Comments received after the time indicated under **DATES** or at locations other than the Harrisburg Field Office will not necessarily be considered in the final rulemaking or included in the Administrative Record.

IV. Procedural Determinations

Executive Order 12866

This proposed rule is exempted from review by the Office of Management and Budget (OMB) under Executive Order 12866 (Regulatory Planning and Review).

Executive Order 12988

The Department of the Interior has conducted the reviews required by section 3 Executive Order 12988 (Civil Justice Reform) and has determined that, to the extent allowed by law, this rule meets the applicable standards of subsections (a) and (b) of that section. However, these standards are not applicable to the actual language of State and Tribal abandoned mine land reclamation plans and revisions thereof since each such plan is drafted and promulgated by a specific State or Tribe, not by OSM. Decisions on proposed abandoned mine land reclamation plans and revisions thereof submitted by a State or Tribe are based on a determination of whether the submittal meets the requirements of Title IV of SMCRA (30 U.S.C. 1231–1243) and 30 CFR Parts 884 and 888.

National Environmental Policy Act

No environmental impact statement is required for this rule since agency decisions on proposed State and Tribal abandoned mine land reclamation plans and revisions thereof are categorically excluded from compliance with the National Environmental Policy Act (42 U.S.C. 4332) by the Manual of the Department of the Interior (516 DM 6, appendix 8, paragraph 8.4B(29)).

Paperwork Reduction Act

This rule does not contain information collection requirements that require approval by OMB under the Paperwork Reduction Act (44 U.S.C. 3507 *et seq.*).

Regulatory Flexibility Act

The Department of the Interior has determined that this rule will not have a significant economic impact on a substantial number of small entities under the Regulatory Flexibility Act (5 U.S.C. 601 *et seq.*). The State submittal which is the subject of this rule is based upon corresponding Federal regulations for which an economic analysis was prepared and certification made that such regulations would not have a significant economic effect upon a substantial number of small entities. Accordingly, this rule will ensure that existing requirements previously promulgated by OSM will be implemented by the State. In making the determination as to whether this rule would have a significant economic impact, the Department relied upon the data and assumptions in the analyses for the corresponding Federal regulations.

Unfunded Mandates

This rule will not impose a cost of $100 million or more in any given year on any governmental entity or the private sector.

List of Subjects in 30 CFR Part 938

Intergovernmental relations, Surface mining, Underground mining.

Dated: July 21, 1998.

Allen D. Klein,

Regional Director, Appalachian Regional Coordinating Center.

[FR Doc. 98–20163 Filed 7–27–98; 8:45 am]

BILLING CODE 4310–05–M

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300693; FRL–6020–6]

RIN 2070–AC18

Spinosad; Time-Limited Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA proposes to establish a time-limited tolerance for residues of spinosad in or on coffee at 0.02 parts per million (ppm). This action is being initiated by EPA under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170). The United States Department of Agriculture/Agricultural Research Service (USDA/ARS) has requested that EPA establish a time-limited tolerance on coffee in order for USDA/ARS to conduct efficacy testing of spinosad to control the Mediterranean Fruit Fly. This testing will be conducted on 80 acres in Hawaii under an Experimental Use Permit (EUP).

DATES: Comments, identified by the docket conrol number [OPP–300693], must be received by EPA on or before August 11, 1998.

ADDRESSES: By mail, submit written comments 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, deliver comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under Unit VI of this document. No Confidential Business Information (CBI) should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential will be included in the public docket by EPA without prior notice. The public docket is available for public inspection in Rm. 119 at the Virginia address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Susan Lewis, 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) 305–7448, e-mail: lewis.susan@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of April 15, 1998 (63 FR 18329)(FRL–5785–7), EPA established permanent tolerances by removing the time limitation for the tolerance for residues of the insecticide spinosad in or on cottonseed at 0.02 ppm and by establishing tolerances in or on almonds at 0.02 ppm; almond hulls at 2.0 ppm; apples at 0.2 ppm; apple pomace, wet at 0.5 ppm; citrus fruits group at 0.3 ppm; dried citrus pulp at 0.5 ppm; citrus oil at 3.0 ppm; cotton gin byproducts at 1.5 ppm; fruiting vegetables (except cucurbits) group at 0.4 ppm; leafy vegetables (except Brassica vegetables) group at 8.0 ppm; Brassica (cole), leafy vegetables, head and stem subgroup at 2.0 ppm; Brassica (cole), leafy vegetables, greens subgroup at 15.0 ppm; fat of cattle, goats, hogs, horses, and sheep at 0.7 ppm; meat of cattle, goats, hogs, horses, and sheep at 0.04 ppm; meat byproducts of cattle, goats, hogs, horses, and sheep at 0.2

ppm; milk fat at 0.5 ppm; and whole milk at 0.04 ppm.

