governments ''to provide meaningful and timely input in the development of regulatory proposals containing significant unfunded mandates.''

Today's rule does not create an unfunded federal mandate on State, local or tribal governments. The rule does not impose any enforceable duties on these entities. Accordingly, the requirements of section 1(a) of Executive Order 12875 do not apply to this rule.

C. Executive Order 13084

Under Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19,1998), EPA may not issue a regulation that is not required by statute, that significantly or uniquely affects the communities of Indian tribal governments, and that imposes substantial direct compliance costs on those communities, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by the tribal governments. If the mandate is unfunded, EPA must provide OMB, in a separately identified section of the preamble to the rule, a description of the extent of EPA's prior consultation with representatives of affected tribal governments, a summary of the nature of their concerns, and a statement supporting the need to issue the regulation. In addition, Executive Order 13084 requires EPA to develop an effective process permitting elected and other representatives of Indian tribal governments ''to provide meaningful and timely input in the development of regulatory policies on matters that significantly or uniquely affect their communities.''

Today's rule does not significantly or uniquely affect the communities of Indian tribal governments. This action does not involve or impose any requirements that affect Indian Tribes. Accordingly, the requirements of section 3(b) of Executive Order 13084 do not apply to this rule.

X. Submission to Congress and the Comptroller General

The Congressional Review Act, 5 U.S.C. 801 *et seq.*, as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives and the Comptroller General of the United States prior to publication of the rule in the **Federal Register**. This is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

Environmental protection, Administrative practice and procedure, Agricultural commodities, pesticides and pests, Reporting and recordkeeping requirements.

Dated: August 21, 1998.

Stephen L. Johnson,

Deputy Director, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180 - [AMENDED]

1. The authority citation for part 180 continues to read as follows:

Authority: 21 U.S.C. 346a and 371.

2. Section 180.1202 is added to subpart D to read as follows:

§ 180.1202 Bacillus sphaericus; exemption from the requirement of a tolerance.

An exemption from the requirement of a tolerance is established for residues of the microbial pesticides, *Bacillus sphaericus* when used in or on all food crops.

[FR Doc. 98–24469 Filed 9–10–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Parts 180 and 185

[OPP–300709; FRL 6026–6]

RIN 2070–AB78

Sulfosate; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes new tolerances to replace recently-expired time-limited tolerances for residues of the herbicide sulfosate (the trimethylsulfonium salt of glyphosate, also known as glyphosate-trimesium) in or on cattle, goats, horses, hogs and sheep, in fat, meat by-products, and meat; in poultry fat, meat-by-products (except liver), meat and liver; in eggs; in milk; in corn stover (field and pop), grain (field and pop), and forage (field); in soybean forage, hay, and seed; and in aspirated grain fractions. Zeneca Ag Products requested these tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170). In addition, this regulation moves existing tolerances for prunes at 0.20 ppm, raisins at 0.20 ppm, and soybean hulls at 7.0 ppm from 40 CFR 185.5375 to 40 CFR 180.489.

DATES: This regulation is effective September 11, 1998. Objections and requests for hearings must be received by EPA on or before November 10, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, OPP–300709, must be submitted to: Hearing Clerk (1900), Environmental Protection Agency, Rm. M3708, 401 M St., SW., Washington, DC 20460. Fees accompanying objections and hearing requests shall be labeled ''Tolerance Petition Fees'' and forwarded to: EPA Headquarters Accounting Operations Branch, OPP (Tolerance Fees), P.O. Box 360277M, Pittsburgh, PA 15251. A copy of any objections and hearing requests filed with the Hearing Clerk identified by the docket control number, OPP–300709, must also be submitted to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring a copy of objections and hearing requests to Rm. 119, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Copies of objections and hearing requests must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Copies of objections and hearing requests will also be accepted on disks in WordPerfect 5.1/6.1 or ASCII file format. All copies of objections and hearing requests in electronic form must be identified by the docket control number OPP–300709. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic copies of objections and hearing requests on this rule may be filed online at many Federal Depository Libraries.

FOR FURTHER INFORMATION CONTACT: By mail: Jim Tompkins, 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: Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, 703–305–5697; e-mail: tompkins.jim@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of March 8, 1996 (61 FR 9355) (FRL 5353–4), time-limited tolerances were established for sulfosate on corn and animal commodities (listed below). In the **Federal Register** of April 10, 1996 (61 FR 15899) (FRL 5782–9), time-limited tolerances were established for unprocessed soybean commodities and aspirated grain fractions (listed below).

In the **Federal Register** of March 4, 1998 (63 FR 10614) (FRL 5772–6), EPA, issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(e) announcing the filing of a pesticide petition for tolerance by Zeneca Ag Products, 1800 Concord Pike, P. O. Box 15458, Wilmington, DE 19850–5458. This notice included a summary of the petition prepared by Zeneca Ag Products, the registrant. There were no comments received in response to the notice of filing.

The petition 0F3860 requested that 40 CFR 180.489 be amended by removing the expiration date of April 10, 1998, for residues of the herbicide sulfosate (glyphosate-trimesium; sulfonium, trimethyl salt with *N*-(phosphonomethyl)glycine (1:1)), in or on soybean forage (2.00 ppm, of which no more than 1 ppm is trimethylsulfonium (TMS)), soybean aspirated grain fractions (210.00 ppm, of which no more than 60 ppm is TMS), soybean hay (5.00 ppm, of which no more than 2 ppm is TMS), and soybean seed (3.00 ppm of which no more than 1 ppm is TMS). The petition 9F3796 requested that 40 CFR 180.489 be amended by removing the expiration date of March 9, 1998 for residues of sulfosate in or on cattle, goat, hog, horse, sheep and poultry fat (0.10 ppm), meat by products (1.00 ppm), and meat (0.20 ppm); poultry liver (0.05 ppm), poultry meat by-products (0.10 ppm), and poultry meat (0.05 ppm); corn fodder (0.30, of which no more than 0.20 is trimethylsulfonium TMS)), corn forage (0.10 ppm), and corn grain (0.20 ppm, of which no more than 0.10 ppm is TMS); milk (0.20 ppm); and eggs (0.02 ppm).

In the corn tolerances for this action, the commodity term ''stover'' replaces the older term ''fodder'' in keeping with current EPA policy for naming this commodity. In this action, the previous tolerance for ''soybean aspirated grain fractions'' is replaced with the tolerance for ''aspirated grain fractions''. The term ''soybean aspirated grain fractions'' was printed in error in the April 10, 1996 FR notice (61 FR 15899); aspirated grain fractions typically contain more than one type of grain and typically contain both soybeans and corn.

This action also moves tolerances for prunes, raisins, and soybean hulls from 40 CFR 185.5375 to 40 CFR 180.489. The Food Quality Protection Act (FQPA) amended the Federal Food, Drug and Cosmetic Act (FFDCA) to consolidate pesticide tolerances for raw and processed agricultural commodities under FFDCA section 408(j)(2). Prior to this change, raw agricultural commodity tolerances were established according to FFDCA section 408 and processed commodities were established according to FFDCA section 409. As a result of the change in the regulations governing FFDCA, all new tolerances for both raw and agricultural commodities are established according to FFDCA section 408(j)(2) in 40 CFR part 180. When 40 CFR part 180 is amended as to a specific pesticide, it is EPA's policy to move existing related regulations governing residues of that pesticide on processed agricultural commodities from 40 CFR parts 185 and 186 and place them in part 180. Ultimately, EPA will amend all tolerance regulations so that all tolerances are listed in 40 CFR part 180.

