8.0 RISK CHARACTERIZATION

8.1 INTRODUCTION

Risk characterization is the final step of the risk assessment process. In this step, the toxicity information provided in Section 7.0 is combined with the calculated chemical intakes from Section 6.0 to develop estimates of individual chemical risk. Risks are presented as comparisons between reference doses (RfDs) and predicted exposures for the noncarcinogenic chemicals. Risks for carcinogenic chemicals are presented as probabilities that individuals will develop cancer over a lifetime of exposure to the chemical.

Standard equations for estimating potential noncarcinogenic and carcinogenic risks are presented in Section 8.2. The application of these standard equations to site specific exposures is presented in Sections 8.3 and 8.4. The potential risks attributable to lead exposures are discussed separately from other chemicals in Section 8.5. Section 8.6 presents an evaluation of the potential risks associated with CKD waste pile fugitive emissions relative to kiln emissions for the metals. Section 8.7 presents a summary of risks for individuals who may be exposed over one or more pathways.

8.2 ESTIMATION OF RISKS

8.2.1 METHODOLOGY FOR ESTIMATING NONCARCINOGENIC RISKS

The potential that the estimated exposures determined in Section 6.0 could result in a noncarcinogenic health effect is evaluated by comparing the chronic daily intake of each noncarcinogenic chemical with the reference dose (RfD) for that chemical. The ratio of estimated intake to the RfD is called the hazard quotient (HQ) and is calculated using the following equation:

$$\text{Hazard Quotient} = \text{CDI}/(\text{RfD})$$

Where:

CDI = chronic daily intake (determined in Section 6), mg/kg/day

RfD = Reference Dose, mg/kg/day

Evaluation of noncarcinogenic effects using the hazard quotient is based on the assumed existence of a threshold level of exposure below which toxic effects are not expected to occur. Therefore, potential toxic effects would not be expected to occur until the threshold level (i.e., RfD) is exceeded.

The overall potential for noncarcinogenic effects posed by exposure to multiple contaminants can be estimated by calculating a hazard index (HI). The HI is the sum of the individual HQs. This approach assumes that the magnitude of adverse effects from multiple exposures to noncarcinogenic chemicals is proportional to the sum of the hazard quotients for individual chemicals for any particular pathway. An HI evaluation assumes that predicted exposures to several individual chemicals (which individually may have hazard quotients less than one) could produce a toxic effect if the sum of their HQs (i.e., their HI) exceeds one.

A significant limitation to the determination of HI's is that only hazard quotients for substances producing effects at similar endpoints (e.g., the liver or kidney) should be added. Thus, the total HI for noncarcinogenic risks should be considered an initial screen of potential total impacts and care should be taken when interpreting the meaning of this value. Total HI's in excess of one should not be interpreted as indicators of a potential health effect until further evaluation of individual toxic endpoints for chemicals contributing to the HI are evaluated.

8.2.2 Methodology for Estimating Carcinogenic Risks

Carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to potential carcinogens at the site. This estimated excess lifetime cancer risk is calculated by multiplying the chronic daily intake calculated using the equations in Section 6.0 by the SF:

$$\text{Risks} = \text{CDI} \times \text{SF}$$

Where:

CDI = Chronic Daily Intake (mg/kg/day), and
SF = Slope Factor $(\text{mg/kg/day})^{-1}$

In the assessment of potential carcinogenic effects, it is assumed that any dose of a potential carcinogen presents some carcinogenic risk to humans. It is also assumed that in the case of multiple exposure to carcinogenic compounds, the potential risks are additive over both chemicals and pathways.

In evaluating the significance of potential exposures to carcinogenic compounds, the U.S. EPA typically adopts a target excess lifetime cancer risk range of 10^{-4} to 10^{-6}. In the discussion that follows, predicted carcinogenic risks are evaluated within the context of this range.

8.3 ESTIMATED NONCARCINOGENIC RISKS

This section presents the calculated noncarcinogenic risks for all exposure pathways. Risks for both typical and high end exposures are discussed for each pathway. In the tables, noncarcinogenic risks for an individual are summed across all chemicals to obtain a total HI for the exposure being modeled. As discussed in section 8.2.1, such summation is considered a preliminary screen to determine the potential for significant noncarcinogenic health effects by each exposure route. If an HI greater than 1 were determined, a more specific evaluation of the individual chemicals causing the HI to exceed 1 would be required. As seen in the following sections, a specific evaluation of individual chemicals is not necessary because the total HI for all exposure routes was well below 1.

8.3.1 INHALATION EXPOSURES

The calculated chemical specific noncarcinogenic HQs and the total HIs for the typical direct inhalation exposure scenario are presented in Table 8-1. The inhalation exposures shown on Table 8-1 represent exposures to both kiln stack emissions and LWDF fugitive emissions at the maximum exposure area. As seen on this table, the total HIs for the child and adult exposures to all chemicals by this route for the maximum exposure area were determined to be 0.0024 and 0.00059, respectively. The chemical specific HQs and the total HIs for both the adult and child are all well below 1, indicating that noncarcinogenic health effects are not expected as a result of this exposure.

8.3.2 Soil Exposures

The calculated chemical specific noncarcinogenic HQs and the total HIs for the typical soil exposure scenario is presented in Table 8-2. This table presents results for both ingestion and dermal contact exposures. As seen on this table, the total HIs for soil ingestion and dermal contact for the child and adult at the maximum exposure area were determined to be 0.0101 and 0.0011, respectively. The chemical specific HQs and the total HIs for adults and children are all well below 1, indicating that noncarcinogenic health effects are not expected as a result of either incidental ingestion or dermal absorption of soil under the typical exposure scenario.

Table 8-2 also presents the calculated chemical specific HQs and total HIs for the high end soil exposure scenario. The total HIs for the child and adult at the maximum exposure area were determined to be 0.0103 and 0.0011, respectively. As with the typical exposure scenario, the chemical specific HQs and the total HIs for the high end soil exposure scenarios are all well below 1, indicating that noncarcinogenic health effects are not expected by the soil exposure routes.

8.3.3 Drinking Water Exposures

The calculated chemical specific noncarcinogenic HQs and the total HIs for the drinking water exposure scenario are presented in Table 8-3. As seen on this table, the total HIs for the child and adult exposures to all chemicals by this route were determined to be 0.0017 and 0.00076, respectively. The chemical specific HQs and the total HIs for both the adult and child are all well below 1, indicating that noncarcinogenic health effects are not expected as a result of this exposure.

8.3.4 Surface Water Exposures

The calculated chemical specific noncarcinogenic HQs and the total HIs for the typical recreational surface water exposure scenarios are presented in Table 8-4. This table presents results for both incidental ingestion and dermal contact exposures. As seen on this table, the total HIs for incidental ingestion and dermal contact of surface water for the child and adult were determined to be 0.0002 and 0.000044 (France Park), respectively, and 0.000022 and 0.0000048 (Wabash River), respectively. For both the Wabash River and France Park, the chemical specific HQs and the total HIs for adults and children are

all well below 1, indicating that noncarcinogenic health effects are not expected as a result of either incidental ingestion or dermal absorption of soil under the typical scenario.