The USDA has requested that EPA establish a time-limited tolerance for residues of spinosad in or on coffee. This tolerance will expire on August 28, 2000. USDA has requested this tolerance in order to conduct efficacy testing of spinosad for control of the Mediterranean Fruit Fly. This testing will be conducted on 80 acres in Hawaii under an Experimental Use Permit (EUP).

The Agency has concluded that a tolerance of 0.02 ppm (which is the Limit of Quantitation (LOQ) for the analytical method) is adequate for coffee. This is based on a very low application rate and the fact that the hull of the coffee bean is removed. No residues are expected to be found on the coffee beans.

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 percent 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 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 hundredfold MOE is based on the same rationale as the hundredfold 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.

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 the existing uses of spinosad. EPA had sufficient data to assess the hazards of spinosad and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerances for residues of spinosad for those uses. EPA's assessment of the dietary exposures and risks associated with establishing the existing tolerances 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 spinosad are discussed below.

1. Acute toxicity studies with technical spinosad (88% – 90.4%): Oral LD_{50} in the rat is > 5,000 milligram/kilogram (mg/kg) for males and females - Toxicity Category IV; dermal LD_{50} in the rat is > 2,800 mg/kg for males and females - Toxicity Category III; inhalation LC_{50} in the rat is > 5.18 mg/L - Toxicity Category IV; primary eye irritation in the rabbit (slight conjunctival irritation) - Toxicity Category IV; primary dermal irritation in the rabbit (no erythema and edema) - Toxicity Category IV. Spinosad is not a sensitizer.

2. Acute toxicity studies with the end-use (44% formulation) product for spinosad: Oral LD_{50} in the rat is > 5,000 mg/kg for males and females - Toxicity Category IV; dermal LD_{50} in the rat is > 2,800 mg/kg for males and females - Toxicity Category III; inhalation LC_{50} in the rat is > 5 mg/L - Toxicity Category IV; primary eye irritation in the rabbit (slight conjunctival irritation) - Toxicity Category IV; primary dermal irritation in the rabbit (slight transient erythema and edema) - Toxicity Category IV; not a sensitizer.

3. In a subchronic feeding study in rats, the no-observed adverse effect level (NOAEL) was 33.9 and 38.8 mg/kg/day for males and females, respectively. The lowest observed effect level (LOEL) was 68.5 and 78.1 mg/kg/day for males and females, respectively based on decreased body weight gain, anemia, and vacuolation in multiple organs (kidney, liver, heart, spleen, adrenals, and thyroid).

4. In a subchronic feeding study in mice, the no observed effect level (NOEL) was 7.5 mg/kg/day and the LOEL was 22.5 mg/kg/day based on cytoplasmic vacuolation in multiple organs (kidney, liver, heart, stomach, lymphoid organs, and ovary).

5. In a subchronic feeding study in dogs, the NOEL was 4.89 and 5.38 mg/kg/day for males and females, respectively. The LOEL was 9.73 mg/kg/day and 10.5 mg/kg/day based on decreased mean body weights and food consumption, and anemia.

6. In a 21–day dermal study in rats, the NOEL for systemic effects was > 1,000 mg/kg/day (limit dose). No systemic toxicity was observed at any dose tested.

7. In a chronic feeding study in dogs, the NOEL was 2.68 mg/kg/day. The LOEL was 8.22 mg/kg/day based on increased liver enzymes (ALT, AST), triglycerides; vacuolated cells (parathyroid), and arteritis.

8. In an carcinogenicity study in mice, the NOEL was 11.4 mg/kg/day. The LOEL was 50.9 mg/kg/day based on decreased body weight gains, increased mortality, hematologic effects, increased thickening of the gastric mucosa, and histologic changes in the stomach of males.

9. In a chronic feeding/carcinogenicity/neurotoxicity study in rats, the NOEL (systemic) was 9.5 and 12.0 mg/kg/day for males and females, respectively. The LOEL (systemic) was 24.1 and 30.3 mg/kg/day for males and females, respectively based on vacuolation of epithelial follicular cells of the thyroid. The neurological NOEL was 46 and 57 mg/kg/day for males and females, respectively. The neurological LOEL was not determined.

10. In a developmental study in rabbits, the maternal NOEL was ≧ 50 mg/kg/day. The maternal LOEL was not established. The developmental NOEL was ≧ 50 mg/kg/day. The developmental LOEL was not established.

11. In a developmental study in rats, the maternal NOEL was > 200 mg/kg/day. The maternal LOEL was not established. The developmental NOEL was > 200 mg/kg/day. The developmental LOEL was not established.

12. In a two-generation reproduction toxicity study in rats, the systemic NOEL was 10 mg/kg/day. The systemic LOEL was 100 mg/kg/day based on increased organ weights (heart, liver, kidney, spleen, thyroid), histopath lesions in the lungs and mesenteric lymph nodes, stomach (F), and prostate. The reproductive NOEL was 10 mg/kg/day. The reproductive LOEL was 100 mg/kg/day based on decreased litter size, decreased pup survival, decreased body weight, increased incidence of dystocia and/or vaginal bleeding post-partum with associated increased mortality of dams.