I. Risk Assessment and Statutory Findings

New section 408(b)(2)(A)(i) of the FFDCA allows EPA to establish a tolerance (the legal limit for a pesticide chemical residue in or on a food) only if EPA determines that the tolerance is ''safe.'' Section 408(b)(2)(A)(ii) defines ''safe'' to mean that ''there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.'' This includes exposure through drinking water and in residential settings, but does not include occupational exposure. Section 408(b)(2)(C) requires EPA to give special consideration to exposure of infants and children to the pesticide chemical residue in establishing a tolerance and to ''ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the pesticide chemical residue. . . .''

EPA performs a number of analyses to determine the risks from aggregate exposure to pesticide residues. First, EPA determines the toxicity of pesticides based primarily on toxicological studies using laboratory animals. These studies address many adverse health effects, including (but not limited to) reproductive effects, developmental toxicity, toxicity to the nervous system, and carcinogenicity. Second, EPA examines exposure to the pesticide through the diet (e.g., food and drinking water) and through exposures that occur as a result of pesticide use in residential settings.

A. Toxicity

1. *Threshold and non-threshold effects.* For many animal studies, a dose response relationship can be determined, which provides a dose that causes adverse effects (threshold effects) and doses causing no observed effects (the ''no-observed effect level'' or ''NOEL'').

Once a study has been evaluated and the observed effects have been determined to be threshold effects, EPA generally divides the NOEL from the study with the lowest NOEL by an uncertainty factor (usually 100 or more) to determine the Reference Dose (RfD). The RfD is a level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. An uncertainty factor (sometimes called a ''safety factor'') of 100 is commonly used since it is assumed that people may be up to 10 times more sensitive to pesticides than the test animals, and that one person or subgroup of the population (such as infants and children) could be up to 10 times more sensitive to a pesticide than another. In addition, EPA assesses the potential risks to infants and children based on the weight of the evidence of the toxicology studies and determines whether an additional uncertainty factor is warranted. Thus, an aggregate daily exposure to a pesticide residue at or below the RfD (expressed as 100% or less of the RfD) is generally considered acceptable by EPA. EPA generally uses the RfD to evaluate the chronic risks posed by pesticide exposure. For shorter term risks, EPA uses a RfD approach or calculates a margin of exposure (MOE) by dividing the estimated human exposure into the NOEL from the appropriate animal study. Commonly, EPA finds MOEs lower than 100 to be unacceptable. This 100-fold MOE is based on the same rationale as the 100-fold uncertainty factor.

Lifetime feeding studies in two species of laboratory animals are conducted to screen pesticides for cancer effects. When evidence of increased cancer is noted in these studies, the Agency conducts a weight of the evidence review of all relevant toxicological data including short-term and mutagenicity studies and structure activity relationship. Once a pesticide has been classified as a potential human carcinogen, different types of risk assessments (e.g., linear low dose extrapolations or MOE calculation based

on the appropriate NOEL) will be carried out based on the nature of the carcinogenic response and the Agency's knowledge of its mode of action.

2. *Differences in toxic effect due to exposure duration.* The toxicological effects of a pesticide can vary with different exposure durations. EPA considers the entire toxicity data base, and based on the effects seen for different durations and routes of exposure, determines which risk assessments should be done to assure that the public is adequately protected from any pesticide exposure scenario. Both short and long durations of exposure are always considered. Typically, risk assessments include ''acute,'' ''short-term,'' ''intermediate term,'' and ''chronic'' risks. These assessments are defined by the Agency as follows.

Acute risk, by the Agency's definition, results from 1–day consumption of food and water, and reflects toxicity which could be expressed following a single oral exposure to the pesticide residues. High end exposure to food and water residues are typically assumed.

Short-term risk results from exposure to the pesticide for a period of 1–7 days, and therefore overlaps with the acute risk assessment. Historically, this risk assessment was intended to address primarily dermal and inhalation exposure which could result, for example, from residential pesticide applications. However, since enaction of FQPA, this assessment has been expanded to include both dietary and non-dietary sources of exposure, and will typically consider exposure from food, water, and residential uses when reliable data are available. In this assessment, risks from average food and water exposure, and high-end residential exposure, are aggregated. High-end exposures from all three sources are not typically added because of the very low probability of this occurring in most cases, and because the other conservative assumptions built into the assessment assure adequate protection of public health. However, for cases in which high-end exposure can reasonably be expected from multiple sources (e.g. frequent and widespread homeowner use in a specific geographical area), multiple high-end risks will be aggregated and presented as part of the comprehensive risk assessment/characterization. Since the toxicological endpoint considered in this assessment reflects exposure over a period of at least 7 days, an additional degree of conservatism is built into the assessment; i.e., the risk assessment nominally covers 1–7 days exposure, and the toxicological endpoint/NOEL is selected to be adequate for at least 7 days of exposure. (Toxicity results at lower levels when the dosing duration is increased.)

Intermediate-term risk results from exposure for 7 days to several months. This assessment is handled in a manner similar to the short-term risk assessment.

Chronic risk assessment describes risk which could result from several months to a lifetime of exposure. For this assessment, risks are aggregated considering average exposure from all sources for representative population subgroups including infants and children.

B. Aggregate Exposure

In examining aggregate exposure, FFDCA section 408 requires that EPA take into account available and reliable information concerning exposure from the pesticide residue in the food in question, residues in other foods for which there are tolerances, residues in groundwater or surface water that is consumed as drinking water, and other non-occupational exposures through pesticide use in gardens, lawns, or buildings (residential and other indoor uses). Dietary exposure to residues of a pesticide in a food commodity are estimated by multiplying the average daily consumption of the food forms of that commodity by the tolerance level or the anticipated pesticide residue level. The Theoretical Maximum Residue Contribution (TMRC) is an estimate of the level of residues consumed daily if each food item contained pesticide residues equal to the tolerance. In evaluating food exposures, EPA takes into account varying consumption patterns of major identifiable subgroups of consumers, including infants and children. The TMRC is a ''worst case'' estimate since it is based on the assumptions that food contains pesticide residues at the tolerance level and that 100% of the crop is treated by pesticides that have established tolerances. If the TMRC exceeds the RfD or poses a lifetime cancer risk that is greater than approximately one in a million, EPA attempts to derive a more accurate exposure estimate for the pesticide by evaluating additional types of information (anticipated residue data and/or percent of crop treated data) which show, generally, that pesticide residues in most foods when they are eaten are well below established tolerances.

Percent of crop treated estimates are derived from federal and private market survey data. Typically, a range of estimates are supplied and the upper end of this range is assumed for the exposure assessment. By using this upper end estimate of percent of crop treated, the Agency is reasonably certain that exposure is not understated for any significant subpopulation group. Further, regional consumption information is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups, to pesticide residues. For this pesticide, the most highly exposed population subgroups (females, infants, and children) were not regionally based.

II. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action, EPA has sufficient data to assess the hazards of sulfosate and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerance for residues of sulfosate on cattle, goats, horses, hogs and sheep at 0.10 ppm in fat, at 1.00 ppm in meat by-products, and at 0.20 ppm in meat; in poultry at 0.05 ppm in fat, meat-by-products (except liver), and meat, and at 0.10 ppm in liver; in eggs at 0.02 ppm; in milk at 0.20 ppm; in corn at 0.30 ppm (of which no more than 0.20 ppm is TMS) in stover (field and pop), at 0.20 ppm (of which no more than 0.10 ppm is TMS) in grain (field and pop), at 0.10 ppm in forage (field); in soybeans at 2.00 ppm (of which no more than 1.0 ppm is TMS) in forage, at 5.00 ppm (of which no more than 2.0 ppm is TMS) in hay, and at 3.00 (of which no more than 1.0 ppm is TMS) ppm in seed; and in aspirated grain fractions at 210 ppm (of which no more than 60 ppm is TMS). EPA's assessment of the dietary exposures and risks associated with establishing the tolerance follows.

