14.0 Human Risk Module

14.1 Purpose and Scope

The Human Risk Module calculates risk measures for a given constituent, waste concentration, and site. Detailed information on the Human Risk Module can be found in the background document (U.S. EPA, 2000). Figure 14-1 shows the relationship and information flow between the Human Risk Module and the 3MRA modeling system. The Human Risk Module uses the annual average daily doses calculated in the Human Exposure Module to calculate receptor risk statistics. These risk statistics are used by the Exit Level Processors to determine national-level risk distributions.

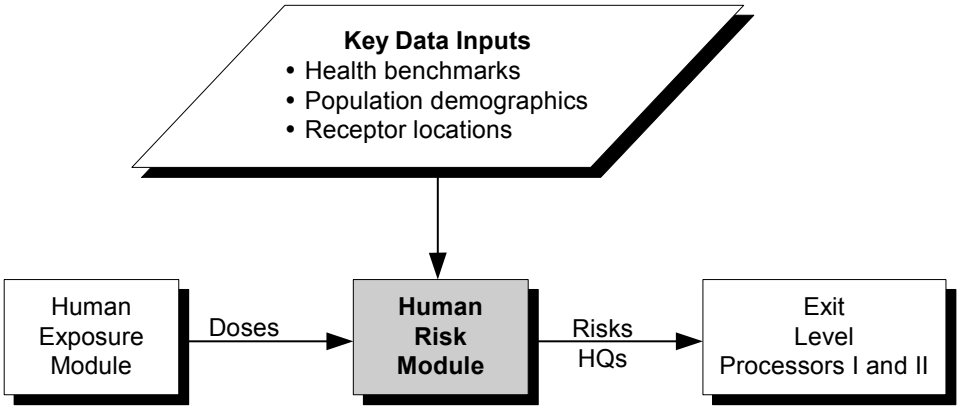

Figure 14-1. Information flow for the Human Risk Module in the 3MRA modeling system.

For each constituent, waste concentration, and site, the Human Risk Module generates risk estimates for each receptor location in the area of interest (AOI) and then calculates the number of receptors that fall within a specified risk or hazard range to describe the distribution of risks for the population at each site. The module also determines the timing of maximum risks. The Human Risk Module has the following functions:

1. **Calculates risk measures.** The Human Risk Module calculates cancer risk, noncancer hazard quotient (HQ), and noncancer margin of exposure (MOE) (for breastfeeding infants only). Depending on the constituent, the Human Risk Module calculates risk, HQ, or both. MOE is only calculated for breastfeeding infants for dioxin-like chemicals. These calculated risk measures are specific to a receptor type, an age cohort, an exposure pathway, a receptor location, and a specific exposure period (identified by starting year). The Human Risk Module

also aggregates risks and HQs from individual exposure pathways (e.g., ground water ingestion) to determine risk for groups of pathways (e.g., all ingestion pathways).

2. **Processes results for decision making.** The Human Risk Module puts exposed and unexposed population in the AOI into risk bins to estimate the number of receptors that experience risk within a specified range. Each risk bin is a range of risks or HQs. For any given exposure pathway and exposure period, the Human Risk Module uses Census data on population for each receptor location to determine the number of people of each receptor type and age cohort that experience risk from the specified pathway in the specified exposure period at risk levels within that bin. These populations are summed across receptor locations that have risks within the same bin. For each exposure pathway or pathway group, the Human Risk Module estimates total risk by multiplying the population at a location by the risk for that location, and uses this to determine the exposure period for which the total risk or HQ across all receptor types and age cohorts is the greatest. This estimate of total risk is not intended as a final risk measure, but is used only to identify the timing of maximum risk. The exposure period is identified by the year in which the risk averaged over a specified exposure period starts.

The scope of the Human Risk Module includes nine exposure pathways, four exposure pathway groupings, four receptor types, and five age cohorts, as follows:

Exposure pathways:

- Air inhalation,
- Shower inhalation,
- Ground water ingestion,
- Soil ingestion,
- Fruit and vegetable ingestion,
- Beef ingestion,
- Dairy ingestion,
- Fish ingestion, and
- Breast milk ingestion (infants only).