13. Studies on gene mutation and other genotoxic effects: In a Gene Mutation Assay (mouse forward mutation) there was no forward mutation induction in mouse lymphoma L5178Y Tk +/- cells at concentrations of 0, 1, 5, 10, 15, 20, or 25 µg/ml without metabolic activation or at concentrations of 15 through 50 µg/ml with metabolic activation. In a Structural Chromosomal Aberration Assay *In vitro* there was no increase in the number of CHO (chinese hamster ovary) cells with chromosomal aberrations at concentrations from 20 to 35 µg/ml (without activation) or concentrations from 100 to 500 µg/ml (with activation). In a Micronucleus Test in mice, there was no increase in the frequencey of micronuclei in bone marrow cells from mice treated at concentrations from 500 to 2,000 µg/ml for 2 days. In Other Genotoxicity Assays, unscheduled DNA synthesis was not induced in adult rat hepatocytes *in vitro* at concentrations of 0.01 to 5 µg/ml tested.

14. The results of three metabolism studies are as follows: i. Approximately 95% of technical spinosad was eliminated by 24 hours mainly in the urine (34%), bile (36%), and tissues and carcass (21%). Metabolites include the glutathione conjugates of the unchanged form as well as N- and O-demethylated forms of XDE-105 (Factor D).

ii. At 100 mg/kg/dose, the radiolabeled XDE-105 (Factor D) was primarily excreted in the feces (68%) after 24–hours. The absorption, distribution, and elimination of 14C-XDE-105 (Factor A) demonstrated no appreciable differences based on dose or repeated dosing.

iii. At high (100 mg/kg) doses, there are no major differences in the bioavailability, routes or rates of excretion or metabolism of 14C-XDE-105 (Factor A) following oral administration.

15. In an acute neurotoxicity study, groups of Fischer 334 rats (10/sex/dose) received a single oral (gavage) administration of spinosad (87.9%) at dose levels of 0, 200, 630, or 2,000 mg/kg. There were no effects on neurobehavioral endpoints or histopathology of the nervous system. For neurotoxicity, the NOEL was ≧ 2,000 mg/kg/day (HDT). A LOEL was not established.

16. In a subchronic neurotoxicity study, groups of Fischer 344 rats (10/sex/dose) were administered diets containing spinosad at levels of 0, 0.003, 0.006, 0.012, or 0.06% (0, 2.2, 4.3, 8.6, or 42.7 mg/kg/day for males and 2.6, 5.2, 10.4, or 52.1 mg/kg/day for females, respectively). There were no effects on neurobehavior endpoints or histopathology of the nervous system. For neurotoxicity, the NOEL was ≧ 42.7 and ≧ 52.1 mg/kg/day in males and females, respectively (HDT).

17. In the 2–year chronic neurotoxicity study, groups of Fischer 344 rats (65/sex/dose) received diets containing spinosad at dose levels of 0, 0.005, 0.02, 0.05, or 0.1% (0, 2.4, 9.5, 24.1, or 49.4 mg/kg/day for males and 0, 3.0, 12.0, 30.3, or 62.2 mg/kg/day for females, respectively). Neurobehavioral testing performed at 3, 6, 9, and 12 months of study was negative, and histopathological evaluation of perfused tissues at study termination did not identify pathology of the central or peripheral nervous system. There was no evidence of neurotoxicity. For neuropathology, the NOEL was 0.1% (≧ 46 mg/kg/day for males and 57 mg/kg/day for females (HDT).

B. Toxicological Endpoints

1. *Acute toxicity*. EPA did not select a dose and endpoint for an acute dietary risk assessment due to the lack of toxicological effects attributable to a single exposure (dose) in studies available in the data base including oral developmental toxicity studies in rats and rabbits. In the acute neurotoxicity study the NOEL was ≧ 2,000 mg/kg/day.

2. *Short - (1 day to 7 days), intermediate- (1 week to several months), and chronic - term occupational and residential dermal and inhalation toxicity*. EPA did not select a dose or endpoint for short-, intermediate and long-term dermal risk assessments because (i) lack of appropriate endpoints; (ii) the combination of molecular structure and size as well as the lack of dermal or systemic toxicity at 2,000 mg/kg/day in a 21-day dermal toxicity study in rats which indicates the lack of dermal absorption; and (iii) the lack of long-term exposure based on the current use pattern. Therefore, a dermal risk assessment is not required. EPA also determined that based on the current use pattern and exposure scenario, and inhalation risk assessment is not required.

