goat fat at 0.05 ppm, goat meat at 0.05 ppm, goat mbyp at 0.10 ppm, hog fat at 0.05 ppm, hog meat at 0.05 ppm, hog mbyp at 0.10 ppm, horse fat at 0.05 ppm, horse meat at 0.05 ppm, horse mbyp at 0.10 ppm, milk at 0.02 ppm, poultry fat at 0.05 ppm, poultry meat at 0.05 ppm, poultry mbyp at 0.10 ppm, sheep fat at 0.05 ppm, sheep meat at 0.05 ppm, and sheep mbyp at 0.10 ppm. Almonds are not considered a poultry feed commodity under present EPA Guidelines, and AgrEvo USA Co. has requested that the proposed tolerances for secondary residues in eggs, poultry fat, meat, and meat byproducts be deleted from the tolerances requested. This document also amends 40 CFR 180.473 to change the chemical expression for the herbicide to that given above in conformity with Chemical Abstract nomenclature.

The chemical expression for glufosinate ammonium has been changed to follow that given by the Chemical Abstracts Index Name for this chemical. This action is taken in concert with the final rule for Premanufacture Notification; Revisions of Premanufacture Notification Regulations, published in the Federal Register of March 29, 1995 (60 FR 16298-16310). The proposed analytical method for determining residues is high-pressure liquid chromatography.

There were no comments or requests for referral to an advisory committee received in response to the notice of filing.

The data submitted in the petition and other relevant material have been evaluated. The toxicology data listed below were considered in support of these tolerances.

1. A battery of acute toxicity studies placing technical glufosinate-ammonium in Toxicity Categories II and III.

2. A 90-day feeding study in rats at dietary intakes of 0, 0.52, 4.1, 32, or 263 mg/kg/day with a no-observed-effect level (NOEL) of 4.1 mg/kg/day. The lowest-observed-effect level (LOEL) was established at 32 mg/kg/day based on increased absolute and relative kidney weights.

3. A 90-day feeding study in mice at dietary intakes of 0, 16.6, 67.1, or 278 mg/kg/day with a NOEL of 16.6 mg/kg/day and an LOEL of 67.1 mg/kg/day based on increased absolute and relative liver weights (both sexes) and an increase in serum potassium levels (males).

4. Three teratology studies in rats at doses from 0.5 to 250 mg/kg/day with no teratogenic effects occurring up to and including 250 mg/kg/day. A NOEL for developmental toxicity was 2.24 mg/kg/day, based upon an increase in the incidence of dilated renal pelvis with hydroureter in the fetuses at 10 mg/kg/day. The maternal NOEL was also 2.24 mg/kg/day.

5. A teratology study in rabbits at doses of 0, 2, 6.3, or 20 mg/kg/day with no teratogenic effects occurring up to and including 20 mg/kg/day, and a maternal NOEL of 6.3 mg/kg/day and a developmental NOEL of 20 mg/kg/day, the highest dose tested.

6. A two-generation reproduction study in rats at dietary concentrations of 0, 40, 120, or 360 ppm with an NOEL for reproductive effects at 120 ppm (equivalent to 12 mg/kg/day) based upon reduced number of pups in the high-dose group. The NOEL for parental toxicity was also 120 ppm based upon increased kidney weights in the high-dose group.

7. A 12-month feeding study in dogs at doses of 0, 2, 5, or 8.5 mg/kg/day. The NOEL was 5.0 mg/kg/day based upon the death of one male and one female dog at 8.5 mg/kg/day with no other treatment-related toxicity.

8. A mouse carcinogenicity study at doses of 0, 2.8, 10.8, or 22.7 mg/kg/day in males and 0, 4.2, 16.2, or 64.0 mg/kg/day in females for 104 weeks with no carcinogenic effects observed under the conditions of the study up to and including 64 mg/kg/day and a systemic NOEL of 10.8 and 16.2 for males and females, respectively, based on the dose-related increase in mortality.

9. A chronic feeding/carcinogenicity study in rats at dietary doses of 0, 2.5, 8.8, or 31.5 mg/kg/day (males) and 0, 2.4, 8.2, or 28.7 mg/kg/day (females) with an NOEL of 2.1 mg/kg/day for systemic effects based on an increase in mortality rate in females at the two higher doses. There were no treatment-related carcinogenic effects at any dose level.

10. Acceptable studies on gene mutation (*Salmonella*, *E coli*., and mouse lymphoma assays), structural chromosomal aberration (*in vivo* micronucleus assay in mice), and other genotoxic effects (unscheduled DNA synthesis assay with rat hepatocytes) yielded negative results.

11. Pharmacokinetic and metabolism studies in rats indicated that approximately 80 to 90 percent of the orally administered dose of glufosinate ammonium remained unabsorbed and was eliminated in the feces. Approximately 10 to 15 percent was eliminated in the urine. The major metabolic pathway is oxidative deamination yielding the metabolite, 3-methyl-phospinico propionic acid.

The chronic analysis used a Reference Dose (RfD) of 0.02 mg/kg/ body weight day, based on an NOEL of 2.1 mg/kg/day and an uncertainty factor of 100. The NOEL is based on a 2-year rat feeding study that demonstrated increased absolute and relative kidney weight in males as an endpoint effect.

Using tolerance-level residues and assumptions that 100 percent of every crop for which glufosinate-ammonium has a proposed use is treated, the total Theoretical Maximum Residue Contribution (TMRC) for the general population and the highest exposed subgroup in DRES are as follows (as percents of RFD): General population, 0.627 percent; nonnursing infants less than 1-year-old, 3.7 percent.

A data gap currently exists for a rat carcinogenicity study. All tolerances are time-limited because of this gap. The time limitation allows for development and review of the data.

The analysis for glufosinate-ammonium using tolerance level residues suggests that the proposed uses on apples, grapes, and tree nut group will not cause exposure to exceed the levels at which the Agency believes there is an appreciable risk. All DRES subgroups are below 100 of the RfD for chronic effects.

The pesticide is useful for the purposes for which these tolerances are sought. The nature of the residues is adequately understood for the purpose of establishing these tolerances. Adequate analytical methodology (gas chromatography with flame photometric detection of phosphorus) is available for enforcement purposes. Because of the long lead time from establishing these tolerances to publication, the enforcement methodology is being made available in the interim to anyone interested in pesticide enforcement when requested by mail from: Calvin Furlow, Public Response Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 1130A, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703)-305-5937.

Based on the information cited above, the Agency has determined that the establishment of the time-limited tolerances by amending 40 CFR 180.473 will protect the public health; therefore, the time-limited tolerances are established as set forth below.

Any person adversely affected by this regulation may, within 30 days after publication of this document in the Federal Register, file written objections and/or request a hearing with the Hearing Clerk, at the address given above (40 CFR 178.20). A copy of the objections and/or hearing requests filed