A. Toxicological Profile

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

Several acute toxicity studies were performed, placing technical-grade sulfosate in Toxicity Category III. The acute toxicity data for sulfosate show that this chemical is not acutely toxic by the oral, inhalation, and dermal routes

of exposure. Sulfosate technical is, however, a slight dermal sensitizer.

In a subchronic feeding study, 6 week old CrL: CD(SD)BR Sprague-Dawley rats were treated with Sulfosate technical at doses of 0, 150, 350, 800 or 2,000 ppm sulfosate in their diet (males for 90 days & females for 96 days). At 2,000 ppm in males (88 mg/kg/day) there was a significant overall decrease in body weight gain of 22%. At 2,000 ppm, the females exhibited some sporadic and minimal decreases in body weight (6% at week 2, 8% at week 11, 21% at week 13) which were due to a decrease in food consumption and is not used to set a lowest effect level (LOEL). No significant changes were observed in clinical chemistry, hematology, clinical observations, organ weight, and macroscopic/microscopic histopathology. The systemic no effect level (NOEL) is 800 ppm in males (36 mg/kg/day) and 2,000 ppm (108 mg/kg/day) in females. The systemic LOEL is 2,000 ppm in males (88 mg/kg/day), based on significant overall decrease in body weight gain of 22%. The maximum tolerated dose (MTD) was achieved only in male rats.

Two subchronic toxicity studies on dogs were conducted. In one subchronic oral study, beagle dogs were treated with Sulfosate technical at doses of 0, 2, 10 or 50 mg/kg/day. The dose volume was 0.5 milliliter per kilogram body weight (ml/kg b.w.) by oral gavage (5 days/week) for 45–50 days. The NOEL is 10 mg/kg/day for both males and females. The LOEL is 50 mg/kg/day for both males and females, based on significant earlier onsets and increased incidence of salivation and emesis. No significant change was observed in body weight, food consumption, urinalysis, organ weights, macroscopic/microscopic histopathology, hematology, and clinical chemistry including cholinesterase activity. In another subchronic toxicity study, Sulfosate was administered to 4 male and 4 female beagle dogs by gelatin capsule at doses of 0, 10, 25, or 50 mg/kg/day for at least 90 days. Evaluations included clinical observations, body weight, food consumption, clinical pathology, organ weights and gross and microscopic histopathology. There were no effects on food consumption, body weight, clinical pathology, organ weights or histopathology. Observed at 50 mg/kg/day in both sexes was salivation at dosing (weeks 2–14) and/or salivation (weeks 1–13) either consistently or intermittently, and resisting dosing (weeks 6–13) occasionally. A female in the 50 mg/kg/day group was sacrificed on day 2 after being found cold and recumbent and replaced with another female dog. The dose was lowered to 40 mg/kg/day in another female dog (50 mg/kg/day group) for most of the remainder of the study following two incidents of tremors, recumbency, and voluntary paddling of the limbs. One high dose male had a unilateral cataract. The LOEL is 50 mg/kg/day, based on clinical signs of neurotoxicity in the females. The NOEL is 25 mg/kg/day.

Two 21–day dermal studies were conducted. In one 21–day dermal study, Rabbits (New Zealand White) were treated with sulfosate soluble concentrate (51.2% a.i.), Sulfosate at doses of 0, 10, 100, 1,000 mg/kg/day, 6 hrs/day, 5 days/wk for 3 weeks. There was no systemic toxicity at any dose. There was mild erythema at application sites in all sulfosate-treated groups. The systemic NOEL is 1,000 mg/kg/day, the highest dose tested (HDT). In another 21–day dermal study, sulfosate emulsifiable concentrate (39.8% a.i.) was applied to the skin of rats (Alpk: AP (Wistar derived), 5/sex/group) at doses of 25, 250, 1,000 mg/kg in 0.0021, 0.027, and 0.0826 ml/100 g body wt. At 25 and 1,000 mg/kg/day (not 250 mg/kg/day) there was a slight increase in testes weight with normal histology (toxicological significance is unclear). There was occasional sciatic nerve fiber degeneration (1 male and 2 females out of a total of 10) at 1,000 mg/kg/day. There was occasional sciatic nerve fiber degeneration (1/5 males, 2/5 females) at 1,000 mg/kg/day with none in controls. Dermal irritation occurred in male rats at 1,000 mg/kg/day including scabbing, erythema, edema and desquamation. There were no histological changes. The systemic LOEL was 1,000 mg/kg/day based on sciatic nerve findings. The NOEL was 250 mg/kg/day.

In a feeding/carcinogenicity study, 60/sex/group Sprague-Dawley (Crl: CD SD BR) rats were tested with sulfosate soluble concentrate (56.2% a.i.) at dose levels of 0 (basal diet, no vehicle), 0 (basal diet plus 1% propylene glycol), 100, 500 or 1,000 ppm a.i. (male - 0, 4.2, 21.2, or 41.8; female - 0, 5.4, 27.0, or 55.7) for 2 years. Rats may have tolerated higher dose levels. At 1,000 ppm there were decreases in bodyweight in both males and females and an increase in incidences of chronic laryngeal and nasopharyngeal inflammation in males. Bodyweight decrease was secondary to the decrease in food consumption. The LOEL and NOEL were at or above 1,000 ppm (41.8 and 55.7 mg/kg/day for males and females, respectively). There was no evidence of carcinogenicity in this study at the doses tested. The study is considered acceptable based on the results of a subchronic and reproduction study. The high dose for a feeding/carcinogenicity study should be near, but not necessarily at, a dose that would produce well defined toxicity. The subchronic rat study indicated well defined toxicity at 2,000 ppm (only twice the high dose in the feeding/carcinogenicity study), a dose that is adequate for estimating a maximum tolerated dose (MTD). Therefore, 1,000 ppm in the feeding/carcinogenicity study is considered a reasonable extrapolation from the subchronic toxicity study results. In addition, at 2,000 ppm in the reproduction study there is well defined toxicity with some evidence of toxicity, although less severe, at 800 ppm. Therefore, it is believed that sulfosate was adequately tested for carcinogenicity in the rat.

In a chronic oral gavage study, beagle dogs (5/sex/dose) were treated with sulfosate soluble concentrate (56.2% a.i.) for 1 year at doses of 0, 2, 10, or 50 mg kg/day. Signs of toxicity were limited to the 50 mg/kg/day group females and included transient salivation (1/5 at 10 mg/kg/day and 5/5 at 50 mg/kg/day) and emesis (single episodes in 3/5 dogs). The decreased lactic acid dehydrogenase (LDH) in females at 12 months is of questionable biological significance. The high dose was however, supported by subchronic studies where transient salivation and emesis again occurred at 50 mg/kg/day in a 90 day study and at 75 mg/kg/day in a 28 day study; with death occurring within 3 days at 150 mg/kg/day in the 28 day study. The LOEL is 50 mg/kg/day based on salivation and emesis and support from shorter term studies also with emesis and salivation. The NOEL is 10 mg/kg/day.