3. *Chronic toxicity*. EPA has established the RfD for spinosad at 0.027 mg/kg/day. This RfD is based on a chronic toxicity study in dogs using a NOEL of 2.68 mg/kg/day. The LOEL was 8.46 mg/kg/day based on vacuolation in glandular cells (parathyroid) and lymphatic tissues, arteritis and increases in serum enzymes such as alanine aminotransferase, and aspartate aminotransferase, and triglyceride levels in dogs fed spinosad in the diet at dose levels of 1.44, 2.68, or 8.46 mg/kg/day for 52 weeks. A hundredfold uncertainty factor (UF) was applied to the NOEL of 2.68 mg/kg/day to account for inter- and intra-species variation.

EPA determined that the 10X factor to account for enhanced sensitivity of infants and children (as required by FQPA) should be removed. Thus, an uncertainty factor of 100 is adequate and the RfD remains at 0.027 mg/kg/day. The FQPA factor is removed because: (i) the data provided no indication of increased susceptibility of rats or rabbits to *in utero* and/or post-natal exposure to spinosad. In the prenatal developmental toxicity studies in rats and rabbits and the two-generation reproduction study in rats, effects in the offspring were observed only at or below treatment levels which resulted in evidence of parental toxicity. (ii) No neurotoxic signs have been observed in any of the standard required studies conducted. (iii) The toxicology data base is complete and there are no data gaps.

4. *Carcinogenicity*. There is no evidence of carcinogenicity in studies in either the mouse or rat.

C. Exposures and Risks

1. *From food and feed uses.* Tolerances have been established (40 CFR 180.495) for the residues of spinosad in or on almonds at 0.02 ppm; almond hulls at 2.0 ppm; apples at 0.2 ppm; apple pomace, wet at 0.5 ppm; citrus fruits group at 0.3 ppm; dried citrus pulp at 0.5 ppm; citrus oil at 3.0 ppm; cottonseed at 0.02 ppm; cotton gin byproducts at 1.5 ppm; fruiting vegetables (except cucurbits) group at 0.4 ppm; leafy vegetables (except Brassica vegetables) group at 8.0 ppm; Brassica (cole), leafy vegetables, head and stem subgroup at 2.0 ppm; Brassica (cole), leafy vegetables, greens subgroup at 15.0 ppm; fat of cattle, goats, hogs, horses, and sheep at 0.7 ppm; meat of cattle, goats, hogs, horses, and sheep at 0.04 ppm; meat byproducts of cattle, goats, hogs, horses, and sheep at 0.2 ppm; milk fat at 0.5 ppm; and whole milk at 0.04 ppm.

For the existing uses referred to above, risk assessments were conducted by EPA to assess dietary exposures and risks from spinosad 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 1 day or single exposure. No acute toxicological endpoints were identified for spinosad due to the lack of toxicological effects attributable to a single exposure (dose). Therefore, the

Agency concludes that there is a reasonable certainty of no harm from acute dietary exposure.

ii. *Chronic exposure and risk.* The RfD used for the chronic dietary analysis is 0.027 mg/kg/day. In conducting this chronic dietary risk assessment, EPA made very conservative assumptions: 100% of citrus, almonds, apples, fruiting (except cucurbit) vegetables, Brassica leafy vegetables, leafy vegetables, cottonseed, and ruminant commodities having spinosad tolerances will contain spinosad residues and those residues will be at the level of the established tolerance. This results in an overestimate of human dietary exposure. This chronic dietary risk assessment used 10 ppm tolerances for the leafy vegetables (except Brassica vegetables) crop group and for the Brassica leafy vegetables head and stem subgroup from section 18 tolerances that were established last year. For the section 3 registrations on these groups, EPA has recommended tolerances of 8 ppm (leafy vegetables) and 2 ppm (Brassica head and stem leafy vegetables). The use pattern for these section 18 registrations is identical to the section 3 registrations proposed in this risk assessment, but due to an incomplete data base at the time the section 18 registrations were reviewed, the tolerances were set high which resulted in a conservative risk assessment. With this action, these section 18 tolerances are replaced by the new section 3 tolerances. Thus, in making a safety determination for this tolerance, EPA is taking into account this conservative exposure assessment.

The existing spinosad tolerances resulted in a Theoretical Maximum Residue Contribution (TMRC) that is equivalent to the following percentages of the RfD: U.S. population (24% of RfD); nursing infants (< 1 year old)(8% of RfD); non-nursing infants (< 1 year old) (24% of RfD); children (1–6 years old) (34% of RfD); children (7–12 years old) (29% of RfD); Northeast Region (25% of RfD); Western Region (27% of RfD); Non-Hispanic Blacks (27% of RfD); Non-Hispanic others (37% of RfD); females 13+ years, nursing (27% of RfD).

The Agency believes that the addition of a 0.02 ppm tolerance for spinosad on coffee will only change the percent of the RfD used for any of the categories listed above by less than 1%. This is based on the fact that the use will be limited to 80 acres in Hawaii for experimental purposes for period of time not to exceed 2 years.

