has actual notice and an opportunity to respond.