Table 8-4 also presents the calculated chemical specific HQs and total HIs for the high end recreational surface water exposure scenarios. The total HIs for the child and adult by the incidental ingestion and dermal contact routes of exposure were determined to be 0.00059 and 0.00012 (France Park), respectively, and 0.000065 and 0.000014 (Wabash River), respectively. As with the typical exposure scenario, the chemical specific HQs and the total HIs for the high end surface water exposure scenarios are all well below 1, indicating that noncarcinogenic health effects are not expected by the surface water exposure routes.

8.3.5 Fish Exposures

The calculated chemical specific noncarcinogenic HQs and the total HIs for the typical exposure scenario for fresh caught fish are presented in Table 8-5. As seen on this table, the total HIs for the child and adult exposures to all chemicals by this route were determined to be 1.00 and 0.569, respectively. As shown on Table 8-5, the chemical specific HQ for methyl mercury under the typical exposure scenario is at 1. The individual HQs for all other chemicals for the child and adult populations are less than 0.0015.

Table 8-5 also presents the calculated chemical specific HQs and total HIs for the high end exposure to caught fish. As seen on this table, the total HIs for the child and adult by this route of exposure were determined to be 4.8 and 6.28, respectively. As with the typical exposure scenario, the chemical specific HQ for methyl mercury causes the hazard index to exceed 1. The individual HQs for all other chemicals are less than 0.0099.

It should be noted that the model used to estimate fish exposures to mercury is highly conservative and likely overestimates the actual risks associated with exposure to mercury through fish ingestion. Specifically, the risk assessment model of exposure to mercury through fish ingestion is based on exposure to the methyl mercury form of mercury present in fish tissue. The methylation of mercury is viewed as the key step in entrance of mercury into the food chain, since; methyl mercury tends to bioaccumulate to

a greater degree in biota relative to other forms of mercury. The uptake of methyl mercury by fish is estimated by multiplying the dissolved phase water concentration of methyl mercury by a fish bioconcentration factor. The 1998 HHRAP guidance, however, recommends that the dissolved phase water concentrations for both the divalent and methyl mercury forms of mercury are summed and the resultant sum multiplied by the bioconcentration factor for methyl mercury to estimate the methyl mercury concentration in fish. However, as described in the December 1997 U.S. EPA report titled *Mercury Report to Congress Volume III: Fate and Transport of Mercury in the Environment* (U.S. EPA mercury report), available measurement data for mercury in fish tissue samples indicates that nearly 100% of the mercury found in fish tissue is found in the methylated form. For the ESSROC risk assessment, the estimated dissolved phase water concentrations for divalent and methyl mercury in the Wabash River were 9.79 E^{-8} mg/L and 2.22 E^{-9} mg/L, respectively. Therefore, for the ESSROC risk assessment, the estimated concentration of methyl mercury in water is less than 2% of the total mercury (divalent plus methyl forms) in the water body modeled in this risk assessment. Clearly, the model used to estimate the uptake of methyl mercury by fish is highly conservative and likely overestimates the actual risks associated with exposure to mercury in fish.

8.3.6 VEGETABLE CONSUMPTION

The calculated chemical specific noncarcinogenic HQs and the total HIs for the typical home grown vegetable consumption exposure scenario are presented in Table 8-6. As seen on this table, the total HIs for the child and adult exposures to all chemicals across all plant groups modeled at the maximum exposure area were determined to be 0.00157 and 0.00145, respectively. The chemical specific HQs and the total HIs for both the adult and child are all well below 1, indicating that noncarcinogenic health effects are not expected as a result of this exposure.

Table 8-6 also presents the calculated chemical specific HQs and total HIs for the high end exposure to home grown vegetables. As seen on this table, the total HIs for the child and adult exposures to all chemicals across all plant groups were determined to be 0.00495 and 0.00329, respectively. All individual HQs for both the child and adult home grown vegetable exposures were determined to be at or below 0.004. As with the typical exposure scenario, the chemical specific HQs and the total HIs for the high end home

grown vegetable exposure scenario are all well below 1, indicating that noncarcinogenic health effects are not expected through the ingestion of home grown vegetables.

8.3.7 ANIMAL PRODUCT CONSUMPTION

The calculated chemical specific noncarcinogenic HQs and the total HIs for the typical animal product consumption scenario are presented in Table 8-7. As seen on this table, the total HIs for the child and adult exposures to all chemicals across all animal product groups modeled for the maximum exposure area are, 0.0014 and 0.00059, respectively. Chemical specific HQs for total animal product consumption for the child and adult were determined to be at or below 0.0007. The chemical specific HQs and the total HIs for both the adult and child are all well below 1, indicating that noncarcinogenic health effects are not expected as a result of this exposure.

Table 8-7 also presents the calculated chemical specific HQs and total HIs for the high end exposure to home produced animal products. As seen on this table, the total HIs for the child and adult exposures to all chemicals across all animal product groups were determined to be 0.0.0056 and 0.00205, respectively. All individual HQs for both the child and adult exposures were determined to be at or below 0.003. As with the typical exposure scenario, the chemical specific HQs and the total HIs for the high end home grown vegetable exposure scenario are all well below 1, indicating that noncarcinogenic health effects are not expected through the ingestion of home produced animal products.

8.4 ESTIMATED CARCINOGENIC RISKS

This section presents the calculated carcinogenic risks for all exposure pathways evaluated. Risks for both typical and high end exposures are discussed for each pathway. In the tables presented in this section, carcinogenic risks are summed across all chemicals to obtain a total carcinogenic risk for the exposure being modeled.

8.4.1 INHALATION EXPOSURES

Table 8-8 presents the carcinogenic risks for inhalation exposures to kiln stack emissions and LWDF fugitive emissions. The calculated chemical specific and total carcinogenic risks for the direct inhalation maximum exposure area are presented in Table 8-8. As seen on this table, the total carcinogenic risks for the child and adult exposures to all

chemicals by this route were determined to be 4.71 x 10^{-8} and 6.79 x 10^{-8}, respectively. As shown on the table, for several of the volatile compounds (e.g., benzene, methylene chloride), the estimated LDWF fugitive emission air concentrations were considerably greater than the estimated air concentrations from kiln emissions. As such, the inhalation risks for these compounds were driven by the estimated LWDF fugtive emissions air concentrations. As described previously, the LWDF fugitive air concentrations were derived assuming no emissions control systems. Therefore, the estimated inhalation risks shown on Table 8-8 for these compounds considerably overestimate actual inhalation risks.

8.4.2 Soil Exposures

The calculated chemical specific and total carcinogenic risks for the typical soil exposure scenario are presented in Table 8-9. This table presents results for both ingestion and dermal contact exposures. As seen on this table, the total carcinogenic risks for soil ingestion and dermal contact for the child and adult at the maximum exposure area were determined to be 3.56 x 10^{-8} and 2.09 x 10^{-8}, respectively. The highest carcinogenic risk for exposure to any individual chemical by these pathways was calculated at 1.46 x 10^{-8}.

