

(SF) is retained due to concerns unique to the FQPA, this additional factor is applied to the RfD by dividing the RfD by such additional factor. The acute or chronic Population Adjusted Dose (aPAD or cPAD) is a modification of the RfD to accommodate this type of FQPA SF.

For non-dietary risk assessments (other than cancer) the UF is used to determine the LOC. For example, when 100 is the appropriate UF (10X to account for interspecies differences and 10X for intraspecies differences) the LOC is 100. To estimate risk, a ratio of the NOAEL to exposures (margin of

exposure (MOE) = NOAEL/exposure) is calculated and compared to the LOC.

The linear default risk methodology (Q*) is the primary method currently used by the Agency to quantify carcinogenic risk. The Q* approach assumes that any amount of exposure will lead to some degree of cancer risk. A Q* is calculated and used to estimate risk which represents a probability of occurrence of additional cancer cases (e.g., risk is expressed as 1×10^{-6} or one in a million). Under certain specific circumstances, MOE calculations will be used for the carcinogenic risk assessment. In this non-linear approach,

a "point of departure" is identified below which carcinogenic effects are not expected. The point of departure is typically a NOAEL based on an endpoint related to cancer effects though it may be a different value derived from the dose response curve. To estimate risk, a ratio of the point of departure to exposure ($\text{MOE}_{\text{cancer}} = \text{point of departure/exposures}$) is calculated. A summary of the toxicological endpoints for bifentazate used for human risk assessment is shown in Table 2 of this unit:

TABLE 2.—SUMMARY OF TOXICOLOGICAL DOSE AND ENDPOINTS FOR BIFENTAZATE FOR USE IN HUMAN RISK ASSESSMENT

Exposure Scenario	Dose Used in Risk Assessment, UF	Special FQPA SF and Level of Concern for Risk Assessment	Study and Toxicological Effects
Acute Dietary; general population and females 13–50 years old	NA	NA	An acute dietary endpoint was not selected based on the absence of an appropriate endpoint attributed to a single dose.
Chronic Dietary; all populations	NOAEL= 1.0 mg/kg/day UF = 100 cRfD = 0.01 mg/kg/day	Special FQPA SF = 1X cPAD = 0.01 mg/kg/day	LOAEL = 8.9/10.4 mg/kg/day [M/F] based on changes in hematological and clinical chemistry parameters, and histopathology in bone marrow, liver, and kidney in the One Year Dog Feeding Study
Incidental Oral, Short Term (1–30 days)	oral NOAEL = 10 mg/kg/day	LOC for MOE \leq 100 (residential)	Maternal LOAEL = 100 mg/kg/day based on clinical signs, decreased body weight and food consumption during the dosing period in the Rat Developmental Study
Incidental Oral, Intermediate Term (30 days–6 months)	oral NOAEL = 0.9 mg/kg/day	LOC for MOE \leq 100 (residential)	LOAEL = 10.4/10.7 mg/kg/day [M/F] based on changes in hematologic parameters in the 90-Day Subchronic Dog Study
Short-, Intermediate- and Long-Term Dermal (1–30 days, 30 days–6 months, and six months to lifetime)	dermal NOAEL= 80 mg/kg/day	LOC for MOE \leq 100 (residential)	LOAEL = 400 mg/kg/day based on decreased body weight and food consumption, hematologic effects, increased spleen weight and extramedullary hemopoiesis in the spleen in the 21-Day Dermal Toxicity Study in Rats
Short-Term Inhalation (1–30 days)	oral NOAEL= 10 mg/kg/day inhalation absorption rate = 100%	LOC for MOE \leq 100 (residential)	LOAEL = 100 mg/kg/day based on decreased body weight and food consumption in the Rat Developmental Study
Intermediate-Term Inhalation (30 days–6 months)	oral NOAEL= 0.9 mg/kg/day inhalation absorption rate = 100%	LOC for MOE \leq 100 (residential)	LOAEL = 10.4/10.7 mg/kg/day based on changes in hematologic parameters in the 90-Day Dog Feeding Study
Long-Term Inhalation six months–lifetime)	Oral study NOAEL= 1.0 mg/kg/day (inhalation absorption rate = 100%)	LOC for MOE \leq 100 (residential)	LOAEL = 8.9/10.4 mg/kg/day [M/F] based on changes in hematological and clinical chemistry parameters, and histopathology in bone marrow, liver, and kidney in the One Year Dog Feeding Study
Cancer (oral, dermal, inhalation)	NA	NA	Bifentazate is classified as not likely to be a human carcinogen

C. Exposure Assessment

1. *Dietary exposure from food and feed uses.* Tolerances have been established (40 CFR 180.572) for the combined residues of bifentazate, and D3598 expressed as bifentazate (diazinecarboxylic acid, 2-(4-methoxy-

1,1'-biphenyl]-3-yl), 1-methylethylester), in or on a variety of food commodities.

Risk assessments were conducted by EPA to assess dietary exposures from bifentazate in food as follows:

i. *Acute exposure.* An acute dietary reference dose (RfD) for the females 13–

50 years of age and the general population, including infants and children, was not selected because an acute oral endpoint attributed to a single-dose exposure could not be identified in any of the studies in the toxicology data base, including