

TABLE 1.—SUMMARY OF TOXICOLOGICAL DOSE AND ENDPOINTS FOR IMIDACLOPRID FOR USE IN HUMAN RISK ASSESSMENT—Continued

Exposure Scenario	Dose Used in Risk Assessment, UF	FQPA SF* and Level of Concern for Risk Assessment	Study and Toxicological Effects
Intermediate-term dermal (1–6 months)	Oral study NOAEL = 9.3 mg/kg/day (dermal absorption rate = 7.2%)	LOC for MOE = 100 (occupational) LOC for MOE = 100 (residential, includes the FQPA SF)	Subchronic neurotoxicity - rat LOAEL = 63.3 mg/kg/day, based upon decreased body weight gain
Long-term dermal (6 months)	Oral study NOAEL = 5.7 mg/kg/day (dermal absorption rate = 7.2%)	LOC for MOE = 100 (occupational) LOC for MOE = 100 (residential, includes the FQPA SF)	Combined chronic toxic/carcinogenicity - rat LOAEL = 16.9 mg/kg/day, based upon increased incidence of mineralized particles in thyroid colloid in males
Short-term inhalation (1–30 days)	Oral study NOAEL = 10 mg/kg/day (inhalation absorption rate = 100%)	LOC for MOE = 100 (occupational) LOC for MOE = 100 (residential, includes the FQPA SF)	Developmental toxicity - rat Maternal LOAEL = 30 mg/kg/day, based upon decreased body weight gain and corrected body weight gain
Intermediate-term inhalation (1–6 months)	Oral study NOAEL = 9.3 mg/kg/day (inhalation absorption rate = 100%)	LOC for MOE = 100 (occupational) LOC for MOE = 100 (residential, includes the FQPA SF)	Subchronic neurotoxicity - rat LOAEL = 63.3 mg/kg/day, based upon decreased body weight gain
Long-term inhalation (> 6 months)	Oral study NOAEL = 5.7 mg/kg/day (inhalation absorption rate = 100%)	LOC for MOE = 100 (occupational) LOC for MOE = 100 (residential, includes the FQPA SF)	Combined chronic toxic/carcinogenicity - rat LOAEL = 16.9 mg/kg/day, based upon increased incidence of mineralized particles in thyroid colloid in males
Cancer (oral, dermal, inhalation)	No evidence of carcinogenicity for humans	Not applicable	No evidence of carcinogenicity in rats and mice

* The reference to the FQPA SF refers to any additional SF retained due to concerns unique to the FQPA.

In its objections to a separate imidacloprid tolerance action, NRDC claims that EPA erred by regulating on the basis of a LOAEL for acute and chronic toxicity. As can be seen from the above table, NRDC is mistaken with regard to use of a LOAEL for estimating the RfD for chronic risk. The acute toxicity endpoint was based upon a LOAEL of 42 mg/kg/day from an acute neurotoxicity study in rats. This value was adjusted with a safety factor of 3X to approximate the value of a NOAEL. EPA has high confidence that this value of 3X is sufficient for several reasons. The effect seen at the LOAEL in the acute neurotoxicity study (decreased motor activity), occurred only in one sex of the rat (females), was characterized as minimal, and may have been a result of the use of the gavage dosing in the study. The decreased motor activity was not replicated following repeated dietary administration (non-gavage) at lower and higher doses (10, 70 or 200 mg/kg/day) in the subchronic neurotoxicity study in the same species (rats). Further, using a safety factor of 3X produces a regulatory endpoint lower than the acute effect levels in

other standard studies for determining an acute endpoint, developmental toxicity studies in two species, and in another study that is on occasion used for such a purpose, the developmental neurotoxicity study in rats. Also in these objections, NRDC claims that EPA failed to calculate residential risks for some scenarios, based on low toxicity (no endpoints were chosen). On October 8, 2002, the Health Effects Division (HED), Hazard Identification Assessment Review Committee (HIARC) reviewed the hazard data base for imidacloprid and established additional endpoints. Endpoints were chosen for each of the following exposure scenarios: Acute dietary, chronic dietary, short-term oral, intermediate-term oral, short-term dermal, intermediate-term dermal, long-term dermal, short-term inhalation, intermediate-term inhalation, and long-term inhalation. In the current risk assessment (Unit II.E. of this document), EPA calculated short-term residential risks (oral, dermal, and inhalation) for both adults and children for a wide-range of representative scenarios, including applications to lawns,

ornamental plantings, indoor and outdoor potted plants, and dogs and cats. Based on current residential use patterns for imidacloprid, EPA expects the duration of exposure to be short-term (1–30 days), and would not result in intermediate-term or long-term exposure. EPA also conducted human health aggregate risk assessments for the following exposure scenarios: Acute aggregate (food + drinking water), short-term aggregate exposure (food + drinking water + residential), and chronic aggregate exposure (food + drinking water).

B. Exposure Assessment

1. *Dietary exposure from food and feed uses.* Tolerances have been established (40 CFR 180.472) for the combined residues of imidacloprid, in or on a variety of raw agricultural commodities. Meat, milk, poultry, and egg tolerances have also been established for the combined residues of imidacloprid. In conducting dietary exposure assessments, EPA used the Dietary Exposure Evaluation Model software with the Food Commodity Intake Database (DEEM™-FCID) which