Exposure pathway groupings:

- All inhalation pathways,
- All ingestion pathways,
- All ground water pathways (ground water ingestion and shower inhalation), and
- All ingestion and inhalation pathways combined (if appropriate for the constituent).

Receptor types:

- Residents,
- Gardeners,
- Farmers, and
- Fishers.

Age cohorts:

- Infants under 1 year (breastfeeding pathway only)
- Children aged 1 to 5 years,
- Children aged 6 to 11 years,
- Children aged 12 to 19 years, and
- Adults.

The module uses the above groupings in calculations, but risk results are output only for the different exposure pathways and pathway groupings; these results are summarized across receptor types and age cohorts.

14.2 Conceptual Approach

The Human Risk Module calculates risk or HQ by pathway, receptor type, and age cohort; aggregates pathway risks across pathway groupings; bins population into risk or HQ bins; estimates total population risk; and determines the timing of maximum risks. The Human Risk Module can calculate risks and HQs for the whole AOI or within radial distance rings within the AOI if they are defined. These functions are described in the following subsections.

14.2.1 Calculate Risk Measures

The first major function of the Human Risk Module is to calculate cancer risk and/or HQ for a given receptor type, age cohort, exposure pathway, receptor location, and exposure period.

Cancer Risk Calculations. The governing equation used to calculate increased incremental cancer risk over a lifetime attributable to a lifetime exposure to a contaminant at a given dose (exposure) is

$$Risk = CSF \times Dose \qquad (14\text{-}1)$$

where

Risk = lifetime risk (probability units)
CSF = contaminant-specific cancer slope factor $(mg/kg\text{-}d)^{-1}$
Dose = annual average daily contaminant dose (mg/kg-d), expressed as an average daily dose over a lifetime.

This equation reflects a daily, lifetime exposure. However, the exposure duration may vary, as may the number of days per year a receptor is actually exposed (exposure frequency).

Assuming that cancer risk is linearly related to exposure duration and exposure frequency, Equation 14-1 can be modified to account for this as follows:

$$Risk = \frac{ExpDur}{LifeTime} \times \frac{ExpFreq}{365} \times CSF \times ADD \qquad (14\text{-}2)$$

where

ExpDur	=	duration of exposure (yr)
Lifetime	=	average lifetime (yr)
ExpFreq	=	exposure frequency (d/yr)
CSF	=	cancer slope factor $(mg/kg\text{-}d)^{-1}$
ADD	=	average daily dose over specified exposure period (mg/kg-d).

Exposure duration and exposure frequency may be set to any reasonable value so long as exposure duration is less than or equal to the lifetime value chosen and exposure frequency is less than or equal to the maximum of 365 d/yr. The exposure duration is currently set to 9 years, which corresponds to the recommended value for median residence time presented in the *Exposure Factors Handbook* (U.S. EPA, 1997a,b,c), and to set exposure frequency to 350 d/yr.

The ADD is calculated as follows:

$$ADD = \frac{\sum_{t=1}^{ExpDur} Dose_t}{N} \qquad (14\text{-}3)$$

where

ADD	=	average daily dose over entire exposure period (mg/kg-d).
t	=	year
ExpDur	=	exposure duration (yr)
$Dose_t$	=	annual average daily dose in year *t* (mg/kg-d)
N	=	number of years in the exposure duration (unitless).

In addition to the straight averaging of dose over the exposure period, a child receptor will age out of his or her initial cohort as the exposure duration progresses. For example, a child beginning the exposure duration at age 3 will, over a 9-year exposure period, increase in age to 11 years old. The annual doses are based on exposure factors (such as body weight or intake rate) that are age-cohort-specific. Therefore, the Human Risk Module ages each child receptor through the appropriate age cohorts as exposure progresses. Child receptors for a specific starting cohort are assumed to begin exposure at the midpoint of the age cohort (e.g., 3 years for a child aged 1 to 5 years). The age of a child receptor is monitored at each year over the exposure duration, and the dose is set in accordance with the relevant cohort as the child ages through them.

