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**THE NEW CASTLE SPILL SITE  
NEW CASTLE, DELAWARE**

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**Final  
Endangerment Assessment  
Volume II**

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*5 May 1989*

Prepared For:  
**Witco Corporation**  
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Woodcliff Lake, New Jersey

Prepared By:



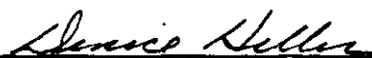
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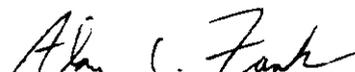
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FINAL  
ENDANGERMENT ASSESSMENT  
FOR  
THE NEW CASTLE SPILL SITE  
VOLUME II

5 May 1989

  
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## EXECUTIVE SUMMARY

During the summer of 1977, tris(2-chloropropyl)-phosphate reportedly leaked from a drum in the drum storage area at Witco Corporation's New Castle facility. The Witco facility is bordered on the south by the New Castle Board of Water and Light (NCBW&L) property, which at the time of the spill served as a water supply source for the City of New Castle.

In December 1982, the New Castle Spill Site was listed on US Environmental Protection Agency's (U.S. EPA's) National Priorities List. An Administrative Consent Order (ACO) between Witco Corporation and U.S. EPA Region III was signed in December 1987. Environmental Resources Management, Inc. (ERM) was then contracted by Witco Corporation to prepare a Work Plan for the Remedial Investigation/Feasibility Study (RI/FS) for the site. ERM began work at the New Castle Spill Site (NCSS) in February 1988.

This Endangerment Assessment (EA) evaluates the risks posed by compounds detected at the New Castle Spill Site under the No-Action Alternative. The ground water data for the site generally indicates that compound concentrations are decreasing. This EA addresses present, existing conditions as well as a hypothetical, future-use ground water scenario.

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U.S. EPA's process for developing a group of site-specific indicator compounds was used for NCSS. The indicators chosen were as follows: trichloroethene, trans-1,2-dichloroethene and tris(2-chloropropyl)phosphate. These compounds were considered to represent a majority of the potential risk to both an actual and hypothetically exposed population based on the RI findings.

A present, existing conditions exposure scenario exists in which ground water transports compounds located on site to the marsh area where it mixes with surface water. A population is potentially exposed through dermal contact with surface water and soils/sediments and incidental ingestion of the soils/sediments.

An exposed population was not available for assessment of potential exposure to ground water via residential use under present, existing conditions scenario because 1) drinking water is supplied by municipal or commercial means, 2) private wells in Columbia aquifer in the downgradient direction do not exist, 3) closest municipal well is located approximately 0.7 miles downgradient, and 4) there are no users of the Columbia aquifer since the Potomac aquifer (regional potable aquifer) is available. Therefore, a hypothetical, future-use ground water scenario involving adults, children 6 to 12 years, and children 2 to 6 years and consisting of a well installed in the Columbia aquifer at the property boundary was employed to determine the hazard or risk to a population using ground water leaving the NCSS property. The concentration of the indicator compounds found in the associated ground water zone was used to assess the potential risk or hazard from the hypothetical ground water use.

Exposure pathways for the hypothetical scenario were ingestion of compounds detected in the Columbia aquifer and dermal contact with and inhalation of these compounds during bathing activities.

Toxicity levels for each compound were found in literature or calculated from acceptable daily intakes. Using the exposure pathways and toxicity levels, the hazard or risk attributable to the levels of the indicator compounds detected at the site to an exposed population was determined.

Upper bound reasonable case and worst case estimates were made for each exposure scenario. The reasonable case is based on realistic exposure durations, frequencies, and pathways; while the worst case assumes that an individual may be chronically exposed to the highest concentrations detected at the site. This type of estimate means a 95 percent probability exists that the risk may be overestimated with a 5 percent chance of underestimating the risk.

All of the carcinogenic risks calculated were within U.S. EPA's range. Table ES-1 is a summary of the calculated carcinogenic risks and noncarcinogenic hazard indices for the New Castle Spill Site. The risk/hazard from present conditions (i.e., exposure to surface water and sediments) were orders of magnitude below EPA's guidelines. That is, exposure by a population to compounds detected in the surface water or sediments does not threaten human health or the environment. Only the subchronic and chronic hazard indices for the hypothetical case exceeded U.S. EPA's guidelines of one. However, a population is not currently exposed to the Columbia aquifer and although these calculated intakes exceed one, in reality, exposure to these intakes may never occur.