Table 8-9 also presents the chemical specific and total carcinogenic risk for the high end direct contact soil exposure scenario. The total carcinogenic risks for the child and adult by the soil ingestion and dermal contact route of exposure were determined to be 3.74 x 10^{-8} and 2.49 x 10^{-8}, respectively. The highest carcinogenic risk for exposure to any individual chemical by these pathways was calculated to be 1.53 x 10^{-8}.

8.4.3 Drinking Water Exposures

The calculated chemical specific and total carcinogenic risks for the drinking water exposure scenario are presented in Table 8-10. As seen on this table, the total carcinogenic risks for the child and adult exposures to all chemicals by this route were determined to be 8.14 x 10^{-9} and 1.74 x 10^{-8}, respectively. The highest carcinogenic risk for exposure to any individual chemical by this pathway was calculated to be 1.02 x 10^{-8}.

8.4.4 SURFACE WATER EXPOSURES

The calculated chemical specific and total carcinogenic risks for the typical surface water exposure scenarios are presented in Table 8-11. This table presents results for both incidental ingestion and dermal contact exposures. As seen on this table, the total carcinogenic risks for surface water exposures for France Park for the child and adult were determined to be 8.4×10^{-10} and 3.33×10^{-10}, respectively. The total carcinogenic risks for exposure surface water for the Wabash River for the child and adult were determined to be 1.03×10^{-10} and 1.11×10^{-10}, respectively. The highest carcinogenic risk for exposure to any individual chemical by these pathways was calculated to be 6.65×10^{-10}.

Table 8-11 also presents the chemical specific and total carcinogenic risk for the high end surface water exposure scenarios. The total carcinogenic risks for the child and adult for surface water exposure at France Park were determined to be 2.39×10^{-9} and 9.52×10^{-10}, respectively. The total carcinogenic risks for the child and adult for surface water exposures at the Wabash River were determined to be 2.95×10^{-10} and 3.19×10^{-10}, respectively. The highest carcinogenic risk for exposure to any individual chemical by these pathways was calculated to be 1.9×10^{-9}.

8.4.5 FISH EXPOSURE

The calculated chemical specific and total carcinogenic risks for the typical exposure to caught fish are presented in Table 8-12. As seen on this table, the total carcinogenic risks for the child and adult exposures to all chemicals by this route were determined to be 2.65×10^{-9} and 7.52×10^{-9}, respectively. The highest carcinogenic risk for exposure to any individual chemical by this pathway was calculated to be 1.68×10^{-9}.

Table 8-12 also presents the chemical specific and total carcinogenic risk for the high end ingestion of caught fish exposure scenario. As seen on this table, the total carcinogenic risk for the child and adult by this route of exposure were determined to be 1.29×10^{-8} and 1.04×10^{-7}, respectively. The highest carcinogenic risk for exposure to any individual chemical by this pathway was calculated to be 2.52×10^{-8}.

8.4.6 VEGETABLE CONSUMPTION

The calculated chemical specific and total carcinogenic risks for the typical consumption of home grown vegetable scenario are presented in Table 8-13. As seen on this table, the total carcinogenic risks for the child and adult from consumption of all chemicals across all vegetable groups for the maximum exposure area were determined to be 2.53×10^{-7} and 2.99×10^{-7}, respectively. The highest carcinogenic risk for exposure to any individual chemical by this pathway was calculated to be 2.53×10^{-7}.

Table 8-13 also presents the chemical specific and total carcinogenic risk for the high end consumption of home grown vegetable scenario exposure scenario. As seen on this table, the total carcinogenic risk for the child and adult by this route of exposure were determined to be 2.55×10^{-7} and 3.73×10^{-7}, respectively. The highest carcinogenic risk for exposure to any individual chemical by this pathway was calculated to be 2.57×10^{-7}.

8.4.7 ANIMAL PRODUCT CONSUMPTION

The calculated chemical specific and total carcinogenic risks for the typical animal product consumption scenario are presented in Table 8-14. As seen on this table, the total carcinogenic risks for the child and adult from consumption of all chemicals across all animal product groups for the maximum exposure area were determined to be 9.16×10^{-8} and 1.97×10^{-7}, respectively. The highest carcinogenic risk for exposure to any individual chemical by this pathway was calculated to be 7.98×10^{-8}.

Table 8-14 also presents the chemical specific and total carcinogenic risk for the high end consumption of home produced animal products. As seen on this table, the total carcinogenic risk for the child and adult by this route of exposure were determined to be 4.07×10^{-7} and 9.83×10^{-7}, respectively. The highest carcinogenic risk from exposure to any individual chemical by this pathway was calculated to be 3.81×10^{-7}.

8.5 RISK CHARACTERIZATION FOR LEAD

As discussed previously, U.S. EPA recommends an alternative method for evaluating the potential risks associated with exposure to lead. Specifically, U.S. EPA recommends the use of the Integrated Exposure Uptake/Biokinetic (IEUBK) model for evaluating potential exposures to this chemical. The IEUBK model, developed by U.S EPA's Office of Air

Quality and Planning, predicts a child's blood lead level based on concurrent exposures to lead in air, soil, drinking water, and food. A child is considered to be the most sensitive subpopulation to lead exposure and is the only population considered in this model. The IEUBK model predicts as its output a child's lead blood concentration, presented as a probability curve around the geometric mean of the blood lead concentration. As generally applied, the U.S. EPA considers exposures resulting in a predicted blood lead level of 10 ug/L or less in 95% of exposed children as acceptable.

For this risk assessment, version 0.99D of the IEUBK model (U.S. EPA 1994b) was used. The IEUBK model requires the input of media-specific concentrations (i.e., soil, ground water, air, and food.). Site-specific media concentrations should be entered in the model when available. The model contains standard default concentrations for these exposure media that may be used where site specific data are unavailable. The IEUBK model's standard default values represent average lead concentrations in various environmental media throughout the U.S. The following table presents the standard default lead values for air, water, diet, and soil used in version 0.99D of the IEUBK model. Also presented in the table are the estimated lead concentrations in air, water, diet and soil under the high end maximum exposure scenario attributable to emissions from the ESSROC facility.

Media	IEUBK Default Values	High End Values	Total IEUBK plus High End
Air (concentration)	0.100 ug/m^3	0.00036 ug/m^3	0.100036 ug/m^3
Diet (total intake)	5.53 to 7.0 ug/day*	0.12 ug/day	5.65 to 7.12 ug/day
Soil (concentration)	200 mg/kg	0.6 mg/kg	200.6 mg/kg
Water (concentration)	4 ug/L	0.0001 ug/L	4.0001 ug/L

*Age group specific.

As seen on this table, the modeled media concentrations due to facility emissions are generally an order of magnitude or more less than the IEUBK model's standard default values. If the model is run using the standard default values and assuming exposure by children 0 to 72 months, 99.57% of the exposed children would have a blood lead level

below 10 ug/dL. Running the model using the sum of the facility's contribution to lead media concentrations (under the high end exposure scenario) and the standard defaults, produces a similar result (99.57% of the exposed children would have a blood lead level below 10 ug/dL). Based on this evaluation, it can be concluded that the ESSROC facility makes no significant contribution to child lead exposures within the study area.