The Human Risk Module calculates a time series of risks averaged over the specified exposure duration, with each average risk representing a different starting year within the period of calculation. Calculations continue until the exposure medium concentration drops to less than 1 percent of the peak concentration, up to a maximum of 10,000 years. If the exposure duration were 9 years and the concentration dropped to 1 percent in year 100, the Human Risk Module would calculate a time series of 92 annual average risks, for years 1 through 9, 2 through 10, and so forth, up to years 92 through 100. These averages are identified by starting year (so in this example, year 1, year 2, and so forth, through year 92), but the risk for "year 1" is the risk for the 9-year period starting in year 1.

The Human Risk Module calculates cancer risks for any given contaminant only if an appropriate health benchmark is available. For ingestion pathways, the calculations are performed if an oral cancer slope factor (CSF) is available; for inhalation pathways, the calculations are performed if an inhalation CSF is available. If neither an inhalation nor an ingestion CSF is available, then no cancer risks are calculated for the contaminant.

Noncancer HQ Calculations. The governing equation used to calculate noncancer HQs depends on the route of exposure. For ingestion exposures, the equation is

$$HQ = \frac{ADD}{RfD} \tag{14-4}$$

where

HQ = hazard quotient for exposure period (unitless)
ADD = average daily dose over exposure period (mg/kg-d)
RfD = reference dose (mg/kg-d).

The ADD is calculated as shown in Equation 14-3 for carcinogens.

For inhalation exposures, the equation is

$$HQ = \frac{C_{avg}}{RfC} \tag{14-5}$$

where

HQ = hazard quotient over exposure period (unitless)
C_{avg} = average air concentration over exposure period (mg/m^3)
RfC = reference air concentration (mg/m^3).

The average air concentration may be an ambient air concentration or a shower/bathroom air concentration, depending on the pathway, and is calculated as

$$C_{avg} = \frac{\sum_{t}^{t+ExpDur-1} \times Conc_t}{ExpDur} \quad (14\text{-}6)$$

where

C_{avg} = average air concentration over exposure period (mg/m^3)
t = first year of exposure period
ExpDur = exposure duration (yr)
$Conc_t$ = annual average air concentration in year *t* (mg/m^3).

HQs are calculated for a specified exposure duration. They do not vary with exposure frequency, but are based on the assumption that the receptor has regular, chronic exposure. Exposure duration may be set to any reasonable value less than or equal to a typical lifetime. The exposure duration is currently set to 9 years, which corresponds to the recommended value for median residence time presented in the *Exposure Factors Handbook* (U.S. EPA, 1997a,b,c).

As for risk, the Human Risk Module calculates a time series of HQs averaged over the specified exposure duration, with each HQ representing a different starting year within the period of calculation. Calculations continue until the exposure medium concentration drops to less than 1 percent of the original WMU concentration, up to a maximum of 10,000 years.

The Human Risk Module calculates noncancer HQs for any given contaminant only if an appropriate health benchmark is available. For ingestion pathways, the calculations are performed if an oral reference dose (RfD) is available; for inhalation pathways, the calculations are performed if an inhalation reference concentration (RfC) is available. If neither an RfD nor an RfC is available, then no noncancer HQs are calculated for the contaminant.

Noncancer MOE Calculations. For infant exposure to breast milk, effects are typically quantified as an MOE rather than a risk or HQ. The MOE for infants is calculated as

$$MOE = \frac{Dose}{BM} \quad (14\text{-}7)$$

where

MOE = margin of exposure (unitless)
Dose = annual average applied dose from breast milk ingestion (mg/kg-d)
BM = contaminant-specific benchmark for breast milk exposure based on background exposure levels (mg/kg-d)

This calculation is currently performed only for 2,3,7,8-TCDD for infant exposures through the breast milk pathway. MOE estimates are single-year averages based on 1-year average doses. The Human Risk Module calculates an average MOE for each year in the calculation period.