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**TABLE ES - 1  
SUMMARY OF CALCULATED CARCINOGENIC RISKS  
AND NONCARCINOGENIC HAZARD INDICES FOR  
THE NEW CASTLE SPILL SITE**

<b><u>PRESENT, EXISTING CONDITIONS*</u></b>		<b><u>Upper bound Reasonable Case</u></b>	<b><u>Upper Bound Worst Case</u></b>	<b>US EPA'S RECOMMENDED GUIDELINE</b>
<b>CARCINOGENIC RISK</b>		~ 0E+00	~ 0E+00	1E-04 to 1E-07
<b>NONCARCINOGENIC HAZARD INDEX</b>	<b>Subchronic</b>	1.45E-05	1.45E-05	1
	<b>Chronic</b>	3.76E-06	1.45E-04	1
<b><u>HYPOTHETICAL FUTURE-USE SCENARIO**</u></b>		<b><u>Upper bound Reasonable Case</u></b>	<b><u>Upper Bound Worst Case</u></b>	<b>US EPA'S RECOMMENDED GUIDELINE</b>
<b>CARCINOGENIC RISK</b>		3E-05	1E-04	1E-04 to 1E-07
<b>NONCARCINOGENIC HAZARD INDEX</b>	<b>Subchronic</b>	<b>6.71E+00</b>	<b>6.71E+00</b>	1
	<b>Chronic</b>	<b>8.69E+00</b>	<b>6.68E+01</b>	1

Bold value indicates an exceedance of US EPA's guideline

Upper bound reasonable case represents the average concentration detected times either the RfD or CPF.

Upper bound worst case represents the maximum concentration detected times either the RfD or CPF.

~ = approximate

\* - Surface water and soils exposure

\*\* - Ground water exposure

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The conclusions of the environmental assessment are that there is 1) no exceedance of ambient water quality criteria by measured surface water concentrations, 2) no exceedance of the toxicity value for the most sensitive aquatic species tested, 3) no imminent threat to the biotic component of the wetlands ecosystem from contaminants migrating off the New Castle Spill Site, and 4) low qualitative potential for bioaccumulation of these compounds in the aquatic species and less potential for biomagnification of the food chain to a human population.

## SECTION 1

### INTRODUCTION

#### 1.1 Objective of the Endangerment Assessment (EA)

The objective of this Endangerment Assessment (EA) is to evaluate the risks to human health and the environment under the No-Action Alternative at the New Castle Spill Site (NCSS). The EA evaluates the risks posed by compounds detected at the site under the present, existing conditions and the hypothetical future use of ground water.

The No-Action Alternative EA examines in detail the present, existing conditions scenario and recognizes the potential for those conditions to change in the future. This potential for change is examined by developing a future-use scenario. The future-use scenario examined in this EA is use of ground water from the Columbia aquifer as a potable water supply. This scenario is strictly hypothetical in that 1) the nearest human receptors use a municipal water supply, 2) the Potomac aquifer is the regional potable aquifer, and 3) although the Columbia aquifer is a GW-2B aquifer, it is not currently used for either domestic or municipal water supply. In order to assess this future-use scenario, a hypothetical well in the downgradient ground water flow direction of the Columbia aquifer will be used to assess the risks and/or to a potentially exposed population.

## 1.2 Site Description

The New Castle Spill Site (NCSS) is located at 900 Wilmington Road, New Castle, Delaware, and is a six acre parcel of land comprised of two adjacent properties: the Witco Chemical Corporation property and the New Castle Board of Water and Light (NCBW&L) property. The properties are located approximately 0.5 miles west of the Delaware River, within the city limits of the Town of New Castle, Delaware (Figure 1-1).