In a feeding carcinogenicity study, mice (60/sex/dose) were given sulfosate technical (56.17% a.i.) in the diet at concentrations of 0a (dietary control), 0b (vehicle control), 100, 1,000 and 8,000 ppm (males at 0, 0, 11.7, 118, or 991 mg/kg/day; and females at 0, 0, 16.0, 159, or 1,341 mg/kg/day) for 2 years. The only signs of toxicity occurred at 8,000 ppm and included (in both sexes) decreased body weight (about 10% lower than controls) and weight gain (about 50% lower than controls). Decreased food consumption (0 to 15% lower than controls in both sexes) was responsible only in part for the decreased weight gain. In addition, there was increased incidence of white matter degeneration in the lumbar region of the spinal cord (males only) (2, 3, 4, 4, 79% response, controls to high dose), and increased incidence of epithelial hyperplasia of duodenum (females only) (10, 13, 16, 15, 24%

response, controls to high dose). The systemic LEL is 8,000 ppm (991, 1,340 mg/kg/day for males and females) based on decreased body weight & food consumption (both sexes), increased incidence of white matter degeneration in lumbar bar region of spinal cord (males only), and increased incidence of epithelial hyperplasia of duodenum (females only). The systemic NOEL is 1000 ppm (118, 159 mg/kg/day for males and females). This study was tested to adequate doses based on decreased body weight and weight gain. There was no evidence of carcinogenicity in this study at the doses tested.

In a developmental toxicity study, rats (25/dose) were treated with sulfosate soluble concentrate (19.2% a.i.) by gavage on gestation days 6 through 20 at dose levels of 0, 30, 100, or 333 mg/kg/day. The test material was dissolved in water and administered in a volume of 5 ml/kg. Treatment related effects were limited to the high dose dams and included decreased body weight (17% less than the control), body weight gain and feed consumption. There was also salivation, chromorhinorrhea and lethargy after dosing in this group ($p < 0.05$). The Maternal LOEL is 333 mg/kg/day based on decreased body weight, feed consumption and body weight gain along with increased incidences of salivation, chromorhinorrhea, and lethargy after dosing. The Maternal NOEL is 100 mg/kg/day. Developmental signs of toxicity were limited to the high dose and included decreased fetal body weight (5.0, 4.9, 4.9, 4.2 gm, controls to high dose). The Developmental toxicity LOEL is 333 mg/kg/day based on decreased fetal body weight. The Developmental toxicity NOEL is 100 mg/kg/day.

In a developmental toxicity study, New Zealand white rabbits (15/group except 21 at the high dose) were treated by gavage with sulfosate soluble concentrate (56.2% ai) from gestation days 7–19. The test material was dissolved in water and administered in a volume of 2 ml/kg at dose levels of 0, 10, 40 or 100 mg/kg/day. The Maternal LOEL is 100 mg/kg/day (6 deaths in 17 pregnant does, 4 abortions in the 11 survivors along with decreased body weight, feed consumption and body weight gain). The Maternal NOEL is 40 mg/kg/day. The developmental LOEL is 100 mg/kg/day based on decreased number of live fetuses/doe for 7 surviving rabbits (5.4 versus 7.4 in controls), 4 rabbits aborted their litters. Having only 7 litters does not give a sufficiently high number of animals to absolutely conclude that no developmental toxicity is occurring, particularly in light of the massive losses to death and abortions. The developmental NOEL is 40 mg/kg/day.

In a 2-generation reproduction study, 20 male and 30 female/group Sprague-Dawley rats received sulfosate soluble concentrate (19.2% a.i.) at dose levels of 0, 150, 800, or 2,000 ppm in the diet (average for P_0 and P_1 - males - 0, 6.0, 35, 88.5 mg/kg/day; females - 0, 8, 41, 98 mg/kg/day). The systemic LEL is 800 ppm (35 and 41 mg/kg/day for males and females) based on a decrease in absolute and sometimes relative organ weights in both generations (thymus, heart, kidney and liver) at 800 and 2,000 ppm and a decrease in body weights and body weight gains during the premating period at 2,000 ppm. The Systemic NOEL is 150 ppm (6 and 8 mg/kg/day for males and females). The reproductive/developmental LOEL is 800 ppm (35 and 41 mg/kg/day for males and females) is based on decreased litter size in F_{0a} and F_{1b} litters at 2,000 ppm and on decrease in mean pup weights during lactation in second litters at 800 ppm & in all litters at 2,000 ppm. The reproductive/developmental NOEL is 150 ppm (6 and 8 mg/kg/day for males and females).

In an acute neurotoxicity study, white leghorn chickens (6 hens/group in control groups, 8 hens/group in treated groups) were treated with technical sulfosate (56.9% a.i.) by gavage at doses of 0, 500 or 5,000 mg/kg in 5 ml/kg water. Tri-ortho-cresylphosphate (TOCP, 500 mg/kg) was the positive control. Each animal was dosed twice during study; day 1 and day 22. Each animal was evaluated up to day 41 (or 42). At 500 mg/kg there was diarrhea starting a few days after each dosing, lasting for 2–3 days. At 5,000 mg/kg there was diarrhea, changes in comb appearance, early decreased food consumption and decrease in egg production. No indications of neurotoxicity were observed. The positive control indicated the appropriate clinical sings of toxicity, increased ataxia and microscopic observations for an organophosphate. The NOEL for systemic toxicity was 500 mg/kg. The LEL for systemic toxicity was 5,000 mg/kg.

In an acute neurotoxicity study, sulfosate technical (59.4% a.i.) was used to treat Alpk: APfsD rats, 10/sex/dose by gavage at 1 ml/100 g bw with doses of 0, 30, 100 or 300 mg/kg. Adequate positive control data were provided. At 300 mg/kg there was death, ptosis, decreased activity, decreased splay reflex, upward curvature of spine, chromodacryorrhea, staining around the nose, decreased bodyweight and food consumption (males), shaking, sides pinched in, signs of urinary incontinence, irregular breathing, hunched posture, abnormal or staggering gait, increased time to tail flick, decreased landing foot splay, decreased forelimb grip strength, decreased hindlimb grip strength, decreased motor activity. There was no microscopic evidence of neurotoxicity. There were no indications of neurotoxicity below a lethal dose. The LEL was 300 mg/kg based on mortality, neurologic signs described above and decreased body weight and food consumption. The NOEL was 100 mg/kg.

Technical sulfosate (59.4% a.i.) was tested in a 90 day neurotoxicity feeding study in Alpk: APfSD rats. Rats (12/sex/group) received either 0, 200, 600, or 2,000 ppm (0, 15.6, 47.6 or 153.2 mg/kg/day for males; 0, 18.2, 54.4 or 171.0 mg/kg/day for females) in the diet. Six/sex/dose group received complete necropsy and neurohistopathology. Positive control data were provided. The other 6/sex/dose were perfused and the neurohistopathology carried out. Clinical signs of toxicity, body weights, food consumption, functional battery, motor activity and neuropathology parameters were measured and recorded regularly. Positive control data were provided. At 2,000 ppm, decreased body weights (16% for males and 9% for females), food consumption and utilization were observed. In addition, mean forelimb grip strength values for high dose females were statistically significantly decreased over the values for the controls during weeks 5–14 (75 – 82% of controls). There was no microscopic evidence of neurotoxicity. The significance of the decreased grip strength as a neurotoxicological effect is less certain since there were no effects in mean hindlimb grip strength for high dose females, in either of the mean grip strength values at any time period for males, in any of the other functional battery parameters, in motor activity values or in neuropathology microscopic examinations for either sex. However, it occurred at all time points, was statistically significant, and signs of neurotoxicity occur in other studies. The LEL is 2,000 ppm (153.2 mg/kg/day) based on decreases in mean body weight, food consumption, food utilization and mean forelimb grip strength values. The NOEL is 600 ppm (47.6 mg/kg/day).