8.6 RISK CHARACTERIZATION CKD WASTE PILE ACTIVITIES

As described in Section 4.0, air concentrations and deposition fluxes were modeled for fugitive emissions associated with CKD waste pile activities. Table 8-15 presents the estimated total deposition fluxes, and wet and dry deposition fluxes for metals identified in CKD emissions. The deposition fluxes shown on Table 8-15 represents those values modeled for a maximum exposure area encompassing a 2.0 kilometer radius from the ESSROC stack. As described previously, unlike stack emissions, fugitive emissions will likely have maximum areas of deposition at points closer to the ESSROC facility. Therefore, a smaller radius was used to estimate the area of maximum exposure for fugitive emissions.

As shown on Table 8-15, the total deposition fluxes for CKD fugitive emissions for antimony, arsenic, cadmium, lead, mercury, and selenium are over an order of magnitude less than those values associated with the kiln emissions. Based on this comparison, it can be assumed that the potential human health risks associated with exposure to CKD waste pile emissions for these metals are not significant relative to the emissions from the cement kiln stack.

With the exception of barium, the total deposition fluxes associated with CKD waste pile emissions for the other chemicals (beryllium, chromium, nickel, silver, and thallium) are slightly less than an order of magnitude less than those values associated with the kiln emissions. However, when considering the total risks associated with exposure to kiln emissions, the relative contribution of CKD waste pile emissions to the total risks is relatively insignificant. Specifically, Table 8-15 presents the total risks and hazard indices for the metals for those land-based indirect exposure pathways that CKD fugitive emissions would likely contribute (i.e., plant and animal consumption and direct contact exposures to soil). The hazard indices and risks presented on the table represent the sum for these three pathways for the adult under the high end exposure scenario. As shown on

the table, the total HI's for these metals associated with kiln emissions are less than 0.001. Therefore, when considering that the total hazard indices for these chemicals and pathways combined for kiln emissions fall somewhere less 0.001, the relative contribution to the total hazard indices from CKD waste pile emissions is considered insignificant.

As shown on Table 8-15, the total deposition associated with CKD emissions for barium slightly exceeds the total deposition associated with kiln emissions. However, the total HI associated with kiln emissions of barium for the land-based indirect exposure pathways is 0.000000825. Therefore, although the potential exists for greater exposures to barium in fugitive emissions from CKD activities versus kiln emissions, the potential risks associated with these exposures are not considered significant.

Based on the above evaluation, the relative contributions of CKD waste pile fugitive emissions to total noncarcinogenic and carcinogenic risks for all chemicals are considered insignificant relative to kiln emissions.

8.7 TOTAL RISKS

In this section the pathway-specific carcinogenic and noncarcinogenic risks presented in Section 8.3 are combined to determine potential total carcinogenic and noncarcinogenic risks for different subpopulations in the study area. The different subpopulations of interest are defined primarily by their involvement in one or more of the activities for which pathway specific risks were modeled. The specific activities that play most significantly into the definition of these subpopulations include home gardening, consumption of home raised animal products and drinking water source. Conversely, some pathway specific risks are assumed to be the same across all individual subpopulations. This includes soil ingestion, soil dermal exposure, and inhalation. A summary of the total noncarcinogenic and carcinogenic risks by each pathway is presented in Table 8-16.

The summation of risks presented in this section assumes that an individual living in a particular area of exposure (i.e., maximum exposure area) receives essentially all of his or her exposure within that area. Clearly, any individual whose residence is within a particular modeled area of exposure will receive a portion of their individual exposure at

different locations in and out of that area of exposure (or in terms of food consumption scenarios, an individual will likely consume vegetables and animal products produced in different locations). It would be impossible, however, to model each potential combination of exposures. The assumption of no exposures outside of the modeled area for a particular population is conservative when determining high end exposures because residents within the particular modeled area could only have lower exposures if they spend significant amounts of time outside the modeled area.

8.7.1 HIGH END EXPOSURES

Total risks for the high end exposure scenario are presented in Table 8-16. For the farm adult and children, the high end carcinogenic and noncarcinogenic risks from inhalation, soil ingestion and dermal contact, surface water exposures (including drinking water and fish consumption), consumption of home grown vegetables, and consumption of farm raised animal products were each summed. The HQ for one chemical (methyl mercury) for the fish ingestion pathway causes the total hazard index for all pathways combined to exceed one. The HQs for all other chemicals and pathways are less than 0.010. Total noncarcinogenic risks for all of these exposures were determined to be 4.91 and 6.29 for the children and adults, respectively. Total carcinogenic risks for these pathways of exposure were determined to be 7.7×10^{-7} and 1.57×10^{-6} for the child and adult, respectively. Although the combined pathway-specific risks were at 1×10^{-6}, the risks for each of the individual pathways were below 1×10^{-6} for both the adult and child populations.

8.7.2 TYPICAL EXPOSURES

Total risks for the adult and child subpopulations for the maximum exposure area are presented in Table 8-16. For the adult and child residents, the typical carcinogenic and noncarcinogenic risks from inhalation, soil ingestion and dermal contact, surface water exposures (including drinking water and fish consumption), consumption of home grown vegetables, and consumption of locally raised animal products were each summed. Total noncarcinogenic risks for all of these exposures at the maximum exposure area were determined to be 1.02 and 0.57 for the child and adult, respectively. The HQ for methyl mercury for the fish ingestion pathway causes the total hazard index for all pathways combined to exceed one. Total carcinogenic risks for these pathways of exposure at the

maximum exposure area were determined to be 4.38×10^{-7} and 6.09×10^{-7} for the child and adult, respectively.

8.8 RISK-BASED EMISSION LIMITS FOR TIER 1A METALS

A total of seven metals (antimony, barium, mercury, nickel, selenium, silver, and thallium) are listed as Tier 1A metals. Estimates of potential non-carcinogenic risks associated with emissions of these Tier 1A metals measured during ESSROC's trial burn have been presented previously. There is, however, considerable uncertainty in the estimation of emission rates for metals because of the complex mechanism affecting metal removal from combustion gases and the high variability in metal feed rates to the kilns. Given this uncertainty, with respect to setting permit limits for these Tier 1A metals ESSROC proposes to establish risk-based emission limits. The Tier 1A emission rates were established by back-calculating, based on a target hazard index, from a combination of direct and indirect exposure pathways. The hazard index (HI) was used as the target risk because the Tier 1A metals are associated only with non-carcinogenic health effects.

For each of the Tier 1A metals, the target risk level (i.e., HI) was established as a cumulative HI at or just below 1.0 across all direct and indirect pathways of exposure for the most sensitive exposure populations evaluated in the risk assessment. The most sensitive exposure populations for each of the direct and indirect pathways of exposure are as follows: air inhalation (child), direct contact exposures to soil (child under high end exposure scenario), drinking water exposures (child), surface water exposures (child under high end exposure scenario – France Park), fish ingestion (adult under high end exposure scenario), plant consumption (adult under high end exposure scenario), and beef consumption (child under high end exposure scenario).