14.2.2 Process Results for Decision Making

The Human Risk Module generates risk estimates for a variety of receptor types, age cohorts, exposure pathways, and receptor locations for each year modeled. The module processes and aggregates these results so that decision makers can answer a variety of questions about potential human risks. Specifically, the Human Risk Module (1) aggregates risk measures across pathways, (2) places population into risk bins, and (3) determines the timing of the maximum risk or hazard.

Aggregate Risk across Pathways. The 3MRA modeling system calculates risks and HQs for individual pathways. However, many receptors are exposed via multiple pathways, so the 3MRA modeling system also aggregates risk across pathways, where appropriate. The Human Risk Module performs the following aggregations:

- **Aggregation across ingestion pathways.** The Human Risk Module sums risks and HQs across the ingestion pathways for ground water, soil, fruits and vegetables, beef, milk, and fish.

- **Aggregation across inhalation pathways.** The Human Risk Module sums risks and HQs across both inhalation pathways: ambient air and shower.

- **Aggregation across all pathways.** When health effects can be combined across routes of exposure, the Human Risk Module aggregates risks for all pathways (both ingestion and inhalation).

- **Aggregation across ground water pathways.** When health effects can be combined across routes of exposure, the Human Risk Module aggregates risks for both ground water pathways: ground water ingestion and shower inhalation.

Whether or not it is appropriate to aggregate risks or HQs across routes of exposure depends on the contaminant and the health effects it causes via different routes of exposure. Cancer and noncancer effects (risks and HQs) are never combined.

Place Population into Risk Bins. Calculated risks or HQs by pathway are a measure of possible individual risk. The second major function of the Human Risk Module is to calculate population risk measures. For each pathway or aggregation of pathways, the Human Risk Module calculates the number of people in each receptor group and age cohort that experience various risk or HQ levels across the AOI or in a distance ring. These population counts are the basis of a series of cumulative frequency histograms, each specific to a receptor type, age cohort, exposure pathway (or aggregation of pathways), and exposure period. Thus, the full collection of cumulative histograms is a time series of conditional histograms for risk and/or HQ. Although the time series of histograms appears to be annual, the histograms are actually based on a multiyear moving average for risks and HQs, as discussed in the section on calculating risk. The length of the averaging time depends on the specified exposure duration.

The 3MRA modeling system currently uses 1990 Census data, 1992 agricultural census data, and 1996 fishing survey data to estimate receptor/age-cohort-specific population estimates

at each residential or farm location for each site, as described in Volume II of this report. These populations are associated with census blocks. For the purpose of calculating and assigning carcinogenic risk and noncarcinogenic hazard, all of the population in a Census block is assumed to be located at the centroid of the block.

The 3MRA modeling system has seven bins for risk and four bins for HQ and MOE. The bins for risk are

- **Bin 1:** 0 to 5×10^{-9}
- **Bin 2:** $>5 \times 10^{-9}$ to 7.5×10^{-8}
- **Bin 3:** $>7.5 \times 10^{-8}$ to 7.5×10^{-7}
- **Bin 4:** $>7.5 \times 10^{-7}$ to 2.5×10^{-6}
- **Bin 5:** $>2.5 \times 10^{-6}$ to 7.5×10^{-6}
- **Bin 6:** $>7.5 \times 10^{-6}$ to 5×10^{-5}
- **Bin 7:** $>5 \times 10^{-5}$.

The HQ/MOE bins are

- **Bin 1:** 0 to 0.05
- **Bin 2:** > 0.05 to 0.5
- **Bin 3:** > 0.5 to 5.0
- **Bin 4:** > 5.0.

All bin ranges are inclusive of the upper bound.

For any exposure period, and given a receptor type, age cohort, and pathway (or pathway aggregation), a cumulative risk histogram is generated that contains the total population corresponding to the receptor/cohort combination, across all receptor locations in the AOI that experiences risks falling within each risk bin range. An HQ cumulative histogram is constructed in a similar fashion.