Several mutagenicity tests were conducted. In some of the *in vitro* mutagenicity tests (forward mutation/mouse lymphoma cells, structural chromosomal aberrations/CHO cells), sulfosate induced a false positive mutagenic effect. A common feature of

these tests was that the pHs of the test incubation media were acidic (pH 5.67–7.07) due to the addition of sulfosate. These positive results were no longer observed when the pH was readjusted to a more physiological level (pH 7.4) before the mutagenicity tests were conducted. Based on the available mutagenicity studies, there are no concerns for mutagenicity at this time.

In a metabolism study, rats were treated with sulfosate soluble concentrate (^{14}C labeled). Radiolabelled trimethylsulfonium ion (TMS) was rapidly excreted unmetabolized in urine and feces; the principal sites of localization of TMS are adrenals, kidneys, bladder, liver, thyroid and stomach.

In a metabolism study, rats were treated with sulfosate (^{14}C-labeled on the anionic part of the molecule, 56.1% ai). Intravenous (IV) or oral ^{14}C-sulfosate was rapidly excreted; over a 5 day period most (86–95%) of the administered dose was excreted in the urine & feces. IV treated male & females eliminated 90% of the administered dose in urine. Absorption of ^{14}C-sulfosate was incomplete by the oral route; most groups eliminated 47–57% of the administered dose in the urine and 36–42% in the feces. Females treated with a high dose eliminated less in the urine (36% of dose) and more in the feces (54% of dose). There was negligible ^{14}C-carbon dioxide ($^{14}CO2$) elimination. Tissue ^{14}C residues were < 0.32% of administered dose. Carcass ^{14}C residues were < 2.2% of administered dose (mostly in bones, 3–7 ppm in low dose rats & 19–32 ppm in high dose rats). Most excreted radioactivity (77–96% of fecal; 80–95% of urinary) was unchanged anion (carboxymethylamino-methylphosphonate). One fecal metabolite (repeated dose females; 8.5% of fecal radioactivity) was aminomethyl phosphonic acid. Several minor unidentified ($\leqq$ 3% of total urinary/fecal radioactivity) metabolites were recovered. The low dose was 25 mg/kg. At the high dose of 250 mg/kg, toxic signs were lethargy, moderate to severe depression, tremors, dehydration, and decreased food consumption in 2 – 5 rats (total of 10 rats tested). Recovery was within 72 hours.

B. Toxicological Endpoints

1. *Acute toxicity.* An acute NOEL of 100 mg/kg was determined based on mortality, decreased body weight and food consumption, and neurotoxicity at 300 mg/kg (LOEL) from an acute rat neurotoxicity study. An acute RFD of 1.0 mg/kg was calculated by dividing the 100 mg/kg NOEL by the uncertainty factor of 100 (10x for inter-species extrapolation and 10x for intra-species variations). Based on FQPA, EPA has determined that an additional safety factor of 3x must be retained for the acute dietary assessment to protect infants and children. Without the 3x safety factor, the level of concern is dietary consumption above the level of 100% of the RfD. With the 3x safety factor, the level of concern is consumption above the level of 33% of the acute RfD.

2. *Short - and intermediate - term toxicity.* There are currently no residential uses for suslfosate; therefore, assessment of short- and intermediate-term toxicity is not necessary for the purpose of establishing sulfosate tolerances.

3. *Chronic toxicity.* EPA has established the RfD for sulfosate at 0.10 milligrams/kilogram/day (mg/kg/day). This RfD is based on an oral NOEL of 10 mg/kg/day (LOEL of 50 mg/kg/day) from a chronic oral gavage study in dogs and an uncertainty factor of 100. Based on FQPA, EPA has determined that an additional safety factor of 3x must be retained for the chronic dietary assessment to protect infants and children. Without the 3x safety factor, the level of concern is dietary consumption above the level of 100% of the RfD. With the 3x safety factor, the level of concern is consumption above the level of 33% of the chronic RfD.

4. *Carcinogenicity.* Sulfosate was classified as a ''Group E'' carcinogen (no evidence for carcinogenicity in humans) based on the lack of evidence of carcinogenicity in mice and rats at doses that were judged to be adequate to assess the carcinogenic potential and the ''Guidelines for Carcinogen Risk Assessment'' [51 FR 33992] for classifying the weight-of-evidence for carcinogenicity.

C. Exposures and Risks

1. *From food and feed uses.* Tolerances have been previously established (40 CFR 180.489) for the residues of sulfosate, in or on a variety of raw agricultural commodities. Time-limited tolerances for soybeans expired on April 10, 1998, and time limited tolerances for corn, ruminants, poultry, milk, and eggs expired on March 9, 1998. Risk assessments were conducted by EPA to assess dietary exposures and risks from sulfosate as follows:

i. *Acute exposure and risk.* Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a one day or single exposure. An acute dietary (food only) risk assessment was conducted for sulfosate. The exposure to the most sensitive population subgroup, in this instance non-nursing infants, was 9.7% of the acute RfD (1.0 mg/kg bwt/day). Based on FQPA, EPA has determined that an additional safety factor of 3x must be retained for the acute dietary assessment to protect infants and children. Without the 3x safety factor, the level of concern is dietary consumption above the level of 100% of the RfD. With the 3x safety factor, the level of concern is consumption above the level of 33% of the acute RfD. Therefore, the acute dietary risk due to food does not exceed the level of concern.

ii. *Chronic exposure and risk.* An chronic dietary (food only) risk assessment was conducted for sulfosate. This risk assessment assumed 100% of the crops with existing tolerances plus those established in this notice were treated and that residues were consumed at the theoretical Maximum Residue Contribution (TMRC, the level of residues consumed daily if each food item contained pesticide residues equal to the tolerance). The exposure to the most sensitive population subgroup, in this instance children 1 to 6 years old, was 20.3% of the chronic RfD (0.1 mg/kg bwt/day). Based on FQPA, EPA has determined that an additional safety factor of 3x must be retained for the acute dietary assessment to protect infants and children. Without the 3x safety factor, the level of concern is dietary consumption above the level of 100% of the RfD. With the 3x safety factor, the level of concern is consumption above the level of 33% of the acute RfD. Therefore, the chronic dietary risk due to food does not exceed the level of concern.

2. *From drinking water.* Results from computer modeling indicate that sulfosate in groundwater will not contribute significant residues in drinking water as a result of sulfosate use at the recommended maximum annual application rate (1 application at 4.75 lbs., a.i., $acre^{-1}$). The computer model uses conservative numbers, therefore it is unlikely that groundwater concentrations would exceed the estimated concentration of 0.00224 ppb, and sulfosate should not pose a threat to ground water.

The surface water estimates are based on an exposure modeling procedure called GENEEC (Generic Expected Environmental Concentration). The assumptions of 1 application of 4.75 lbs., a.i., $acre^{-1}$ resulted in calculated estimated maximum concentrations of 125 ppb (acute, based on the highest 56 day value) and 35 ppb (chronic, average). GENEEC modeling procedures assumed that sulfosate was applied to a

10-hectare field that drained into a 1-hectare pond, 2-meters deep with no outlet for all crops.

As a conservative assumption, because sulfosate residues in ground water are expected to be insignificant compared to surface water, EPA assumed that 100% of drinking water consumed was derived from surface water in all drinking water exposure and risk calculations.