The pathway-cumulative target HI of 1.0 was selected because it represents a conservative target risk level for setting the emission limits for the Tier 1A metals. Specifically, the emission rates measured during ESSROC's trial burn provide the best approximation of emissions that are expected to occur under normal operating conditions. As such, the trial burn emission rates provide the best estimate of the emissions that would be expected to occur close to 100% of the operating life of the kilns. Conversely, the emission rates used to set the permit levels for the Tier 1A metals would represent

worst case, high-end emission rates that would only be expected to occur infrequently. Consequently, a target risk level of 1.0 for the Tier 1A metals emissions still provides an adequate margin of protection to human health and the environment.

Table 8-17 presents Tier 1A metal emission rates derived by backcalculating using a target HI of 1.0. As shown on the table, with the exception of mercury, the cumulative HI's summed across all pathways of exposure for each Tier 1A metal fall at or below 1.0. With respect to mercury, ESSROC proposes to set the permit emission rate at the actual emission rate measured during the 1998 trial burn.

8.8.1 Documentation Used to Support Tier 1A Feed Rate Limits

According to 40 CFR 266.106(d)-(e), conformance with the adjusted Tier I (Tier IA) metals controls is demonstrated by air dispersion modeling to predict the maximum annual off-site ground level concentration and a demonstration that acceptable ambient levels are not exceeded. In addition, the feed rate of the Tier IA metals can be adjusted from the 40 CFR 266 Appendix I default feed rates by accounting for site-specific dispersion modeling. Under this approach, the Tier IA feed rates are determined by back-calculating from the acceptable ambient levels using dispersion modeling to determine the maximum allowable emission rate. The emission rates become the Tier IA feed rate screening limit. For risk assessment purposes, these allowable Tier IA emission rate screening limits are calculated to the discharge of the main stack. These screening rate limits can and will differ from the Tier IA feed rate limits (as fed to the kiln) based on site-specific partitioning conditions.

Using "metals partitioning" (i.e., system removal efficiencies) to back-calculate from the Tier IA feed rate screening limit to a Tier IA metal feed rate limit is permitted under *Appendix IX To Part 266 – Methods Manual For Compliance With The BIF Regulations.* More specifically, *Section 9.0 – Procedures for Determining Default Values for Partitioning of the Metals, Ash and Total Chloride/Chlorine* provides an approach to use either engineering judgments or site-specific testing in determining system removal efficiencies of each metal.

While it is true that the U.S. EPA default partitioning factor is 100% (SRE = 0%)[1], the approach allows for ESSROC to use a "supportable, site-specific value developed following the guidelines of Section 9.4". Referencing Section 9.4, one approach allowed is for ESSROC to use site-specific emission data to support an SRE.[2]

8.8.1.1 JUSTIFICATION FOR SREs USED IN CALCULATIONS

In calculating the Tier IA feed rate for each metal, ESSROC used the following SREs based on site-specific testing conducted under Phase II of the Trial Burn:

Metal	System Removal Efficiency (SRE)
Antimony	99.891%
Barium	99.987%
Mercury	96.693%
Nickel	99.866%
Selenium	99.702%
Silver	99.860%
Thallium	99.891%

These SRE values were derived by averaging the three, Phase II test runs (Runs 4-6) of the Trial Burn.[3] ESSROC is justified in using these SREs because:

1. Phase II of the Trial Burn achieved all QA/QC procedures as defined in the Trial Burn protocol and Quality Assurance Project Plan (QAPP);

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[1] 40 CFR 266, Appendix IX, Section 9.1, assumes that 100% of the metal input is emitted from the main stack.

[2] 40 CFR 266, Appendix IX, Section 9.4, Bullet Item #1.

[3] See Table 5. All metal inputs were calculated by using the average test run process feed stream feed rates by the reported lab metal concentration of each feed stream.

2. U.S. EPA approved all Trial Burn results;

3. The Tier IA metal emissions conform with the established operating conditions of the Trial Burn and post-Trial Burn operating conditions;

4. The emissions of the Tier IA metals were above the laboratory detection limit (DL) for all test runs of Phase II conditions.

Table 8-17A summarizes the methods used by ESSROC to calculate its proposed Tier 1A metals feed rate limits. As shown on Table 8-17A, column one presents the site-specific system removal efficiencies (SRE) calculated for the Tier 1A metals. The SRE values were derived as described previously. The second column presents the site-specific Tier 1A total feed stream metal feed rates (at the kiln) developed by using historical data and adding a 3-sigma variability factor to the average. These values are in units of grams/hour for one kiln. The third column presents the theoretical "forward-calculated" Tier 1A metal emission rates (at the stack) based on the site-specific SRE. The values are in units of grams/hour for one kiln. The fourth column presents the theoretical Tier 1A metal emission rates in units of grams/second assuming the operation of two kilns. As shown on Table 8-17A in the fourth column, the calculated Tier 1A metals emission rates are well below the derived risk-based 2 kiln emission rates shown on Table 8-17. In fact, with the exception of mercury and thallium, the calculated Tier 1A metals emission rates based on the site-specific SRE's are three or more orders of magnitude less than the derived risk-based 2 kiln emission rates presented on Table 8-17. Therefore, as demonstrated on Table 8-17 and Table 8-17A, ESSROC's proposed feed rate limits for the Tier 1A metals are well within the acceptable level of risks associated with indirect and direct pathways of exposure, and as such, are protective of human health and the environment.

8.9 IMPACT OF MACT STANDARDS ON ESTIMATED RISKS

This section presents an evaluation of the risks associated with stack emissions from the ESSROC facility under the scenario where the stack emissions meet the proposed Maximum Achievable Control Technology Standards. On September 30, 1999 the U.S. EPA published Final Standards for Hazardous Air Pollutants for Hazardous Waste Combustors (termed the Phase I rule). In the Phase I rule, the U.S. EPA adopted National

Emissions Standards for Hazardous Air Pollutants pursuant to Section 112(d) of the Clean Air Act (CAA) to control toxic emissions from the burning of hazardous waste in incinerators, cement kilns, and lightweight aggregate kilns. The emission standards proposed in the Phase I rule created a technology-based national cap for hazardous air pollutant emissions from the combustion of hazardous waste in these units. Section 112(d) of the CAA requires emission standards for hazardous air pollutants to be based on the performance of the Maximum Achievable Control Technology (MACT).

On February 14, 2002 the U.S. EPA published a notice finalizing specific changes to the Phase I rule. The notice published on February 14, 2002 finalized Interim Standards for existing and new incinerators. The interim standards for existing cement kilns that burn hazardous wastes are described as follows.

