SUPPLEMENTARY INFORMATION: EPA issued a notice in the Federal Register of October 12, 1988 (53 FR 39785), that announced that the Monsanto Co., 1101 17th St., NW., Washington, DC 20036, proposed amending 40 CFR 180.249 by establishing a regulation under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a, to permit the residues of the herbicide alachlor (2-chloro-2',6'-dimethyl-*N*-(methoxymethyl) acetanilide) and its metabolites in or on sorghum forage at 2.0 parts per million (ppm) (pesticide petition (PP) 8F3671). This increased tolerance was necessary because review of additional data submitted in response to reregistration indicated that the current tolerance of 1.0 for sorghum forage was not adequate and needed to be increased. EPA issued a notice in the Federal Register of March 23, 1989 (54 FR 12010), which announced that the Monsanto Co. proposed amending 40 CFR parts 185 and 186 by establishing a regulation under section 409 of the FFDCA, 21 U.S.C. 348, permitting residues of the herbicide alachlor in or on sorghum milling fractions at 0.5 ppm, sorghum milling fractions (except germ) at 0.3 ppm, and sorghum germ at 0.5 ppm (food/feed additive (FAP) 9H5576).

No comments were received in response to these notices of filing.

During the course of its review, the Agency determined that the food/feed additive tolerances for sorghum milling fractions and sorghum germ were not needed and that there is no current evidence of use of sorghum milling fractions as a human food and very limited evidence of use of soghum milling fractions as livestock feed. The petitioner subsequently withdrew FAP No. 9H5576. Because it has been longer than 5 years since the original proposal, the tolerance of 2.0 ppm for sorghum forage is being proposed for 30 days following the date of publication in the Federal Register to allow for public comment.

The data submitted in the petition and other relevant material have been evaluated. The pesticide is considered useful for the purpose for which the tolerance is sought. The toxicological data listed below were considered in support of the proposed tolerance.

1. Several acute toxicology studies place technical alachlor in acute toxicity category IV for primary eye and dermal irritation and, acute toxicity category III for acute oral, dermal, and inhalation.

2. A 1-year feeding study with dogs fed dose levels of 0, 1, 3, and 10 milligrams/kilograms/day (mg/kg/day) with a no-observed effect level (NOEL) of 1.0 mg/kg/day based on hemosiderin storage in kidney and spleen in males at 10 mg/kg.

3. A 2-year chronic feeding/carcinogenicity study in rats fed epichlorohydrin-free alachlor at dose levels of 0, 0.5, 2.5, and 15 mg/kg/day with a NOEL for nonneoplastic toxicity at 2.5 mg/kg/day based on ocular lesions and hepatoxicity at 10 mg/kg/day. Carcinogenic effects included a nasal turbinate tumor in females at 2.5 mg/kg/day, significant increases in nasal turbinate tumors in both males and females at 15 mg/kg/day (highest dose tested (HDT)) and a significant increase in thymus lymphosarcomas and adrenal pheochromocytomas in high-dose females.

4. A second chronic feeding/carcinogenic study with rats fed alachlor, with epichlorohydrin, at dose levels of 0, 14, 42, and 126 mg/kg/day with a systemic NOEL of less than 14 mg/kg/day based on ocular lesions and hepatotoxicity at 14 mg/kg/day. Carcinogenic effects included increased number of nasal turbinate tumor in males and females at 42 mg/kg/day and mg/kg/day, an increase in stomach tumors in both sexes at 126 mg/kg/day, and an increase in thyroid follicular tumors in males at 126 mg/kg/day (HDT).

5. A special chronic feeding study in rats fed a dose level of 126 mg/kg/day. Ocular lesions, mainly, the uveal degeneration syndrome (UDS) occurred in 100% of the animals at the end of the study. This syndrome was irreversible once it began. Alachlor was a positive oncogen with increased nasal turbinate tumors, stomach tumors, and thyroid tumors.

6. An 18-month carcinogenicity study in mice fed dose levels of 0, 26, 78, and 260 mg/kg/day with carcinogenic effects (increased lung bronchiolaraveolar tumors in females at 260 mg/kg/day).

7. A three-generation reproduction study with rats fed dose levels of 0, 3, 10, 11, and 30 mg/kg/day with a reproductive NOEL of 10 mg/kg/day based on kidney effects in F2 and F3 pups at 30 mg/kg/day (HDT).

8. A developmental toxicity study in rats fed dose levels of 0, 50, 150, and 400 mg/kg/day with a developmental toxicity equal to a greater than 400 mg/kg/day with a fetotoxic NOEL of 150 mg/kg/day based on an increase in post-implantation loss and a slight decrease in mean number of viable fetuses at 400 mg/kg/day . The maternal toxicity NOEL for this study is 150 mg/kg/day based on soft stools, hair loss, anogenital staining, and death at 400 mg/kg/day.

9. A developmental toxicity study in rabbits fed doses of 50, 100, and 150 mg/kg/day with a developmental NOEL greater than 150 mg/kg/day greater than 150 mg/kg/day. The maternal NOEL was 100 mg/kg/day based on reduced body weight gain.

10. Mutagenicity studies include several Ames Tests. Alachlor and its metabolites were negative in four Ames assays with *Salmonella* with and without S9 activation at 0.1 to 10 mg/plate. Two metabolites of alachlor were positive in an Ames test with and without S9 activation at 0.01 to 10 mg/plate. Bile from alachlor-treated rates did not induce a mutagenic response towards *Salmonella* strains TA98, TA100, TA1535, and TA1537. Other mutagenicity tests include DNA damage/repair in rat positive for UDS at the HDT = LD_{50} at the 4 doses tested (50, 200, and 1,000 mg/kg)—weakly genotoxic; gene mutation in CHO/HGPRT—negative, and *in vivo* bone marrow chromosome aberration assay—negative.

Alachlor has been classified as a B_2 carcinogen—''Probable Human Carcinogen'' by the Agency. Alachlor met all but one of the criteria specified for the B_2 classification. Alachlor produced an increased incidence of nasal turbinate tumors (mostly benign) at the mid and high doses, in both sexes, thyroid follicular tumors in male rats and malignant stomach tumors in male and female rats in Long-Evans rats in three different experiments at more than one dose level via dietary administration. Alachlor also produced a statistically significant increase in lung tumors in female CD-1 mice at two dose levels. In another experiment with Long-Evans rats, nasal turbinate tumors occurred only 5 to 6 months after exposure. The tumor incidence was as high at 50% and tumor site was unusual, i.e., not an increase of normal high background tumor type. A metabolite of alachlor was mutagenic in the Ames Test at 6 dose levels, and alachlor is structurally similar to acetochlor and metolachlor, two other known carcinogens. A detailed discussion of the Agency's classification of alachlor as a B_2 carcinogen was published in the Federal Register of December 31, 1987 (52 FR 49480). The publication was entitled ''Alachlor, Notice of Intent to Cancel Registrations, Conclusion of Special Review.''

For the purpose of risk characterization of alachlor, the use of the linearized multi-stage model, as recommended to EPA's Carcinogenic Risk Assessment Guidelines, was applied to the rat oncogenicity data discussed above. As a result, the cancer potency value for alachlor, known as the ''Q^{*1}'', was calculated to be 8×10^{-2} or $0.08\ (mg/kg/day)^{-1}$. Refer to the