To calculate the maximum acceptable acute and chronic exposures to sulfosate in drinking water, the dietary food exposure (acute or chronic) was subtracted from 33% of the appropriate (acute or chronic) RfD. DWLOCs were then calculated using the maximum acceptable acute or chronic exposure, default body weights (70 kg - adult, 10 kg - child) and drinking water consumption figures (2 litres - adult, 1 litre - child).

i. *Acute exposure and risk*. OPP has calculated drinking water levels of concern (DWLOCs) for acute exposure to be 9,740 ug/l parts per billion (ppb) for U.S. population, 2,360 ug/l (ppb) for non-nursing infants (<1 year old), and 2600 ug/l (ppb) for children (1–6 years old). These levels include the FQPA additional safety factor of 3x to protect infants and childern. The estimated maximum concentration of sulfosate in surface water of 125 ppb (highest 56 day value) is less than all of the calculated acute DWLOCs. Therefore, taking into account the present uses plus uses on corn and soybeans, EPA concludes with reasonable certainty that acute exposure to residues of sulfosate in drinking water (when considered along with other sources of exposure for which EPA has reliable data) would not result in unacceptable levels of aggregate human health risk at this time.

ii. *Chronic exposure and risk*. OPP has calculated DWLOCs for chronic (non-cancer) exposure to be 925 ug/l (ppb) for U.S. population and 130 ug/l (ppb) for the most sensitve population group, in this instance children 1 to 6 years old. These levels include the FQPA additional safety factor of 3x to protect infants and childern. The estimated concentration 35 ppb (chronic, average) of sulfosate in surface water of is less than all of the calculated chronic DWLOCs. Therefore, taking into account the present uses plus uses on corn and soybeans, EPA concludes with reasonable certainty that chronic exposure to residues of sulfosate in drinking water (when considered along with other sources of exposure for which EPA has reliable data) would not result in unacceptable levels of aggregate human health risk at this time.

3. *From non-dietary exposure*. Sulfosate is currently not registered for use on any residential non-food sites: Therefore, residential exposure to sulfosate residues will be through dietary exposure only.

4. *Cumulative exposure to substances with common mechanism of toxicity*. Sulfosate is structurally similar to glyphosate. Further, other pesticides may have common toxicity endpoints with sulfosate. Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' The Agency believes that ''available information'' in this context might include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on chemical specific data, much of which may not be presently available.

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

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

D. Aggregate Risks and Determination of Safety for U.S. Population

1. *Acute risk*. Since there are no residential uses for sulfosate, the acute aggregate exposure only includes food and water. For the U.S. population, 5.8% of the acute RfD is occupied by dietary (food) exposure. The estimated average concentrations of sulfosate in surface and ground water are less than EPA's levels of concern for sulfosate in drinking water as a contribution to acute aggregate exposure. The above calculations include the FQPA safety factor of 3x. Therefore, EPA concludes with reasonable certainty that residues of sulfosate in drinking water do not contribute significantly to the aggregate acute human health risk at the present time considering the present uses and uses proposed in this action.

2. *Chronic risk*. Using the exposure assumptions TMRCs described above, EPA has concluded that aggregate exposure to sulfosate from food will utilize 7.6% of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is children 1 to 6 years old (discussed below). EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. For infants, children, and women, EPA determined that the 10x factor for increased susceptibility of infants and children (as required by FQPA) should be reduced to 3x. Therefore, for infants, children, and women, there is no concern for exposures below 33% of the RfD. Despite the potential for exposure to sulfosate in drinking water, EPA does not expect the aggregate exposure to exceed 33% of the RfD.

3. *Aggregate cancer risk for U.S. population*. Sulfosate was classified as a

''Group E'' carcinogen (no evidence for carcinogenicity in humans, see section B.4 of this document).

4. *Conclusions.* EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to sulfosate residues.

E. Aggregate Risks and Determination of Safety for Infants and Children

1. *Safety factor for infants and children— i. In general.* In assessing the potential for additional sensitivity of infants and children to residues of sulfosate, EPA 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 adverse effects on the developing organism resulting from maternal pesticide exposure gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

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

ii. *Developmental toxicity studies.* In a prenatal developmental toxicity study, sulfosate was administered by gavage to groups of pregnant Sprague-Dawley rats on gestation days 6–20 at dose levels of 0, 30, 100, or 333 mg/kg/day. The maternal NOEL was 100 mg/kg/day and LOEL was 333 mg/kg/day based on decreased body weight, food consumption, and increased clinical signs. The developmental NOEL was 100 mg/kg/day and LOEL was 333 mg/kg/day based on decreased fetal body weight.

In another prenatal developmental toxicity study, Sulfosate was administered by gavage to groups of New Zealand White rabbits on gestation days 6–18 at doses of 0, 10, 40, or 100 mg/kg/day. The maternal NOEL was 40 mg/kg/day and LOEL was 100 mg/kg/day based on abortions, deaths, decreased body weight and food consumption. The developmental NOEL was 40 mg/kg/day and LOEL was 100 mg/kg/day based on decreased number (7) of surviving does, and decrease in number of live fetuses/doe (5.4 vs 7.4 in controls).

iii. *Reproductive toxicity study.* Sulfosate was administered by diet to Sprague-Dawley rats at dose levels of 0, 150, 800, or 2,000 ppm for 2-generations. The parental systemic NOEL was 140 ppm (7.5 mg/kg/day) and the LOEL was 800 ppm (40 mg/kg/day) based on decreased body weight, decreased organ weights and decreased food consumption. The reproductive/offspring NOEL was 7.5 mg/kg/day (140 ppm) and LOEL was 40 mg/kg/day (800 ppm) based on decreased pup body weight during lactation.

iv. *Pre- and post-natal sensitivity.* The data provided no indication of increased susceptibility in rats or rabbits from in utero and/or post natal exposure to sulfosate. In the prenatal developmental toxicity study in rats, evidence of developmental toxicity was seen only in the presence of maternal toxicity. In the developmental toxicity study in rabbits, developmental toxicity was seen in the presence of maternal toxicity at the highest dose level. In the 2-generation reproduction study in rats, effects in the offspring were observed only at or above treatment levels which results in evidence of parental toxicity. It should be noted that a developmental neurotoxicity study is required.

v. *Developmental neurotoxicity.* A developmental neurotoxicity study is not available. One is required due to neurotoxicity observed in the rat, dog and mouse. Sulfosate is a neurotoxic chemical, which produces clinical findings such as salivation, tremors, emesis, and decreased activity in dogs and/or rats. Salivation was the most consistent sign, and in dogs may have served as a precursor to more severe symptoms. In one study, salivation stopped upon withdrawal of sulfosate and recurred upon reintroduction of treatment. Dogs appear to be the most sensitive species for these effects, with high intra-individual variability in sensitivity. Acute neurotoxicity effects observed after a single dose of 300 mg/kg in the rat included ptosis, decreased activity, decreased splay reflex, upward curvature of spine, shaking, sides pinched in, signs of urinary incontinence, irregular breathing, hunched posture, abnormal or staggering gait, increased time to tail flick, decreased landing foot splay, decreased forelimb grip strength, decreased hindlimb grip strength, decreased motor activity. There was also death at this dose. In the subchronic rat neurotoxicity study, the decreased forelimb grip strength observed at 153 mg/kg/day, in females only, may also have been due to treatment. Hydrocephalus or dilated ventricles were observed in at least one animal at the HDT (50 mg/kg/day) in adult dogs in all the dog studies, following both 90-days (gavage or capsule) and one year of dosing. This finding was never seen in controls or low dose groups. Hydrocephaly and/or dilated ventricles in dogs of this age may have been due to inherent asymptomatic incidences in the beagle (*Vullo et al., 1997*), but it was noted that these animals were not supplied by the same breeding colony, and the incidences were only observed at the high dose levels across several studies. Therefore, these findings can not be dismissed. Neuropathology was observed in the 21-day rat dermal study (sciatic nerve degeneration) at 1000 mg/kg, and the 2-year chronic mouse study (degeneration of the sciatic nerve, lumbar spinal root, and lumbar spinal white matter in males) at 991 mg/kg. Although these findings were previously discounted due to lack of supporting neuropathology data in the acute and subchronic neurotoxicity studies in rats, the overall neurotoxicity profile of the chemical indicated that the neuropathology could be a treatment-related effect of concern.