2. *From drinking water.* The Agency has determined that spinosyns Factor A and Factor D are immobile in soil and will not leach into ground water. Based on structure/activity relationships, the Agency concluded that the spinosad metabolites/fermentation impurities (spinosyns Factor B, Factor B of D, Factor K, and other related Factors) were of no more toxicological concern than the two parent compounds (spinosyns Factor A and Factor D) and therefore, only these were considered in the drinking water assessment. EPA used the ''Interim Approach for Addressing Drinking Water Exposure in Tolerance Decision Making'' issued on 11/17/97. Thus, the PRZM/EXAMS Models were run to produce estimates of spinosad in surface water. The primary use of these models is to provide a screen for sorting out pesticides for which OPP has a high degree of confidence that the true levels of the pesticide in drinking water will be less than the human health drinking water levels of concern (DWLOCs). A human health DWLOC is the concentration of a pesticide in drinking water which would result in acceptable aggregate risk, after having already factored in all food exposures and other non-occupational exposures for which OPP has reliable data. PRZM/EXAMS was used to conduct a Tier 2 surface water analysis. The Tier 2 estimated drinking water concentration (EEC) of spinosad from surface water sources is not likely to exceed 0.059 µg/l from use on apples, 0.092 µg/l from use on Brassica vegetables, 0.065 µg/l from use on cotton, and 0.075 µg/l from use on citrus.

i. *Acute exposure and risk.* Because no acute dietary endpoint was determined, the Agency concludes that there is a reasonable certainty of no harm from acute exposure from drinking water.

ii. *Chronic exposure and risk.* Based on the chronic dietary (food) exposure and using default body weights and water consumption figures, chronic drinking water levels of concern (DWLOC) were calculated. The chronic drinking water exposure and risk estimates are 0.019890 mg/kg/day (690 µg/l DWLOC) for the overall U.S. population; 0.01896 mg/kg/day (570 µg/l DWLOC) for females 13+ years, nursing; and 0.016865 mg/kg/day (170 µg/l DWLOC) for children age 1–6 years.

3. *From non-dietary exposure.* There are no current residential uses for spinosad. However, the proposed use of a 0.5% spinosad product on structural lumber may have residential uses. This product is injected into drilled holes and then sealed after treatment. Due to the lack of toxicity endpoints (hazard) and minimal contact with the active ingredient during and after application, exposure to residential occupants is not expected.

4. *Cumulative exposure to substances with common mechanism of toxicity.* Spinosad has not yet been grouped with any other insecticides into a class.

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

spinosad 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, spinosad does not appear to produce a toxic metabolite produced by other substances. For the purposes of these tolerance actions, therefore, EPA has not assumed that spinosad has a common mechanism of toxicity with other substances.

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

Chronic risk. The following information is based on the review of the existing uses of spinosad: Using the TMRC exposure assumptions described above, EPA has concluded that aggregate exposure to spinosad from food will utilize 24% of the RfD for the U.S. population. For the most highly exposed populations subgroup, children (1–6 years old), chronic dietary (food only) exposure occupies 34% of the RfD. This is a conservative risk estimate for reasons described above. 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. The chronic DWLOC for the infants and children subgroup is 170 parts per billion (ppb). The chronic modeling estimates (EECs) for spinosad residues in surface water are as high as 0.092 ppb from use on Brassica leafy vegetables. The maximum estimated concentrations of spinosad in surface water are less than EPA's levels of concern for spinosad in drinking water as a contribution to chronic aggregate exposure. Taking into account present uses and uses proposed in this risk assessment, EPA concludes with reasonable certainty that residues of spinosad 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. Therefore, the Agency concludes that there is a reasonable certainty that no harm will result from chronic aggregate exposure to spinosad residues from food and water.

No dermal or inhalation endpoints were identified. Due to the nature of the non-dietary use, EPA believes that the use of spinosad in treating structural lumber will not result in any exposure through the oral route. Therefore, the chronic aggregate risk is the sum of food and water.

Based on the above information, the Agency concludes that there is a reasonable certainty that no harm will result from chronic aggregate exposure to spinosad from food and water resulting from the addition of the time-limited experimental use on coffee as described above.

E. Aggregate Cancer Risk for U.S. Population

The RfD Committee determined that there is no evidence of carcinogenicity in studies in either the mouse or rat. Therefore, a carcinogenic risk assessment is not required.

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

1. *Safety factor for infants and children*— a. *In general.* In assessing the potential for additional sensitivity of infants and children to residues of spinosad, EPA considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development to one or both parents. 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 MOE and 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.

b. *Developmental toxicity studies.* i. In a prenatal developmental toxicity study, groups of pregnant Sprague-Dawley rats (30/group) received oral (gavage) administration of spinosad (88.6%) in aqueous 0.5% methycellulose at dose levels of 0, 10, 50, 200 mg/kg/day during gestation days 6 through 17. For maternal toxicity, the NOEL was ≧ 200 mg/kg/day (HDT); a LOEL was not established. Marginal maternal toxicity was reported at this dose level (decreased body weight gain). Based upon the results of a range-finding study, which showed maternal toxicity (body weight and food consumption decreases at 100 and 300 mg/kg/day), the dose level of 200 mg/kg/day in the main study was considered adequate. For developmental toxicity, the NOEL was > 200 mg/kg/day; a LOEL was not established. In the range-finding study, fetal body weight decrements occurred at 300 mg/kg/day.

