

annual cancer cases within a population is the total number of excess lifetime cancer cases divided by a 70-year lifetime.

EPA used this procedure to derive the results of the revised risk assessment. Specifically, in the exposure assessment EPA estimated the HEI's dose of each 29 dioxin-like congeners detected in sewage sludge. The dose of each congener was converted to a TEQ dose by multiplying the congener's dose by the congener's toxicity equivalency factor (TEF). The TEQ doses for each of the 29 congeners were then summed to yield an overall TEQ dose to the individual for each exposure pathway (e.g., inhalation, ingestion). Finally, the TEQ dose was multiplied by the cancer slope factor (Q1\*) to estimate the excess lifetime cancer risk to the individual for each pathway of exposure. EPA estimated total lifetime average daily dioxins exposure and excess lifetime risk to the HEI by summing lifetime average daily dioxins exposures and excess lifetime cancer risks across all of the exposure pathways relevant to each modeled individual (adult, child, infant).

Many commenters questioned EPA's use of the 1985 guidance Q1\* rather than the slope factor presented for TCDD in the September 2000 Draft Dioxin Reassessment (USEPA, 2000). They argued that it made no sense to assess cancer risk based on the 1985 cancer slope factor when EPA itself had developed an alternate value. Another commenter said that given the uncertainties in the assessment of the carcinogenicity of dioxin and dioxin-like compounds, quantifying a cancer slope factor and adopting a linear extrapolation model only magnified the uncertainty. EPA conducted its risk assessment utilizing the cancer slope factor from the 1985 guidance. Because of the terms of the Consent Decree, in the NODA we also evaluated the cancer risks to the modeled population by considering the cancer slope factor for dioxins in the Draft Dioxin Reassessment (USEPA, 2000). EPA's final decision not to regulate dioxins in land applied sewage sludge is in harmony with either cancer slope. One commenter believed EPA's existing slope factor was outdated and that the ongoing dioxin reassessment, or perhaps the Great Lakes Initiative (GLI) cancer slope factor (USEPA, 1995) reflected more current science.

EPA believes that use of the 1985 guidance Q1\* is reasonable. While alternative cancer slope factor calculations have been under review, there remains sufficient uncertainty as to whether a different Q1\* should be

used for assessing cancer risk from dioxins exposure and what the new Q1\* should be. EPA reevaluated the 1985 cancer slope in 1990 in the GLI (USEPA, 1995), by examining the pathological data from the study upon which the cancer slope factor was derived. From this reevaluation, both new tumor incidences and a new scaling factor were employed to produce a new cancer slope factor. The GLI cancer slope is approximately one half the value of the 1985 cancer slope factor. The GLI cancer slope factor was used to establish water quality standards for those water bodies. The Agency never officially adopted the GLI cancer slope factor in its risk assessments for other programs because by 1995 the Dioxin Reassessment was underway and additional science on the carcinogenic mechanism for 2,3,7,8-TCDD was evolving. In addition, the difference between the cancer risk estimate using the 1985 guidance Q1\* and other proposals (e.g., the GLI, alternate Q1\* used in the NODA) would not lead EPA to reach a different conclusion with respect to whether the predicted adverse health effects (cancer) from dioxins in land-applied sewage sludge requires EPA to regulate dioxins in land-applied sewage sludge. A more detailed discussion of the cancer slope factor is provided in section VII ("Discussion of Scientific Information Presented in the NODA").

4. *Method of Calculating Risk to the Modeled HEI Population:* As explained previously, using the results of all samples from the EPA 2001 dioxin update survey, EPA modeled all 29 dioxin and dioxin-like congeners individually, and then summed the results for all congeners to arrive at the risk for dioxins expressed as TEQ. EPA estimated excess lifetime cancer risks and corresponding average lifetime daily exposure to dioxins for a highly exposed farm adult and child (see section V.D. for a discussion of the EPA 2001 dioxins update survey).

As described in the NODA, the revised risk includes an analysis of exposures to individuals using 3,000 iterations of the Monte Carlo analysis. Individuals were subdivided into two exposure scenarios, those whose exposures begin during childhood and those whose exposures begin in adulthood. To account for the fact that children's intake rates vary with age, the analysis used separate sets of exposure parameters for four age cohorts: ages 1–5, ages 6–11, ages 12–19, and ages 20–70. To capture the higher intake-rate-to-body weight ratio of children, a start age between the ages of 1 and 6 was randomly selected for all children for

each iteration in the probabilistic analysis.

Children (defined as between one year and six years of age) are an important sensitive population in risk assessment because they may be more highly exposed than adults. This age range was selected because this represents the highest consumption rate (intake/body weight) for most of the exposure pathways evaluated in this risk assessment. Compared to adults, children may eat more food and drink more fluids per unit of body weight. This higher intake-rate-to-body weight ratio can result in a higher average daily dioxins dose per body weight for children as compared to adults. The estimated excess lifetime cancer risk for individuals whose exposure begins in childhood is less than or equal to the estimated excess lifetime cancer risk for adults whose exposure begins later in life. The reason for this is that children's mobility generally is greater than that of adults. That is, overall, the period of time that a child will occupy a given residence is shorter than the period of time an adult will occupy a given residence. Therefore, individuals whose exposures to dioxins from land-applied sewage sludge in home-produced foods begins in childhood are, in general, assumed to be exposed for a shorter duration than those whose exposure begins in adulthood (USEPA, 2003a).

Infants are also an important sensitive population considered in the revised risk assessment. Infants may be exposed to dioxin-like compounds via the ingestion of breast milk. The characterization of risks to infants of farmers and home gardeners was considered separately from the characterization of risks to older children (i.e., aged 1 year or older). While risks to children and adults were integrated to assess individuals for whom exposure first occurs during childhood but continues into adulthood, the lifetime risks to infants were calculated separately from the risks to older children (i.e., ages 1 year or older) and adults. For infants, exposure during the first year of life was averaged over an expected lifetime of seventy years to derive a LADD that was then used to calculate risk. The "lifetime" risk to infants thus should be thought of as the contribution to an individual's lifetime risk that is due to ingestion of breast milk from a mother exposed to dioxins in home-produced foods derived from land-applied sewage sludge.

Table 1 below provides percentiles of the distribution of estimated excess lifetime cancer risk to a farm family adult and child who consume home-