v. *Conclusion.* EPA concludes that the 10x factor for increased susceptibility of infants and children (as required by FQPA) should be reduced to 3x. The Agency determined that the data indicate that there is no increased susceptibility to young rats or rabbits following *in utero* exposure in prenatal studies or in the postnatal study in rats, and the guideline requirements for the toxicology data base are completed. Additionally, the exposure assessments for sulfosate do not indicate a concern for potential risk to infants and children since: (1) The dietary exposure assessments are unrefined (assuming that all commodities contain tolerance level residues) resulting in an over estimate of dietary exposure; (2) data from modeling are used for the ground and surface source drinking water exposure assessments, resulting in estimates considered to be reasonable upper-bound concentrations; and (3) there are currently no registered residential uses for sulfosate.

However, the FQPA safety factor was reduced to 3x instead of being removed because of the concern for the overall neurotoxicity exhibited in long-term studies in adult animals (mice, rats, and dogs) and the Agency's determination based on these findings that additional data are needed. In mice, sulfosate induced degeneration of the sciatic nerve, lumbar spinal root and lumbar spinal white matter was reported. In rats, degeneration of the sciatic nerve was seen following dermal applications. In dogs, hydrocephalus and/or dilated ventricles were observed following subchronic and chronic exposures. In addition, clinical signs indicative of neurotoxicity such as salivation, tremors, emesis, decreased activity was seen in rats and dogs. Based on these factors, the Agency determined that a developmental neurotoxicity study in rats is required to characterize the observed neuropathology in the subchronic and chronic studies.

2. *Acute risk.* Since there are no residential uses for sulfosate, the acute aggregate exposure only includes food and water. For infants and children, 7.3–9.4% of the acute RfD is occupied by dietary (food) exposure. The estimated average concentrations of sulfosate in surface and ground water are less than EPA's levels of concern for sulfosate in drinking water as a contribution to acute aggregate dietary exposure. The above calculations include the FQPA safety factor of 3x. Therefore, EPA concludes with reasonable certainty that residues of sulfosate in drinking water do not contribute significantly to the aggregate acute human health risk at the present time considering the present uses and uses proposed in this action. EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate acute exposure to sulfosate residues.

3. *Chronic risk.* Using the conservative exposure assumptions described above, EPA has concluded that aggregate exposure to sulfosate from food will utilize 11.9–20.3% of the RfD for infants and children. EPA has no concern for exposures below 33% 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 health of infants and children. Despite the potential for exposure to sulfosate in drinking water, EPA does not expect the aggregate dietary exposure to exceed 33% of the RfD. EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to sulfosate residues.

III. Other Considerations

A. Metabolism In Plants and Animals

The nature of the residues in plants and animals is understood. EPA has determined that the tolerance expression for sulfosate must include both of the parent ions.

B. Analytical Enforcement Methodology

1. *Plants.* Analytical methods are available for enforcement. There is currently a PAM II enforcement method for the *N*-(phosphonomethyl)glycine anion (PMG) in crops. For TMS, the registrant has proposed gas chromatography (GC) Method RR 93–105B as the analytical enforcement method. A successful petition method validation (PMV) of this analytical enforcement method for the TMS moiety in plants has been completed by the EPA laboratory. EPA concludes that Method RR 93-105B is adequate for enforcement of the permanent tolerances.

2. *Animals.* Analytical methods are available for enforcement. For PMG, the registrant has proposed GC Method RR 93–104B as the analytical enforcement method. For TMS, the registrant has proposed GC Method RR 93–100B as the analytical enforcement method. Successful PMV of these analytical enforcement methods for the PMG and TMS moieties in meat, milk and eggs have been completed by the EPA laboratory. EPA concludes that Method RR 93–104B and Method RR 93–100B are adequate for enforcement of the permanent tolerances.

C. Magnitude of Residues

The crop field trial data are adequate to support these tolerances.

D. International Residue Limits

There are no Codex, Canadian or Mexican tolerances or maximum residue limits for residues of sulfosate in the subject crops. Therefore, a compatibility issue is not relevant to the proposed tolerances.

E. Rotational Crop Restrictions.

EPA has previously reviewed two confined rotational crop studies for sulfosate and concluded that rotational crop restrictions were not required .

IV. Conclusion

Therefore, the tolerance is established for residues of sulfosate in cattle, goats, horses, hogs and sheep at 0.10 ppm in fat, at 1.00 ppm in meat by-products, and at 0.20 ppm in meat; in poultry at 0.05 ppm in fat, meat-by-products (except liver), and meat, and at 0.10 ppm in liver; in eggs at 0.02 ppm; in milk at 0.20 ppm; in corn at 0.30 ppm (of which no more than 0.20 ppm is TMS) in stover (field and pop), at 0.20 ppm (of which no more than 0.10 ppm is TMS) in grain (field and pop), at 0.10 ppm in forage (field); in soybeans at 2.00 ppm (of which no more than 1.0 ppm is TMS) in forage, at 5.00 ppm (of which no more than 2.0 ppm is TMS) in hay, and at 3.00 (of which no more than 1.0 ppm is TMS) ppm in seed; and in aspirated grain fractions at 210 ppm (of which no more than 60 ppm is TMS). In addition, the existing tolerances for prunes at 0.20 ppm, raisins at 0.20 ppm, and soybean hulls at 7.0 ppm are moved from 40 CFR 185.5375 to 40 CFR 180.489.

V. Objections and Hearing Requests

The new FFDCA section 408(g) provides essentially the same process for persons to ''object'' to a tolerance regulation issued by EPA under new section 408(e) and (l)(6) as was provided in the old section 408 and in section 409. However, the period for filing objections is 60 days, rather than 30 days. EPA currently has procedural regulations which govern the submission of objections and hearing requests. These regulations will require some modification to reflect the new law. However, until those modifications can be made, EPA will continue to use those procedural regulations with appropriate adjustments to reflect the new law.

Any person may, by November 10, 1998, file written objections to any aspect of this regulation and may also request a hearing on those objections. Objections and hearing requests must be filed with the Hearing Clerk, at the address given above (40 CFR 178.20). A copy of the objections and/or hearing requests filed with the Hearing Clerk should be submitted to the OPP docket for this rulemaking. The objections submitted must specify the provisions of the regulation deemed objectionable and the grounds for the objections (40 CFR 178.25). Each objection must be accompanied by the fee prescribed by 40 CFR 180.33(i). If a hearing is requested, the objections must include a statement of the factual issues on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the requestor (40 CFR 178.27). A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established, resolve one or more of such issues in favor of

the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32). Information submitted in connection with an objection or hearing request may be claimed confidential by marking any part or all of that information as Confidential Business Information (CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the information 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.

