based on decreases in mean body weights, body weight gains, and food consumption, and an increase in liver *N*-demethylase activity.

3. A 1-year dog feeding study with a NOEL of 1 mg/kg bw/day (40 ppm) and an LEL of 5 mg/kg bw/day (200 ppm), based on lenticular and corneal opacity and hepatic toxicity in either sex (the current Reference Dose was determined based on this study). A subsequent 1-year dog feeding study, using lower doses to further define the NOEL for tebuconazole, defines a systemic LOEL of 150 ppm (based on adrenal effects in both sexes) and a systemic NOEL of 100 ppm.

4. A 2-year rat chronic feeding study defined, a NOEL of 7.4 mg/kg bw/day (100 ppm), and an LEL of 22.8 mg/kg bw/day (300 ppm) based on body weight depression, decreased hemoglobin, hematocrit, MCV and MCHC, and increased liver microsomal enzymes in females. Tebuconazole was not oncogenic at the dose levels tested (0, 100, 300, and 1,000 ppm).

5. A rat oral developmental toxicity study with a maternal NOEL of 30 mg/kg bw/day and an LEL of 60 mg/kg bw/day based on elevation of absolute and relative liver weights. For developmental toxicity, a NOEL of 30 mg/kg bw/day and an LEL of 60 mg/kg bw/day was determined, based on delayed ossification of thoracic, cervical and sacral vertebrae, sternum, fore and hind limbs and increase in supernumerary ribs.

6. A rabbit oral developmental toxicity study with a maternal NOEL of 30 mg/kg bw/day and an LEL of 100 mg/kg bw/day based on depression of body weight gains and food consumption. A developmental NOEL of 30 mg/kg bw/day and an LEL of 100 mg/kg bw/day were based on increased post-implantation losses, from both early and late resorptions and frank malformations in eight fetuses of five litters.

7. A mouse oral developmental toxicity study with a maternal NOEL of 10 mg/kg bw/day and an LEL of 20 mg/kg bw/day based on a supplementary study indicating reduction in hematocrit and histological changes in liver. A developmental NOEL of 10 mg/kg bw/day and an LEL of 30 mg/kg bw/day based on dose-dependent increases in runts/dam at 30 and 100 mg/kg bw/day.

8. A mouse dermal developmental toxicity study with a maternal NOEL of 30 mg/kg bw/day and an LEL of 60 mg/kg bw/day based on a supplementary study indicating increased liver microsomal enzymes and histological changes in liver. The NOEL for developmental toxicity in the dermal study in the mouse is 1,000 mg/kg bw/day, the highest dose tested (HDT).

9. A two-generation rat reproduction study with a dietary maternal NOEL of 15 mg/kg bw/day (300 ppm) and an LEL of 50 mg/kg bw/day (1,000 ppm) based on depressed body weights, increased spleen hemosiderosis, and decreased liver and kidney weights. A reproductive NOEL of 15 mg/kg bw/day (300 ppm) and an LEL of 50 mg/kg bw/day (1,000 ppm) were based on neonatal birth weight depression.

10. An Ames mutagenesis study in *Salmonella* that showed no mutagenicity with or without metabolic activation.

11. A micronucleus mutagenesis assay study in mice that showed no genotoxicity.

12. A sister chromatid exchange mutagenesis study using CHO cells that was negative at dose levels 4 to 30 *u*g/mL without activation or 15 to 120 *u*g/mL with activation.

13. An unscheduled DNA synthesis (UDS) study that was negative for UDS in rat hepatocytes.

Additionally, a mouse oncogenicity study at dietary levels of 0, 20, 60, and 80 ppm for 21 months did not reveal any oncogenic effect for tebuconazole at any dose tested. Because the maximum-tolerated-dose (MTD) was not reached in this study, the study was classified as supplementary. A followup mouse study at higher doses (0, 500, and 1,500 ppm in the diet), with an MTD at 500 ppm, revealed statistically significant incidences of hepatocellular adenomas and carcinomas in males and carcinomas in females. The initial and followup studies, together with supplementary data submitted by Miles, Inc., were classified as core minimum.

The Office of Pesticide Programs' Health Effects Division's Carcinogenicity Peer Review Committee (CPRC) has classified tebuconazole as a Group C carcinogen (possible human carcinogen). This classification is based on the Agency's ''Guidelines for Carcinogen Risk Assessment'' published in the Federal Register of September 24, 1986 (51 FR 33992). The Agency has chosen to use the reference dose calculations to estimate human dietary risk from tebuconazole residues. The decision supporting classification of tebuconazole as a possible carcinogen (Group C) rather than a probable carcinogen (Group B) was primarily based on the statistically significant increase in the incidence of hepatocellular adenomas, carcinomas, and combined adenomas/carcinomas in both sexes of NMRI mice both by positive trend and pairwise comparison at the HDT, and the structural correlation with at least six other related triazole pesticides that produce liver tumors.

The Reference Dose (RfD) is established at 0.01 mg/kg of body weight (bwt)/day, based on a no-observed-effect level (NOEL) of 1.00 mg/kg bwt/day and an uncertainty factor of 100. The NOEL is based on a 1-year dog-feeding study that demonstrated lenticular and corneal opacity and hepatic toxicity as an endpoint effect. A chronic exposure analysis was performed using tolerance level residues and 100 percent crop-treated information to estimate the Theoretical Maximum Residue Contribution (TMRC) for the general population and 22 subgroups.

The Theoretical Maximum Residue Contribution (TMRC) from the published uses is estimated at 0.000008 mg/kg bwt/day and utilizes 0.075% of the RfD for the general population of the lower 48 States. The proposed use on peaches, cherries, and necatrines contributes 0.000377 mg/kg bwt/day (3.8% of the RfD) which raises the TMRC to 0.000385 mg/kg bwt/day or 3.9% of the RfD.

The TMRC for the most highly exposed subgroup, nonnursing infants (less than 1-year old) is 0.000003 mg/kg bwt/day which represents 0.03% of the RfD. The proposed use on peaches, cherries, and nectarines for nonnursing infants (less than 1-year old) raises the TMRC to 0.002525 or 25.3% of the RfD.

The nature of the residue in cherries, peaches, and nectarines is adequately understood. An adequate analytical method using gas chromatography is available for enforcement purposes.

The enforcement methodology has been submitted to the Food and Drug Administration for publication in the Pesticide Analytical Manual, Volume II (PAM II). Because of the long lead time for publication of the method in PAM II, the analytical methodology is being made available in the interim to anyone interested in pesticide enforcement when requested from: Calvin Furlow, Public Response and Program Resources 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. 1132, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703)-305-5232.

There is no reasonable expectation that secondary residues will occur in milk, eggs, or meat of livestock and poultry since there are no livestock feed items associated with this action.