

carcinogenic to humans. As such, famoxadone is not expected to pose a cancer dietary risk.

*iv. Anticipated residue and percent crop treated (PCT) information.*

Section 408(b)(2)(E) of the FFDCA authorizes EPA to use available data and information on the anticipated residue levels of pesticide residues in food and the actual levels of pesticide chemicals that have been measured in food. If EPA relies on such information, EPA must require that data be provided 5 years after the tolerance is established, modified, or left in effect, demonstrating that the levels in food are not above the levels anticipated. Following the initial data submission, EPA is authorized to require similar data on a time frame it deems appropriate. No PCT information was used in the risk assessment. The Agency used 100% which would over estimate exposure.

*2. Dietary exposure from drinking water.*

The Agency lacks monitoring exposure data to complete a comprehensive dietary exposure analysis and risk assessment for famoxadone in drinking water because this is a new chemical. Because the Agency does not have comprehensive monitoring data, drinking water concentration estimates are made by reliance on simulation or modeling taking into account data on the physical characteristics of famoxadone.

The Agency uses the FQPA Index Reservoir Screening Tool (FIRST) or the Pesticide Root Zone/Exposure Analysis Modeling System (PRZM/EXAMS) to estimate pesticide concentrations in surface water and SCI-GROW, which predicts pesticide concentrations in groundwater. In general, EPA will use FIRST (a tier 1 model) before using PRZM/EXAMS (a tier 2 model) for a screening-level assessment for surface water. The FIRST model is a subset or meta-model of the PRZM/EXAMS model that uses specific high-end runoff scenario for pesticides. FIRST incorporates an index reservoir environment and a percent crop area (PCA), while PRZM/EXAMS incorporate an index reservoir environment, PCA, all available information on the pesticide's fate and use pattern, and site-specific cropping information.

None of these models include consideration of the impact processing (mixing, dilution, or treatment) of raw water for distribution as drinking water would likely have on the removal of pesticides from the source water. The primary use of these models by the Agency at this stage is to provide a screen for sorting out pesticides for which it is highly unlikely that drinking

water concentrations would exceed human health levels of concern.

Since the models used are considered to be screening tools in the risk assessment process, the Agency does not use estimated environmental concentrations (EECs) from these models to quantify drinking water exposure and risk as a %RfD or %PAD. Instead drinking water levels of comparison (DWLOCs) are calculated and used as a point of comparison against the model estimates of a pesticide's concentration in water. DWLOCs are theoretical upper limits on a pesticide's concentration in drinking water in light of total aggregate exposure to a pesticide in food, and from residential uses. Since DWLOCs address total aggregate exposure to famoxadone they are further discussed in the aggregate risk sections in Unit E.

Based on the PRZM/EXAMS and SCI-GROW models the EECs of famoxadone for chronic exposures are estimated to be 0.47 parts per billion (ppb) for surface water and 0.23 ppb for ground water.

*3. From non-dietary exposure.* The term "residential exposure" is used in this document to refer to non-occupational, non-dietary exposure (e.g., for lawn and garden pest control, indoor pest control, termiticides, and flea and tick control on pets).

Famoxadone is not registered for use on any sites that would result in residential exposure.

*4. Cumulative exposure to substances with a common mechanism of toxicity.* Section 408(b)(2)(D)(v) of the FFDCA 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."

EPA does not have, at this time, available data to determine whether famoxadone 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, famoxadone does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that famoxadone has a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such

chemicals, see the final rule for Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997).

*D. Safety Factor for Infants and Children*

*1. In general.* Section 408 of the FFDCA provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base on toxicity and exposure 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.

*2. Prenatal and postnatal sensitivity.* The Agency concluded that there is not a concern for pre- and/or postnatal toxicity resulting from exposure to famoxadone.

No quantitative or qualitative evidence of increased susceptibility, as compared to adults, of rat or rabbit fetuses to *in utero* exposure to famoxadone was observed in the developmental toxicity studies. No quantitative or qualitative evidence of increased susceptibility, as compared to adults, of rat fetuses or neonates was observed in the 2-generation reproduction study.

In the rat developmental toxicity study, the NOAEL for maternal toxicity was 250 mg/kg/day and the LOAEL was 500 mg/kg/day, based on transient decreases in body weight gain and food consumption. At 1,000 mg/kg/day, no additional treatment-related effects were observed in the dams. No developmental toxicity was observed in the rat study. The NOAEL for developmental toxicity was 1,000 mg/kg/day, the highest dose tested.

In the rabbit developmental toxicity study, the maternal and developmental NOAELs and LOAELs were the same. The NOAEL for maternal toxicity and developmental toxicity was 350 mg/kg/day. The LOAEL for maternal toxicity was 1,000 mg/kg/day, based on abortions in 4 out of 17 does; markedly decreased body weight, reduced body weight gain and reduced food consumption in the same 4 does, and increased number of does with abnormal or little or no stools. The LOAEL for developmental toxicity was 1,000 mg/kg/day; based on abortions in 4 out of 17 does; and equivocal increases in percent post implantation loss and mean number of resorptions