VI. Public Record and Electronic Submissions

EPA has established a record for this rulemaking under docket control number OPP–300709 (including any comments and data submitted electronically). 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 public record is located in Room 119 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Electronic comments may 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.

The official record for this rulemaking, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer any copies of objections and hearing requests received electronically into printed, paper form as they are received and will place the paper copies in the official rulemaking record which will also include all comments submitted directly in writing. The official rulemaking record is the paper record maintained at the Virginia address in ''ADDRESSES'' at the beginning of this document.

VII. Regulatory Assessment Requirements

A. Certain Acts and Other Executive Orders

This final rule establishes tolerances under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled Regulatory Planning and Review (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104–4). Nor does it require any prior consultation as specified by Executive Order 12875, entitled Enhancing the Intergovernmental Partnership (58 FR 58093, October 28, 1993), or special considerations as required by Executive Order 12898, entitled Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations (59 FR 7629, February 16, 1994), or require OMB review in accordance with Executive Order 13045, entitled Protection of Children from Environmental Health Risks and Safety Risks (62 FR 19885, April 23, 1997).

B. Executive Order 12875

Under Executive Order 12875, entitled Enhancing Intergovernmental Partnerships (58 FR 58093, October 28, 1993), EPA may not issue a regulation that is not required by statute and that creates a mandate upon a State, local or tribal government, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by those governments. If the mandate is unfunded, EPA must provide to the Office of Management and Budget (OMB) a description of the extent of EPA's prior consultation with representatives of affected State, local and tribal governments, the nature of their concerns, copies of any written communications from the governments, and a statement supporting the need to issue the regulation. In addition, Executive Order 12875 requires EPA to develop an effective process permitting elected officials and other representatives of State, local and tribal governments ''to provide meaningful and timely input in the development of regulatory proposals containing significant unfunded mandates.''

Today's rule does not create an unfunded federal mandate on State, local or tribal governments. The rule does not impose any enforceable duties on these entities. Accordingly, the requirements of section 1(a) of Executive Order 12875 do not apply to this rule.

C. Executive Order 13084

Under Executive Order 13084, entitled Consultation and Coordination with Indian Tribal Governments (63 FR 27655, May 19,1998), EPA may not issue a regulation that is not required by statute, that significantly or uniquely affects the communities of Indian tribal governments, and that imposes substantial direct compliance costs on those communities, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by the tribal governments. If the mandate is unfunded, EPA must provide OMB, in a separately identified section of the preamble to the rule, a description of the extent of EPA's prior consultation with representatives of affected tribal governments, a summary of the nature of their concerns, and a statement supporting the need to issue the regulation. In addition, Executive Order 13084 requires EPA to develop an effective process permitting elected and other representatives of Indian tribal governments ''to provide meaningful and timely input in the development of regulatory policies on matters that significantly or uniquely affect their communities.''

Today's rule does not significantly or uniquely affect the communities of Indian tribal governments. This action does not involve or impose any requirements that affect Indian Tribes. Accordingly, the requirements of section 3(b) of Executive Order 13084 do not apply to this rule.

In addition, since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerances in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. Nevertheless, the Agency has previously assessed whether establishing tolerances, exemptions from tolerances, raising tolerance levels or expanding exemptions might adversely impact small entities and concluded, as a generic matter, that there is no adverse economic impact. The factual basis for the Agency's generic certification for tolerance actions published on May 4, 1981 (46 FR 24950) and was provided to the Chief Counsel for Advocacy of the Small Business Administration.

VIII. Submission to Congress and the General Accounting Office

The Congressional Review Act, 5 U.S.C. 801 *et seq*., as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of the rule in the **Federal Register**. This is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects

40 CFR Part 180

Environmental protection, Administrative practice and procedure, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.

40 CFR Part 185

Environmental protection, Food additives, Pesticides and pests.

Dated: August 31, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180 — [AMENDED]

1. The authority citation for part 180 continues to read as follows:

Authority: 21 U.S.C. 346a and 371.

2. Section 180.489 is revised to read as follows:

§180.489 Sulfosate (Sulfonium, trimethyl-salt with N-(phosphonomethyl)glycine (1:1)); tolerances for residues.

(a) *General* . Tolerances are established for residues of the herbicide sulfosate (sulfonium, trimethyl-salt with *N*-(phosphonomethyl)glycine (1:1)) in or on the following raw and processed agricultural commodities:

Commodity	Parts per million
Almond, hulls (of which no more than 0.30 ppm is trimethylsulfonium (TMS)).	1.00
Aspirated grain fractions (of which no more than 60 ppm is TMS).	210.00
Bananas (imported only)a.	0.05
Cattle, fat	0.10
Cattle, mbyp	1.00
Cattle, meat	0.20
Citrus fruit group	0.05
Corn, field, forage	0.10
Corn, field and pop, grain (of which no more than 0.10 ppm is TMS).	0.20
Corn, field and pop, stover (of which no more than 0.20 ppm is TMS).	0.30
Eggs	0.02
Goats, fat	0.10
Goats, mbyp	1.00
Goats, meat	0.20
Grape	0.10
Hogs, fat	0.10
Hogs, mbyp	1.00
Hogs, meat	0.20
Horses, fat	0.10
Horses, mbyp	1.00
Horses, meat	0.20
Milk	0.20
Poultry, fat	0.05
Poultry, liver	0.05
Poultry, mbyp (except liver).	0.10
Poultry, meat	0.05
Prune (of which no more than 0.05 ppm is TMS).	0.20
Raisin (of which no more than 0.05 ppm is TMS).	0.20
Sheep, fat	0.10
Sheep, mbyp	1.0
Sheep, meat	0.20
Soybean, forage (of which no more than 1 ppm is TMS).	2.0
Soybean, hay (of which no more than 2 ppm is TMS).	5.0
Soybean, hulls (of which no more than 2 ppm is TMS).	7.0
Soybean, seed (of which no more than 1 ppm is TMS).	3.0
Stone fruit group	0.05
Tree nut group	0.05

aThere are no U.S. registrations as of the date of publication of the tolerance in the FEDERAL REGISTER.

(b) *Section 18 emergency exemptions.* [Reserved]

(c) *Tolerances with regional registrations.* [Reserved]

(d) *Indirect or inadvertent residues.* [Reserved]

PART 185 — [AMENDED]

1. The authority citation for part 185 continues to read as follows:

Authority: 21 U.S.C. 346a and 348.

§185.5375 [Removed]

2. By removing § 185.5375 Sulfonium, trimethyl-salt with *N*-(phosphonomethyl)glycine (1:1).

[FR Doc. 98–24468 Filed 9–10–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300708; FRL 6026–5]

RIN 2070–AB78

Esfenvalerate; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for residues of esfenvalerate, ((*S*)-cyano-(3-phenoxyphenyl)methyl (*S*)-4-chloro-alpha-(1-methylethyl) benzeneacetate in or on the raw agricultural commodities mustard greens at 5.0 parts per million (ppm), kiwifruit at 0.5 ppm, globe artichoke at 1.0 ppm, and kohlrabi at 2.0 ppm. Esfenvalerate is the *S,S*-isomer of fenvalerate which consists of a racemic mixture of four isomers (*S,S;R,S;S,R*; and *RR*). Technical grade esfenvalerate, Asana, the only fenvalerate formulation sold in the United States for agricultural use at this time, is enriched in the insecticidally active *S,S*-isomer (84%). Tolerance expressions for esfenvalerate are based on the sum of all isomers. The Interregional Research Project Number 4 (IR-4) requested this tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170).