

acute exposures are estimated to be 100 parts per billion (ppb) for surface water and 1 ppb for ground water. The EECs for chronic exposures are estimated to be 20 ppb for surface water and 1 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). Sethoxydim is currently registered for use on the following residential non-dietary sites: Ornamentals and flowering plants, recreational areas, and buildings/structures (non-agricultural-outdoor). The risk assessment was conducted using the following exposure assumptions:

i. *Residential handler.* There is potential sethoxydim exposure to residential handlers who mix, load and apply sethoxydim for use on residential turf and ornamentals. Because dermal toxicity endpoints were not identified, only the following exposure scenarios were assessed:

- Adult inhalation exposure from mixing/loading/applying sethoxydim for spot treatment with a low-pressure handwand.
- Adult inhalation exposure from mixing/loading/applying sethoxydim for spot treatment with a hose-end sprayer.

ii. *Residential post-application.* The labeled use pattern for sethoxydim only suggests spot treatments for non-agricultural sites (e.g., fence lines, at base of ornamental plantings, etc.). The Agency considered the potential residential post-application exposure from spot treatment to be negligible. However, an exposure/risk assessment for broadcast turf treatment, using the applicable endpoints, was included in this assessment because there is no labeled recommendation against broadcast treatment of lawns. Sethoxydim treatment may take up to 3 weeks before visible burnback of turf is seen, and the previous risk assessment for other agricultural use sites included residential post-application exposure from turf use.

Broadcast turf treatment would result in the potential for dermal (adults and children) and incidental oral exposure (children only) during post-application activities. However, because the appropriate dermal toxicity endpoints for sethoxydim were not identified, and because inhalation is considered negligible for post-application exposure, only the following post-application exposure scenarios were assessed:

1. Incidental non-dietary ingestion of pesticide residues on lawns from hand-to-mouth transfer.

2. Incidental non-dietary ingestion of residues from object-to-mouth activities (pesticide-treated turfgrass).

3. Incidental non-dietary ingestion of soil (base of pesticide-treated ornamentals).

Post-application exposures from various activities following lawn treatment are considered to be the most common and significant in residential settings.

The exposure via incidental non-dietary ingestion involving other plant material (i.e., resulting from children's handling of treated ornamentals) may occur but is considered negligible.

The exposure and risk estimates for the three residential exposure scenarios are assessed for the day of application (day "0") because it is assumed that toddlers could contact the lawn immediately after application. On the day of application, it was assumed that 5% of the application rate is available from the turf grass as transferrable residue (20% for object-to-mouth activities). Intermediate-term exposure is also expected (up to 6 months) because reapplications are not limited, and may be necessary to continue suppression of grass. The application rates used for turf and ornamental gardens are 0.33 and 0.49 lb active ingredient acres (ai/A) respectively.

4. *Cumulative effects from 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 sethoxydim has a common mechanism of toxicity with other substances. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, EPA has not made a common mechanism of toxicity finding as to sethoxydim and any other substances and sethoxydim 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 sethoxydim 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 policy statements released by EPA's Office of Pesticide Programs concerning common mechanism determinations and procedures for cumulating effects from substances found to have a common mechanism on EPA's website at <http://www.epa.gov/pesticides/cumulative/>.

#### *D. Safety Factor for Infants and Children*

1. *In general.* Section 408 of the FFDCA provides that EPA will 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 margin of exposure (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.* EPA determined that there are no residual uncertainties for prenatal and/or postnatal toxicity based on the following:

i. There was evidence of qualitative susceptibility in the developmental rat study with the occurrence of more severe effects in the fetuses (tail abnormalities and delayed ossification) than the maternal animals (clinical signs of neurotoxicity). Tail abnormalities were also seen in the F1a and F1b offspring of the 2-generation reproduction rat study. However, the degree of concern is low for the fetal effects in the developmental rat study since the fetal anomalies were seen only at the high dose 650 mg/kg/day which is close to the Limit Dose (LTD) (1,000 mg/kg/day). They were seen in the presence of maternal toxicity (clinical signs of neurotoxicity) and clear NOAELs/LOAELs were established for maternal and developmental toxicities.

ii. Evidence of quantitative susceptibility was indicated in the 2-generation reproduction rat study, by a slightly higher decrease (11–13%) in the body weights of offspring during lactation as compared to an 8–10% decrease in the body weights of the parental animals. Again, the degree of concern is also low for the 2-generation reproduction rat study since the LOAEL for offspring toxicity is based on a conservative determination of a minimal response in pup body weight decrements at the same dose that also caused decreases in body weights in the parental animals.