Hazardous Air Pollutant	Interim Standard
Dioxin/Furan	0.2 TEQ* ng/dry standard cubic meter (dscm) at temperatures greater than 400° F inlet at ESP 0.4 TEQ* ng/dry standard cubic meter (dscm) at temperatures less than 400° F inlet at ESP
Mercury**	132 ug/dscm
Particulate Matter	0.15 kg/Mg dry kiln feed
Semivolatile Metals**	330 ug/dscm
Low Volatile Metals**	56 ug/dscm
Hydrochloric acid/Chlorine Gas	130 ppmv
Hydrocarbons	20 ppmv
Destruction and Removal Efficiency	99.99% for each principal organic hazardous constituent

*Toxicity Equivalent Quotient, the international method of relating the toxicity of various dioxin/furan congeners to the toxicity of 2,3,7,8-TCDD.
** Corrected at 7% Oxygen

ESSROC has previously conducted trial burn tests in support of its Part B permit application. The RCRA trial burn testing included the measurement of dioxin/furan congeners, as well as other metal, semivolatile and volatile constituents, in stack emissions. The results of the RCRA stack emissions testing were used in this risk assessment to evaluate the potential human health risks associated with long term emissions from the ESSROC facility. However, with the promulgation of the MACT

Interim Standards Rule, in the future ESSROC stack emissions will be required to meet the hazard air pollutant MACT standards described above. Therefore, this section is intended to provide an evaluation of the risks associated with stack emissions under the MACT standard scenario.

With respect to the results of the risk evaluation of stack emissions from the ESSROC facility, the dioxin/furan congeners were determined to be the greatest contributors to risk for the indirect pathways of exposure evaluated. The dioxin/furan congeners typically are the "risk drivers" for most indirect risk assessments, relative to other constituents typically present in stack emissions, due to their potent toxicity as well as their significant bioaccumulative properties in environmental media and aquatic and terrestrial biota. Because of the significant bioaccumulative properties of dioxin/furan congeners, food chain exposures are the indirect pathways of exposure most significantly impacted by dioxin/furan emissions. Therefore, for purposes of assessing the impact of MACT standards on the ESSROC human health risk assessment, the potential risks posed by dioxin/furan emissions on the fish, homegrown vegetable, and beef/dairy products consumption pathways of exposure are evaluated. The methods used to evaluate risks for these indirect exposure pathways are described in the following section.

8.9.1 METHODS

Information provided in ESSROC March 18, 1999 RCRA Trial Burn Report was used to convert the MACT standard of 0.2 ng/dscm for 2,3,7,8-TCDD equivalents to a stack gas emission rate in units of grams/sec. First, a stack gas flow rate of 58,185 dry cubic feet per minute (dscfm) was assumed. This value represents the average stack gas flow rate for the Phase I stack testing conducted by ESSROC during their 1998 RCRA trial burn testing. This value represents the average of three stack test runs completed on October 13 and 14, 1998. The average stack gas temperature during these three stack tests was 366 degrees Fahrenheit.

The MACT standard in units of ng/dscm was converted to a stack gas emission rate in units of g/sec as follows:

$(0.2\ ng/m^3)\ (1g/10^9\ ng)\ (58{,}185\ ft^3/min)\ (1\ m^3/35.315\ ft^3)\ (1\ min/60\ sec)$

$= 5.49 \times 10^{-9}\ g/s$

Using a stack gas emission rate of 5.49 x 10^{-9} g/s for 2,3,7,8-TCDD equivalents, the potential risks associated with stack emissions of 2,3,7,8-TCDD equivalents was evaluated for the fish, homegrown vegetable, and beef/dairy products consumption pathways. For this evaluation, the exposure assumptions for an adult under the high end exposure scenario described in Section 6.3.4 were used to evaluate risks from fish consumption. Additionally, the exposure assumptions for an adult under the high end exposure scenario described in Section 6.4 were used to evaluate risks from homegrown vegetable consumption and the exposure assumptions described in Section 6.5 were used to evaluate risks from consumption of beef/dairy products.

Table 8-18A presents the dioxin/furan risks to an adult under the high end exposure scenario from consumption of fish. As shown on Table 8-18A, the estimated total risk from consumption of fish containing dioxin/furan at concentrations associated with stack emission rates measured during the RCRA trial burn is 5.50 x 10^{-8}. The estimated risk from consumption of fish containing dioxin/furan at concentrations associated with stack emission rates at the MACT standard is 5.24 x 10^{-9}. As shown on the table, the estimated risks for the MACT standard are almost an order of magnitude less than the risks associated with the RCRA trial burn emission rates.

Table 8-18B presents the estimated dioxin/furan risks to an adult under the high end exposure scenario from consumption of homegrown vegetables. As shown on Table 8-18B, the estimated risk from consumption of homegrown vegetables containing dioxin/furan at concentrations associated with stack emission rates measured during the RCRA trial burn is 3.28 x 10^{-7}. The total risk from consumption of homegrown vegetables containing dioxin/furan congeners at concentrations associated with stack emission rates at the MACT standard is 2.51 x 10^{-7}.

Table 8-18C presents the estimated risks to an adult under the high end exposure scenario from consumption of beef/dairy products containing dioxin/furans. As shown on Table 8-18C, the total estimated risk from consumption of beef/dairy products containing dioxin/furan at concentrations associated with stack emission rates measured during the RCRA trial burn is 6.22 x 10^{-7}. The total estimated risk from ingestion of beef/dairy products containing dioxin/furan at concentrations associated with stack emission rates at the MACT standard is 1.28 x 10^{-7}.

Section 9.4 presents an estimation of the carcinogenic risks for hypothetical subsistence fisher and farmer populations. For each of these populations, high-end consumption rates were used and it was assumed that these populations obtained 100% of their fish, vegetable, and animal product diets from products grown or caught within the maximum exposure area modeled in this risk assessment.

Table 8-18A presents the dioxin/furan risks to an adult subsistence fisherman assuming that 100% of his fish diet is caught within the maximum exposure area. As shown on Table 8-18A, the estimated risk for the subsistence fisherman from consumption of fish containing dioxin/furan congeners at concentrations associated with stack emission rates measured during the RCRA trial burn is 2.20×10^{-7}. The estimated risk for the subsistence fisherman from consumption of fish containing dioxin/furan at concentrations associated with stack emission rates at the MACT standard is 4.96×10^{-8}. As shown on the table, the estimated risks for the MACT standard are almost an order of magnitude less than the risks associated with the RCRA trial burn emission rates.

Table 8-18B presents the estimated dioxin/furan risks to an adult subsistence farmer from consumption of homegrown vegetables. As shown on Table 8-18B, the total estimated risk for the subsistence farmer from consumption of homegrown vegetables containing dioxin/furan congeners at concentrations associated with stack emission rates measured during the RCRA trial burn is 5.33×10^{-7}. The total estimated risk for the subsistence farmer from consumption of homegrown vegetables containing dioxin/furan at concentrations associated with stack emission rates at the MACT standard is 2.52×10^{-7}.