ii. In a prenatal developmental toxicity study, groups of pregnant New Zealand White rabbits (20/group) received oral (gavage) administration of spinosad (88.6%) in 0.5% aqueous methyl cellulose at doses of 0, 2.5, 10, or 50 mg/kg/day during gestation days 7 through 19. For maternal toxicity, the NOEL was ≧ 50 mg/kg/day (HDT); a LOEL was not established. At this dose, slight body weight loss was observed in the first few days of dosing, but this finding was not supported by other signs. In the range-finding study, inanition was observed at doses of 100, 200, and 400 mg/kg/day, with significant decreases in body weight gain during dosing. All does at these dose levels were sacrificed prior to scheduled termination; no fetal data were available. No evidence of developmental toxicity was noted. For developmental toxicity, the NOEL was ≧ 50 mg/kg/day; a LOEL was not established. (No fetal effects were noted for fetuses of the range-finding study at doses up to 50 mg/kg/day).

c. *Reproductive toxicity study.* In a two-generation reproduction study, groups of Sprague-Dawley rats (30/sex/group) received diets containing spinosad (88%) at dose levels of 0, 0.005, 0.02, or 0.2% (3, 10, or 10 mg/kg/day, respectively) for two successive generations. For parental systemic toxicity, the NOEL was 0.02% (10 mg/kg/day) and the LOEL was 0.2% (100 mg/kg/day), based on increased heart, kidney, liver, spleen, and thyroid weights (both sexes), histopathology in the spleen and thyroid (both sexes), heart and kidney (males), and histopathologic lesions in the lungs and mesenteric lymph nodes (both sexes), stomach (females), and prostate. For offspring toxicity, the NOEL was 0.02% (10 mg/kg/day) and the LOEL was 0.2% (100 mg/kg/day) based on decreased litter size, survival (F2), and body weights. Reproductive effects at that

dose level included increased incidence of dystocia and/or vaginal bleeding after parturition with associated increase in mortality of dams.

d. *Neurotoxicity*. i. In an acute neurotoxicity study, groups of Fischer 344 rats (10/sex/dose) received a single oral (gavage) administration of spinosad (87.9%) at dose levels of 0, 200, 630, or 2,000 mg/kg. There were no effects on neurobehavioral endpoints or histopathology of the nervous system. For neurotoxicity, the NOEL was > 2,000 mg/kg (HDT); a LOEL was not established.

ii. In a subchronic neurotoxicity study, groups of Fisher 344 rats (10/sex/dose) were administered diets containing spinosad at levels of 0, 0.003, 0.006, 0.012, or 0.06% (0, 2.2, 4.3, 8.6, or 42.7 mg/kg/day for males and 2.6, 5.2, 10.4, or 52.1 mg/kg/day for females, respectively). There were no effects on neurobehavioral endpoints or histopathology of the nervous system. For neurotoxicity, the NOEL was ≧ 42.7 for males and ≧ 52.1 mg/kg/day for females (HDT).

iii. In the 2–year chronic toxicity study, groups of Fischer 344 rats (65/sex/dose) received diets containing spinosad at dose levels of 0, 0.005, 0.02, 0.05, or 0.1% (0, 2.4, 9.5, 24.1, or 49.4 mg/kg/day for males and 0, 3.0, 12.0, 30.3, or 62.2 mg/kg/day for females, respectively). Neurobehavioral testing performed at 3, 6, 9, and 12 months of study was negative, and histopathological evaluation of perfused tissues at study termination did not identify pathology of the central or peripheral nervous system. There was no evidence of neurotoxicity. For neuropathology, the NOEL was 0.1% (> 49.4 mg/kg/day for males and 62.8 mg/kg/day for females).

e. *Pre- and post-natal sensitivity*. There was no increased susceptibility to rats or rabbits following *in utero* and/or postnatal exposure to spinosad.

f. *Conclusion*. The data provided no indication of increased susceptibility of rats or rabbits to *in utero* and/or postnatal exposure to spinosad. In the prenatal developmental toxicity studies in rats and rabbits and the two-generation reproduction study in rats, effects in the offspring were observed only at or below treatment levels which resulted in evidence of parental toxicity. In addition, all neurotoxicity studies were negative for effects on the central or peripheral nervous system.

EPA determined that the 10X factor to account for enhanced sensitivity of infants and children (as required by FQPA) should be removed. The FQPA factor is removed because (i) the data provided no indication of increased susceptibility of rats or rabbits to *in utero* and/or post natal exposure to spinosad. In the prenatal developmental toxicity studies in rats and rabbits and the two-generation reproduction study in rats, effects in the offspring were observed only at or below treatment levels which resulted in evidence of parental toxicity. (ii) No neurotoxic signs have been observed in any of the standard required studies conducted. (iii) The toxicology data base is complete and there are no data gaps.