As an example, assume 150 adult residents reside collectively at all residential receptor locations within an AOI. Assume that for a given contaminant and exposure pathway for a specified exposure period starting in year 1, they fall into the bins as shown in Table 14-1. The conditional, cumulative population for these two example exposure periods would be as shown in Table 14-2.

Table 14-1. Example HQ Counts for Hypothetical Sites

	Population in Each HQ Range			
	Bin 1	**Bin 2**	**Bin 3**	**Bin 4**
Exposure starting in...	**<0.05**	**0.05 to 0.5**	**0.5 to 5**	**>5**
Year 1	30	45	75	0
Year 2	15	45	60	30
Year 3	6	24	60	60

Table 14-2. Example Cumulative Frequency at Hypothetical Site

	Percent of Population in this HQ Range or Lower			
Exposure starting in...	**Bin 1**	**Bin 2**	**Bin 3**	**Bin 4**
Year 1	20	50	100	
Year 2	10	40	80	100
Year 3	4	20	60	100

Determine Timing of Maximum Risk. The third major function of the Human Risk Module is to determine the timing of maximum risk and/or HQ across all receptor/cohort individuals for a given exposure pathway or aggregation of pathways. The time series of risk histograms is analyzed to determine that year in which the maximum total risk and/or HQ over time occurs.

Specifically, the Human Risk Module first estimates the total risk/HQ for all individuals at a site by multiplying the population at each receptor location by the calculated risk or HQ at that location. This is then summed across all locations, receptor types, and age cohorts. The Human Risk Module calculates this total risk for each exposure period in the time series. The module then determines the exposure period for which this total risk is the highest.

14.3 Module Discussion

14.3.1 Strengths and Advantages

The major strengths and advantages of this module include the following:

- **Provides coverage for key receptor populations and exposure pathways**. The Human Risk Module supports risk characterization for four types of resident receptors and four types of farmer receptors. Together, this set of receptor populations includes the majority of those typically considered in evaluating multipathway exposure and risk. Further, in modeling each of these receptor populations, the 3MRA modeling system provides coverage for key exposure pathways related to receptor behavior (e.g., ambient air inhalation, crop ingestion). To enhance the representativeness for risk estimates, the modeling of specific exposure pathways is linked to known receptor activity characterized using demographic (or other relevant) data. The 3MRA modeling system also includes five age cohorts in modeling risk for each receptor population to reflect age-specific differences in exposure and risk. Specifically, cohort aging is considered in modeling risk such that the ingestion or inhalation rates used for a given receptor are adjusted as that receptor "ages" into the next age cohort to reflect the exposure parameters relevant for that older age group.

- **GIS-based representative modeling of population-level risk.** The Human Risk Module uses a GIS-based framework to support spatially representative modeling of risk for modeled receptors. The use of U.S. Census, Census of Agriculture and GIRAS land use data to place residents, drinking water wells, farmers, farms, and recreational fishers for modeling risk supports a more representative analysis of population-level exposure which explicitly considers the spatial distribution of these receptors across AOIs.

- **Generation of time series of risk profiles for modeled receptor populations provides coverage for contaminants with different temporal exposure profiles.** The Human Risk Module generates a time series of risk estimates for each combination of receptor/pathway/location/simulation year/chemical-endpoint that allows the temporal profile of risk over longer simulation periods (up to 10,000 years) to be evaluated. Providing detailed tracking of temporal risk profiles over longer modeling periods can be important in assessing multipathway risk for pathways involving contaminants with widely varying fate and transport profiles (e.g., an inhalation toxicant that produces risk shortly after WMU release, versus a contaminant with low mobility that can take many years to reach an off-site well).