Table 8-18C presents the estimated risks to an adult subsistence farmer from consumption of beef/dairy products containing dioxin/furans. As shown on Table 8-18C, the total estimated risk for the subsistence farmer from consumption of beef/dairy products containing dioxin/furan at concentrations associated with stack emission rates measured during the RCRA trial burn is 1.43×10^{-6}. The total estimated risk for the subsistence farmer from consumption of beef/dairy products containing dioxin/furan at concentrations associated with stack emission rates at the MACT standard is 3.05×10^{-7}.

In summary, the MACT limits will have a positive impact on the potential risks posed by dioxin/furan congeners, as well as other constituents present, in stack emissions from the ESSROC facility. Specifically, meeting the MACT limits will result in an approximate

order of magnitude reduction in the actual risks posed by emissions from the ESSROC facility. As such, meeting the MACT limits will provide an added measure of protection to public health and the environment associated with stack emissions from the ESSROC facility.

Table 8-1

Hazard Quotients and Indices from Inhalation Exposures to Kiln and Fugitive Emissions

ESSROC

Logansport, Indiana

Chemical	Maximum Exposure Area Adult	Maximum Exposure Area Child
Metals		
Antimony	1.161E-06	3.9179E-06
Arsenic	1.803E-06	6.082E-06
Barium	7.205E-08	2.4307E-07
Beryllium	3.426E-06	1.1558E-05
Cadmium	0.0001266	0.00042705
Chromium (VI)	5.931E-07	2.0009E-06
Chromium, total	8.937E-10	3.0149E-09
Elemental Mercury	2.432E-07	8.2034E-07
Divalent Mercury	4.906E-05	0.00016549
Nickel	6.869E-08	2.3171E-07
Selenium	3.55E-07	1.1977E-06
Silver	5.276E-08	1.7798E-07
Thallium	7.964E-06	2.6865E-05
Volatiles		
Acetone	1.65E-04	6.09E-04
Acrylonitrile	1.418E-05	4.7842E-05
Allyl Chloride	4.37E-06	1.4742E-05
Bromodichloromethane	1.26E-09	4.252E-09
Bromomethane	1.563E-06	5.2736E-06
Bromoform	2.343E-09	7.9047E-09
2-Butanone	2.42E-05	8.91E-05
Carbon disulfide	4.458E-08	1.5039E-07
Carbon tetrachloride	7.903E-07	2.6662E-06
Chlorobenzene	1.05E-05	3.95E-05
Chloroethane	1.966E-10	6.632E-10
Chloroform	5.092E-06	1.7179E-05
Cumene (isopropylbenzene)	5.211E-10	1.7579E-09
Dibromochloromethane	1.447E-09	4.8798E-09
1,2-Dichlorobenzene	1.027E-09	3.4658E-09
1,4-Dichlorobenzene	4.287E-09	1.4463E-08
1,1-Dichloroethane	1.859E-10	6.2724E-10
1,1-Dichloroethene	5.621E-09	1.8961E-08
cis 1,2-Dichloroethene	2.216E-09	7.4767E-09
trans 1,2-Dichloroethene	2.208E-09	7.4482E-09
1,2-Dichloropropane	3.73E-08	1.2585E-07
cis 1,3-Dichloropropene	4.53E-09	1.5282E-08
trans 1,3-Dichloropropene	5.70E-05	2.17E-04
Dichlorodifluoromethane	1.768E-09	5.9642E-09
Ethylbenzene	1.57E-06	5.82E-06
Methylene Chloride	1.18E-07	3.99E-07

Table 8-1

Hazard Quotients and Indices from Inhalation Exposures to Kiln and Fugitive Emissions

ESSROC

Logansport, Indiana

Chemical	Maximum Exposure Area Adult	Maximum Exposure Area Child
n-Hexane	2.01E-06	7.70E-06
Styrene	2.88E-07	1.06E-06
Tetrachloroethylene	8.01E-08	2.70E-07
1,1,1-Trichloroethane	5.344E-09	1.8027E-08
Trichlorofluoromethane	1.99E-05	7.30E-05
Toluene	7.00E-05	2.27E-04
Xylene (m/p)	9.05E-10	3.05E-09
o-Xylene	2.732E-10	9.2174E-10
Vinyl acetate	1.52E-06	5.59E-06
Semi-Volatiles		
Anthracene	4.771E-10	8.0474E-09
Benzoic Acid	1.683E-09	2.8394E-08
Bis(2-ethyl hexyl)phthalate	3.485E-07	5.8786E-06
Butyl benzyl phthalate	4.563E-10	7.696E-09
4-Chloroaniline	1.04E-08	1.755E-07
2-Chloronaphthalene	4.179E-10	7.0486E-09
2-Chlorophenol	3.018E-08	5.091E-07
1,2-dichlorobenzene	5.752E-10	9.7026E-09
1,3-dichlorobenzene	5.466E-10	9.2196E-09
1,4-dichlorobenzene	1.319E-09	2.2248E-08
Dimethylphthalate	2.369E-12	3.9952E-11
Diethyl phthalate	1.37E-10	2.3115E-09
2,4-Dimethylphenol	2.605E-09	4.3943E-08
di-n-butylphthalate	7.78E-10	1.3122E-08
Di-n-octyl phthalate	3.164E-09	5.3364E-08
2,4-Dinitrophenol	9.508E-08	1.6037E-06
2,4-Dinitrotoluene	4.449E-08	7.505E-07
2,6-Dinitrotoluene	1.13E-07	1.9063E-06
Fluoranthene	3.231E-08	5.4492E-07
Fluorene	3.706E-09	6.2507E-08
Hexachlorobenzene	9.178E-08	1.5481E-06
Hexachlorobutadiene	4.45E-07	7.5052E-06
Hexachlorocyclopentadiene	4.162E-06	7.0201E-05
Hexachloroethane	9.373E-08	1.5809E-06
2-Methylphenol	3.932E-09	6.632E-08
4-Methylphenol	4.548E-09	7.6707E-08
Naphthalene	1.476E-05	0.00024903
2-Nitroaniline	1.711E-06	2.8865E-05
Nitrobenzene	9.237E-08	1.5581E-06
Pentachlorophenol	3.521E-09	5.9383E-08
Phenanthrene	7.655E-08	1.2912E-06

Table 8-1

Hazard Quotients and Indices from Inhalation Exposures to Kiln and Fugitive Emissions

ESSROC

Logansport, Indiana

Chemical	Maximum Exposure Area Adult	Maximum Exposure Area Child
Phenol	5.47E-09	9.227E-08
Pyrene	4.306E-08	7.2627E-07
1,2,4-Trichlorobenzene	9.711E-10	1.638E-08
2,4,5-Trichlorophenol	7.613E-10	1.2842E-08
PCBs		
Total Mono CB	8.239E-08	2.796E-07
Total Di CB	5.871E-08	1.98E-07
Total Tri CB	7.563E-08	2.5506E-07
Total Tetra CB	5.583E-08	1.883E-07
Total Penta CB	5.245E-08	1.7689E-07
Total Hex CB	1.895E-07	6.3908E-07
Total Hepta CB	2.91E-07	9.8145E-07
Total Octa CB	6.615E-08	2.2254E-07
Total Nona CB	3.35E-09	1.1355E-08
Total Deca CB	5.566E-10	1.8773E-09
Total Hazard Indices	**0.0005927**	**0.00238639**