2. *Acute risk*. An acute risk assessment is not required because no acute toxicological endpoints were identified for spinosad.

3. *Chronic risk*. Using the conservative exposure assumptions described above, EPA has concluded that aggregate exposure to spinosad from food will utilize 34% of the RfD for children age 1–6 years old. 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. EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to spinosad residues.

Based on the above information, EPA concludes that there is a resonable certainty that no harm will result to infants and children from aggregate exposure to spinosad residues as a result of the use on coffee in an experimental use program in Hawaii.

G. Endocrine Disruption

EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect...'' The Agency is currently working with interested stakeholders, including other government agencies, public interest groups, industry and research scientists in developing a screening and testing program and a priority setting scheme to implement this program. Congress has allowed 3 years from the passage of FQPA (August 3, 1999) to implement this program. At that time, EPA may require further testing of this active ingredient and end use products for endocrine disrupter effects.

III. Other Considerations

A. Metabolism In Plants and Animals

EPA has reviewed the results of plant metabolism studies (apples, cabbage, cotton, tomatoes, turnips) and livestock metabolism studies (goat and hen). The metabolism of spinosad in plants and animals is adequately understood for the purposes of these tolerances. Based on structure/activity relationships, EPA concluded that the spinosad metabolites/fermentation impurities (spinosyns Factor B, Factor B or D, Factor K, and other related Factors) were of no more toxicological concern than the two parent compounds (spinosyns Factor A and Factor D).

EPA focused on the following data/information: the overall low toxicity of spinosad; the low levels of metabolites/fermentation impurities present; and that spinosad appears to photodegrade rapidly and become incorporated into the general carbon pool. EPA concluded that only 2 parent compounds (spinosyns Factor A and Factor D) need to be included in the tolerance expression and used for dietary risk assessment purposes.

B. Analytical Enforcement Methodology

Method GRM 94.02 (method for determination of spinosad residues in cottonseed and related commodities using HPLC/UV) underwent successful independent lab validation and EPA lab validation and has been submitted to FDA for inclusion in PAM II as Method I. Additional methods have been submitted for other crop matrices (leafy vegetables - GRM 95.17; citrus - GRM 96.09; tree nuts - GRM 96.14; fruiting vegetables - GRM 95.04; and cotton gin byproducts - GRM 94.02.S1). All of these methods are essentially similar to GRM 94.02 and have been submitted to FDA for inclusion in PAM II as letter methods. These methods are adequate for regulation of the tolerance expression.

Method RES 94094 (method for determination of spinosad residues in ruminant commodities using HPLC/UV) underwent successful independent lab validation and EPA lab validation and has been submitted to FDA for inclusion in PAM II as Method I. This method is adequate for regulation of the tolerance expression.

Method RES 95114 (method for determination of spinosad residues in ruminant commodities using immunoassay) underwent successful independent lab validation and EPA lab validation and has been submitted to FDA for inclusion in PAM II as Method I. This method is adequate for regulation of the tolerance expression.

C. International Residue Limits

No CODEX, Canadian, or Mexican MRLs have been established for residues of spinosad on any crops.

IV. Conclusion

A time-limited tolerance is being proposed for residues of spinosad in coffee at 0.02 ppm.

V. Public Comment Procedures

EPA invites interested persons to submit written comments, information, or data in response to this proposed rule. After consideration of comments, EPA will issue a final rule. Such rule will be subject to objections. Failure to file an objection within the appointed period will constitute waiver of the right to raise in future proceedings issues resolved in the final rule.

The period for comments on this proposed rule has been shortened to 14 days because the Agency believes that it is in the public interest to do so. The purpose of this temporary tolerance is to allow for efficacy testing to determine whether this reduced risk chemical will control the Mediterranean Fruit Fly. This quarantine insect is a serious economic pest which is threatening continental U.S. borders. The USDA/ARS needs to begin their experimental use program in Hawaii no later than August 15, 1998 and therefore, the Agency is allowing a 14 day public comment period to accommodate this need.

VI. Public Docket and Submission of Electronic Comments

The official record for this rulemaking, as well as the public version, has been established for this rulemaking under docket control number [OPP–300693] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official rulemaking record is located at the Virginia address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [OPP–300693]. Electronic comments on this proposed rule may be filed online at many Federal Depository Libraries.

VIII. Regulatory Assessment Requirements

This action proposes a time-limited tolerance under FFDCA section 408(e). 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). In addition, this proposed 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 special 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).

In addition, under the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 et seq.), the Agency 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.

List of Subjects in 40 CFR Part 180

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

Dated: July 23, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Therefore, it is proposed that 40 CFR part 180 be 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. In § 180.495, by revising paragraph (a) to read as follows:

§ 180.495 Spinosad; tolerances for residues.

