incidence of hepatocellular adenomas at 1,500 ppm for female mice, and borderline statistical significance was attained for hepatocellular adenomas at

50 ppm for male mice.

6. A developmental toxicity study in rats at dietary levels of 50, 125, or 625 ppm (5.0, 12.1, or 49.8 mg/kg/day), administered on days 6 to 15 of gestation with NOELs for maternal systemic toxicity and developmental toxicity established at 125 ppm. The LOEL of 625 ppm for maternal systemic toxic effects was based upon decreased body weight and food consumption values. The developmental toxicity LOEL of 625 ppm was based on increases in post-implantation loss and increases in the litter and fetal incidence of resorptions.

7. A developmental toxicity study in rabbits given gavage dosages of 5, 25, or 100 mg/kg/day on days 7 through 19 of gestation with a NOEL for developmental toxicity of 25 mg/kg/day and a NOEL for maternal toxicity of 5 mg/kg/day. The LOEL for maternal systemic toxicity (reduced body weight) was established at 25 mg/kg/day. The LOEL for developmental toxicity was established at 100 mg/kg/day based on an increased number of abortions, decreased mean number of fetuses per litter, decreased fetal body weight, and increased incidence of fetuses with skeletal variations of the skull at that

dosage level.

8. A two-generation reproductive toxicity study in rats, which were fed diets containing 12.5, 100, or 625 ppm (equivalent to 0.84, 6.8, or 44.75 mg/kg/ day for males; 1.0, 8.3, or 54.1 mg/kg/ day for females), with no evidence of adverse effects on fertility or reproductive performance under the conditions of the study. The NOEL for parental systemic toxicity was established at 12.5 ppm based upon decrements in parental body weight gain. In addition, the results of this study support the hypothesis that rats exposed to linuron could develop interstitial cell hyperplasia and subsequent adenomas (Leydig cell tumors) of the testicular tissue via a mechanism of sustained hypersecretion of luteinizing hormone induced by the antiandrogenic potential of linuron.

9. Linuron did not produce gene mutation in an Ames assay or in an *in vitro* assay using Chinese hamster ovary cells. Linuron did not induce bone marrow chromosome aberrations *in vivo* and in other tests for genotoxicity. Linuron did not induce unscheduled DNA synthesis in isolated rat

hepatocytes.

10. Metabolism studies in rats show that linuron was extensively

metabolized by male and female rats when administered by gavage, and there is no indication of accumulation of linuron or its metabolites in tissues and organs.

Linuron was placed in Special Review for carcinogenicity in 1982. It was later classified as a group C carcinogen (possible human carcinogen) with quantified cancer risk on the basis of a dose-related increase in interstitial cell hyperplasia and adenomas in the 2-year rat feeding study and hepatocellular tumors that appeared in low-dose male and high-dose female mice in a 2-year feeding study. Subsequent review by the Office of Pesticide Programs, Health Effects Division, Peer Review Committee and the Science Advisory Panel resulted in the decision to regulate linuron as a possible human carcinogen without quantified cancer risk. This decision was based on the weight-of-evidence, which suggested that the carcinogenic potential of linuron in humans is weak.

Dietary risk assessments for linuron were conducted using the Reference Dose (RfD) to assess chronic exposure and risk and the Margin of Exposure (MOE) for acute toxicity. The RfD for linuron is established at 0.008 mg/kg of body weight/day, based on a NOEL of 0.77 mg/kg/day from the 1-year feeding study in dogs and an uncertainty factor of 100. The anticipated residue contribution (ARC) from published tolerances and the proposed 7-ppm tolerance for asparagus utilizes 2 percent of the RfD for the general population. The ARC for the subgroup most highly exposed, nonnursing infants (less than 1-year old), utilizes 6 percent of the RfD. EPA concludes that established tolerances and the proposed increased tolerance for asparagus pose a negligible dietary risk to humans. The MOE is a measure of how closely acute dietary exposure comes to the NOEL from the toxicity endpoint of concern. For linuron, the MOE was calculated as a ratio of the NOEL (25 mg/kg/day) from the rabbit developmental toxicity study to dietary exposure (0.03125 mg/kg/ day), as estimated by the high-end exposure for the population subgroup at greatest risk (females of childbearing age). The MOE for this subgroup is estimated at 800 for high-end exposure. Acute dietary margins of exposure of less than 100 are generally of concern to EPA. A MOE of 800 poses minimal risk.

The nature of the residue in plants is adequately understood. An adequate analytical method has been published in Pesticide Analytical Manual, Vol. II (PAM Vol. II).

There is no reasonable expectation that secondary residues will occur in

milk, and eggs, or meat, fat and meat byproducts of livestock and poultry; there are no livestock feed items associated with asparagus.

There are currently no actions pending against the continued registration of this chemical.

Based on the information and data considered, the Agency has determined that amending 40 CFR 180.184 to increase the tolerance for linuron from 3 ppm to 7 ppm would protect the public health. Therefore, it is proposed that the tolerance be established as set forth below.

Any person who has registered or submitted an application for registration of a pesticide, under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) as amended, which contains any of the ingredients listed herein, may request within 30 days after publication of this notice in the Federal Register that this rulemaking proposal be referred to an Advisory Committee in accordance with section 408(e) of the FFDCA.

A record has been established for this rulemaking under docket number [PP 5E4464/P629] (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 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The public record is located in Rm. 1132 of the Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

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.

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 all comments 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 address in "ADDRESSES" at the beginning of this document.

Under Executive Order 12866 (58 FR 51735, Oct. 4, 1993), the Agency must