Table 8-2
Hazard Quotient and Indices from Direct Contact Exposures to Soil
ESSROC
Logansport, Indiana

Chemical	Typical Exposure Scenario Maximum Exposure Area			High End Exposure Scenario Maximum Exposure Area	
	Adult	Child		Adult	Child
Antimony	6.66E-06	6.05E-05		6.73E-06	6.14E-05
Arsenic	8.51E-06	7.73E-05		8.60E-06	7.84E-05
Barium	3.99E-07	3.62E-06		4.03E-07	3.67E-06
Beryllium	8.19E-08	7.43E-07		8.27E-08	7.54E-07
Cadmium	4.83E-05	4.39E-04		4.89E-05	4.45E-04
Chromium (VI)	2.89E-08	2.62E-07		2.92E-08	2.66E-07
Chromium, total	7.66E-09	6.96E-08		7.74E-09	7.06E-08
Divalent Mercury	9.82E-04	8.92E-03		9.92E-04	9.05E-03
Methyl Mercury	8.39E-06	7.62E-05		8.48E-06	7.73E-05
Nickel	4.43E-07	4.02E-06		4.47E-07	4.08E-06
Selenium	4.00E-07	3.63E-06		4.04E-07	3.69E-06
Thallium	5.25E-05	4.77E-04		5.31E-05	4.84E-04
Acenaphthene	6.51E-10	4.84E-09		7.03E-10	5.49E-09
Bis(2-ethyl hexyl)phthalate	1.23E-07	9.13E-07		1.33E-07	1.04E-06
2-Chlorophenol	1.81E-08	1.34E-07		1.95E-08	1.52E-07
1,4-dichlorobenzene	9.18E-10	6.82E-09		9.92E-10	7.74E-09
2,4-Dimethylphenol	1.85E-10	1.37E-09		2.00E-10	1.56E-09
2,4-Dinitrotoluene	4.18E-09	3.11E-08		4.51E-09	3.52E-08
2,6-Dinitrotoluene	9.19E-09	6.83E-08		9.92E-09	7.74E-08
Fluoranthene	1.98E-07	1.47E-06		2.14E-07	1.67E-06
Hexachlorobutadiene	1.35E-05	1.01E-04		1.46E-05	1.14E-04
Hexachlorocyclopentadiene	4.97E-09	3.69E-08		5.36E-09	4.19E-08
Naphthalene	4.65E-07	3.45E-06		5.02E-07	3.92E-06
2-Nitroaniline	1.30E-07	9.68E-07		1.41E-07	1.10E-06
Nitrobenzene	1.97E-08	1.46E-07		2.13E-08	1.66E-07
Pentachlorophenol	2.21E-09	1.65E-08		2.39E-09	1.87E-08
Phenol	2.19E-10	1.63E-09		2.36E-10	1.84E-09

Table 8-2
Hazard Quotient and Indices from Direct Contact Exposures to Soil
ESSROC
Logansport, Indiana

Chemical	Typical Exposure Scenario Maximum Exposure Area			High End Exposure Scenario Maximum Exposure Area	
	Adult	Child		Adult	Child
Pyrene	1.00E-06	7.46E-06		1.08E-06	8.45E-06
1,2,4-Trichlorobenzene	7.27E-09	5.40E-08		7.85E-09	6.12E-08
2,4,5-Trichlorophenol	1.12E-09	8.31E-09		1.21E-09	9.42E-09
Total Mono CB	1.01E-08	8.78E-08		1.19E-08	9.16E-08
Total Di CB	7.20E-09	6.22E-08		8.40E-09	6.49E-08
Total Tri CB	9.27E-09	8.01E-08		1.08E-08	8.36E-08
Total Tetra CB	6.85E-09	5.92E-08		7.99E-09	6.17E-08
Total Penta CB	6.43E-09	5.56E-08		7.50E-09	5.79E-08
Total Hex CB	2.32E-08	2.01E-07		2.71E-08	2.09E-07
Total Hepta CB	3.57E-08	3.08E-07		4.16E-08	3.22E-07
Total Octa CB	8.11E-09	6.99E-08		9.44E-09	7.29E-08
Total Nona CB	4.06E-10	3.57E-09		4.82E-10	3.72E-09
Total Deca CB	6.82E-11	5.90E-10		7.96E-11	6.15E-10
Total Hazard Indices	**0.001123445**	**0.010175926**		**0.001136444**	**0.01033787**

Table 8-3
Hazard Quotient and Indices from Drinking Water Exposure
ESSROC
Logansport, Indiana

Chemical	Eel River	
	Adult	Child
Antimony	4.374E-05	0.0001021
Arsenic	8.661E-05	0.0002021
Barium	2.873E-06	6.704E-06
Beryllium	1.988E-08	4.639E-08
Cadmium	0.0001905	0.0004445
Chromium (VI)	4.479E-07	1.045E-06
Chromium, total	7.107E-13	1.658E-12
Divalent Mercury	3.869E-06	9.028E-06
Methyl Mercury	8.633E-08	2.014E-07
Nickel	2.013E-06	4.697E-06
Selenium	2.311E-05	5.392E-05
Thallium	0.0002186	0.0005101
Acenaphthene	6.751E-11	1.575E-10
Bis(2-ethyl hexyl)phthalate	3.64E-08	8.494E-08
2-Chlorophenol	2.812E-06	6.56E-06
1,4-dichlorobenzene	6.05E-08	1.412E-07
2,4-Dimethylphenol	8.827E-08	2.06E-07
2,4-Dinitrotoluene	4.276E-06	9.978E-06
2,6-Dinitrotoluene	1.093E-05	2.549E-05
Fluoranthene	2.052E-07	4.789E-07
Hexachlorobutadiene	6.41E-07	1.495E-06
Hexachlorocyclopentadiene	2.249E-08	5.249E-08
Hexachloroethane	2.909E-06	6.787E-06
Naphthalene	2.573E-07	6.003E-07
2-Nitroaniline	0.0001616	0.000377
Nitrobenzene	9.126E-06	2.129E-05
Pentachlorophenol	2.702E-07	6.305E-07
Phenol	4.024E-07	9.39E-07
Pyrene	5.365E-09	1.252E-08
1,2,4-Trichlorobenzene	1.822E-07	4.251E-07
Total Mono CB	5.421E-09	1.265E-08
Total Di CB	3.863E-09	9.014E-09
Total Tri CB	4.976E-09	1.161E-08
Total Tetra CB	3.674E-09	8.572E-09
Total Penta CB	3.451E-09	8.052E-09
Total Hex CB	1.247E-08	2.909E-08
Total Hepta CB	1.915E-08	4.468E-08
Total Octa CB	4.353E-09	1.016E-08
Total Nona CB	1.01E-09	2.358E-09
Total Deca CB	3.663E-11	8.546E-11
Total Hazard Indices	**0.0007658**	**0.0017868**