(a) *General* . Tolerances are established for residues of the insecticide Spinosad. Factor A is 2-[(6-deoxy-2,3,4-tri-*O*-methyl-α-L-manno-pyranosyl)oxy]-13-[[5-(dimethylamino)-tetrahydro-6-methyl-2*H*-pyran-2-yl]oxy]-9-ethyl-2,3,3a,5a,5b,6,9,10,11,12,13,14,16a,6b-tetradecahydro-14-methyl-1*H*-as-Indaceno[3,2-d]oxacyclododecin-7,15-dione. Factor D is 2-[(6-deoxy-2,3,4-tri-*O*-methyl-α-L-manno-pyranosyl)oxy]-13-[[5-(dimethylamino)-tetrahydri-6-methyl-2*H*-pyran-2-yl]oxy]-9-ethyl-2,3,3a,5a,5b,6,9,10,11,12,13,14,16a,16b-tetradecahydro-4,14-dimethyl-1*H*-as-Indaceno[3,2-d]oxacyclododecin-7,15-dione.

Commodity	Parts per million	Expiration/Revocation Date
Almonds	0.02	None
Almond hulls	2.0	None
Apples	0.2	None
Apple pomace, wet	0.5	None
Brassica (cole), leafy vegetables, greens subgroup	10.0	None
Brassica (cole), leafy vegetables, head and stem subgroup	2.0	None
Cattle, fat	0.6	None
Cattle, meat	0.04	None
Cattle, meat byproducts	0.2	None

Commodity	Parts per million	Expiration/Revocation Date
Citrus fruits group	0.3	None
Citrus oil	3.0	None
Citrus pulp, dried	0.5	None
Coffee	0.02	8/28/00
Cotton gin byproducts	1.5	None
Cottonseed	0.02	None
Fruiting vegetables (except cucurbits) group	0.4	None
Goat, fat	0.6	None
Goat, meat	0.04	None
Goat, meat byproducts	0.2	None
Hogs, fat	0.6	None
Hogs, meat	0.04	None
Hogs, meat byproducts	0.2	None
Horses, fat	0.6	None
Horses, meat	0.04	None
Horses, meat byproducts	0.2	None
Leafy vegetables (except Brassica vegetables) group	8.0	None
Milk, fat	0.5	None
Milk, whole	0.04	None
Sheep, fat	0.6	None
Sheep, meat	0.04	None
Sheep, meat byproducts	0.2	None

* * * * *

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 300

[FRL–6131–1]

National Priorities List for Uncontrolled Hazardous Waste Sites, Proposed Rule No. 25

AGENCY: Environmental Protection Agency.

ACTION: Proposed rule.

SUMMARY: The Comprehensive Environmental Response, Compensation, and Liability Act (''CERCLA'' or ''the Act''), requires that the National Oil and Hazardous Substances Pollution Contingency Plan (''NCP'') include a list of national priorities among the known releases or threatened releases of hazardous substances, pollutants, or contaminants throughout the United States. The National Priorities List (''NPL'') constitutes this list. The NPL is intended primarily to guide the Environmental Protection Agency (''EPA'' or ''the Agency'') in determining which sites warrant further investigation to assess the nature and extent of public health and environmental risks associated with the site and to determine what CERCLA-financed remedial action(s), if any, may be appropriate.

This rule proposes to add 14 new sites to the NPL, 11 to the General Superfund section and 3 to the Federal facilities section.

DATES: Comments regarding any of these proposed listings must be submitted (postmarked) on or before September 28, 1998, EPA has changed its policy and will normally no longer respond to late comments.

ADDRESSES: By Postal Mail: Mail original and three copies of comments (no facsimiles or tapes) to Docket Coordinator, Headquarters; U.S. EPA; CERCLA Docket Office; (Mail Code 5201G); 401 M Street, SW; Washington, DC 20460; 703/603–9232.

By Express Mail: Send original and three copies of comments (no facsimiles or tapes) to Docket Coordinator, Headquarters; U.S. EPA; CERCLA Docket Office; 1235 Jefferson Davis Highway; Crystal Gateway #1, First Floor; Arlington, VA 22202.

By E-Mail: Comments in ASCII format only may be mailed directly to SUPERFUND. DOCKET@EPA.GOV. E-mailed comments must be followed up by an original and three copies sent by mail or Federal Express.

For additional Docket addresses and further details on their contents, see Section II, ''Public Review/Public Comment,'' of the Supplementary Information portion of this preamble.

FOR FURTHER INFORMATION CONTACT: Terry Keidan, phone (703) 603–8852, State, Tribal and Site Identification Center, Office of Emergency and Remedial Response (Mail Code 5204G), U.S. Environmental Protection Agency, 401 M Street, SW, Washington, DC, 20460, or the Superfund Hotline, Phone (800) 424–9346 or (703) 412–9810 in the Washington, DC, metropolitan area.

SUPPLEMENTARY INFORMATION:

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