- **Use of cumulative risk histograms provides a ready means for identifying the year of maximum risk given widely varying risk profiles for different receptor/pathway/contaminant combinations.** The Human Risk Module produces cumulative risk histograms to characterize the distribution of risk across specific receptor populations for a given exposure pathway/simulation year/contaminant combination. These cumulative risk histograms can be used as the basis for identifying the simulation year which has the maximum cumulative risk for a given regulatory percentile of the population (e.g., the maximum cumulative risk for the 95^{th} percentile of beef farmers resulting from beef ingestion). The 3MRA modeling system includes an automated procedure for querying the entire time series of cumulative risk distributions to identify this maximum risk year and then outputs the risk distributions for all exposure pathways modeled for that receptor population for that year. Not only does this procedure provide a ready means to identify the significant maximum risk year and extract the full risk distributions generated for that year, this approach also represents an effective means of data reduction, which is a key issue given the large number of simulation years and receptor/pathway/location/contaminant combinations that can be modeled. Cumulative risk distributions can be generated for user-specified distance rings within the AOI. This provides a distance-differentiated set of risk metrics that can be used to support decision making.

14.3.2 Uncertainty and Limitations

The following limitations or uncertainties are inherent in the Human Risk Module:

- **Risk/HQ/MOE estimates are aggregated for certain receptor types.** The four receptor types considered by the Human Risk Module (resident, residential

gardener, farmer, fisher) are fewer than the number of receptor types simulated by the Human Exposure Module in order to maintain output storage requirements at reasonable levels. The Human Risk Module internally aggregates dairy farmers and beef farmers into a single farmer (dairy and beef) and aggregates all of the Human Exposure Module's receptor type-specific fishers (e.g., resident fisher) into a single fisher receptor. Some resolution is lost by this aggregation; for example, the risks specific to farmers who drink contaminated milk, but do not consume contaminated beef, would not be available.

- **Synergistic or antagonistic effects among multiple contaminants or individual contaminant species on risk/HQ/MOE are not considered.** The Human Risk Module considers only one contaminant at a time, and the risk associated with that contaminant is implicitly considered to be independent of the risks posed by other contaminants.

- **Cancer slope factors do not vary with cohort age.** Age-specific differences in exposure responses are not considered.

- **The fraction of residents in a Census block that ingest ground water is assumed to equal the fraction of residents in the Census block group that have ground water wells.** The Census data report the number of households within a Census block group that are served by ground water wells. However, this information is available only at the Census block group level, and it is not possible to determine from Census data alone whether individual Census blocks within a block group with wells have wells or not. The Human Risk Module calculates at the Census block level when considering residential exposure areas. Consequently, the actual fraction of residents on wells for any individual residential exposure area is uncertain. The assumption is made that the fraction of residents in an exposure area (a Census block) consuming ground water is equal to the fraction of the population in the Census block group that are served by ground water wells. That is not to say that all well water is contaminated. Only those wells lying within the ground water plume from the WMU source are potentially contaminated. To the extent that the fraction of residents on wells differs among Census blocks within the plume and outside the plume, population risks may be over- or underestimated.

14.4 References

U.S. EPA (Environmental Protection Agency). 1997a. *Exposure Factors Handbook. Volume I-General Factors*. EPA/600/P-95/002Fa. Office of Research and Development, Washington, DC. Website at http://www.epa.gov/mcea/exposfac.htm. August.

U.S. EPA (Environmental Protection Agency). 1997b. *Exposure Factors Handbook. Volume II-Food Ingestion Factors*. EPA/600/P-95/002Fa. Office of Research and Development, Washington, DC. Website at http://www.epa.gov/ncea/exposfac.htm. August.

U.S. EPA (Environmental Protection Agency). 1997c. *Exposure Factors Handbook. Volume III-Activity Factors*. EPA/600/P-95/002Fa. Office of Research and Development, Washington, DC. Website at http://www.epa.gov/ncea/exposfac.htm. August.

U.S. EPA (Environmental Protection Agency). 2000. Background Document for the Human Exposure and Human Risk Modules for the Multimedia, Multipathway, Multireceptor Risk Assessment (3MRA) Model for HWIR99. Office of Solid Waste, Washington, DC. August.