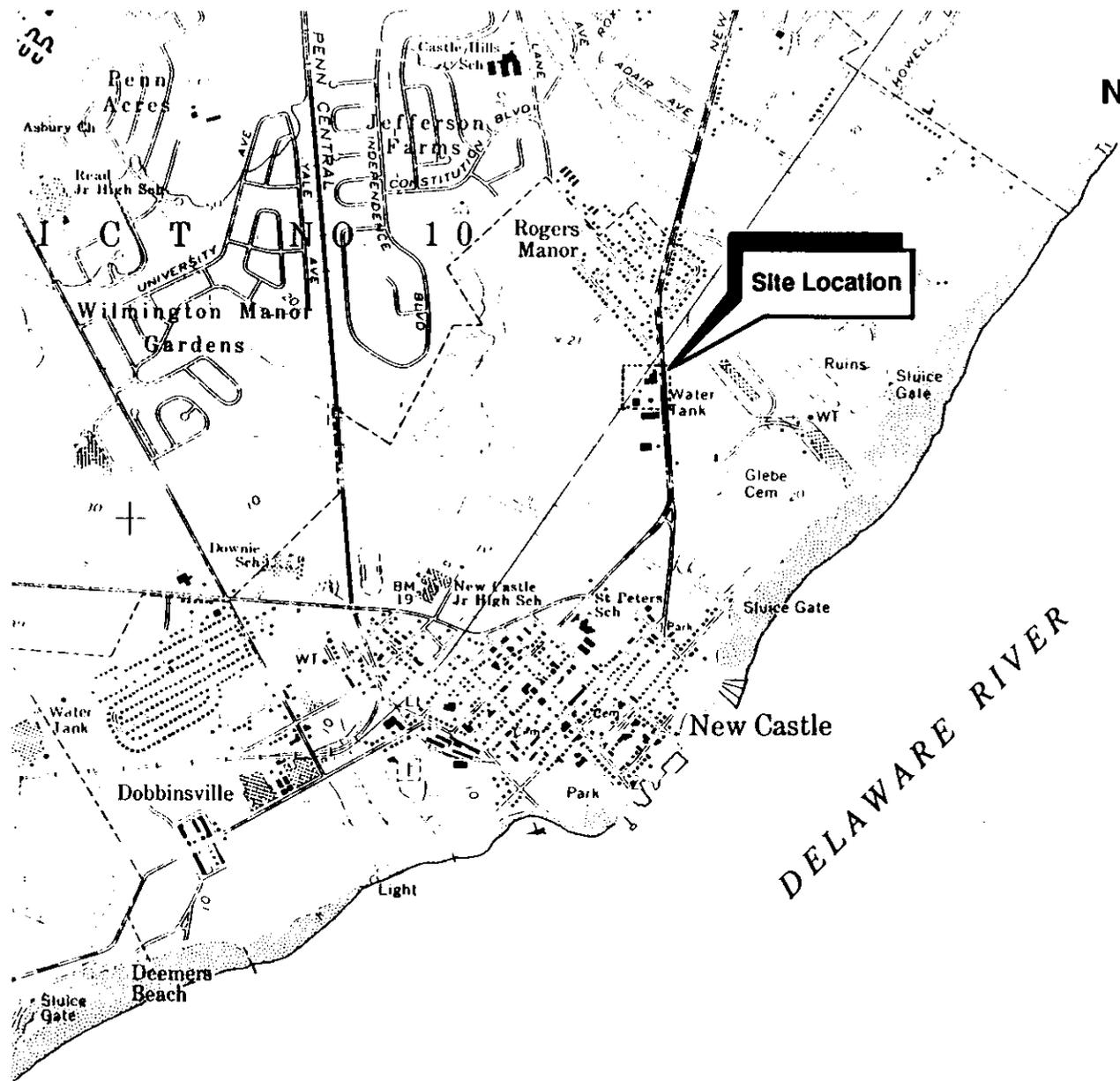
## 1.3 Site History

### 1.3.1 Background

The NCSS property was once used by Witco Corporation as a manufacturing plant, producing materials used in the production of plastic foams. The plant used prepolymers as feedstocks with two manufacturing processes taking place at the site. The largest process was a blending operation of polyether polyols with amine and/or tin catalysts, plus fluorocarbon-11, flame retardants, and silicone surfactants. The second process was the formation of a polymer from the reaction of a polyether polyol with diisocyanate.

The NCBW&L property was once used as a treatment facility, designed to process water extracted both from on-site production wells and an infiltration gallery. The gallery collected water from the Columbia aquifer, while the production wells pumped water from the underlying Potomac aquifer. Neither of these sources are currently being used by NCBW&L.

Figure 1-1  
Location Map  
New Castle Spill Site



Site Location

DELAWARE RIVER



0 1000 2000



Scale In Feet

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Source: USGS 7.5 Min. Topographic Quadrangle; Wilmington South, DE-NJ

### 1.3.2 Spill History

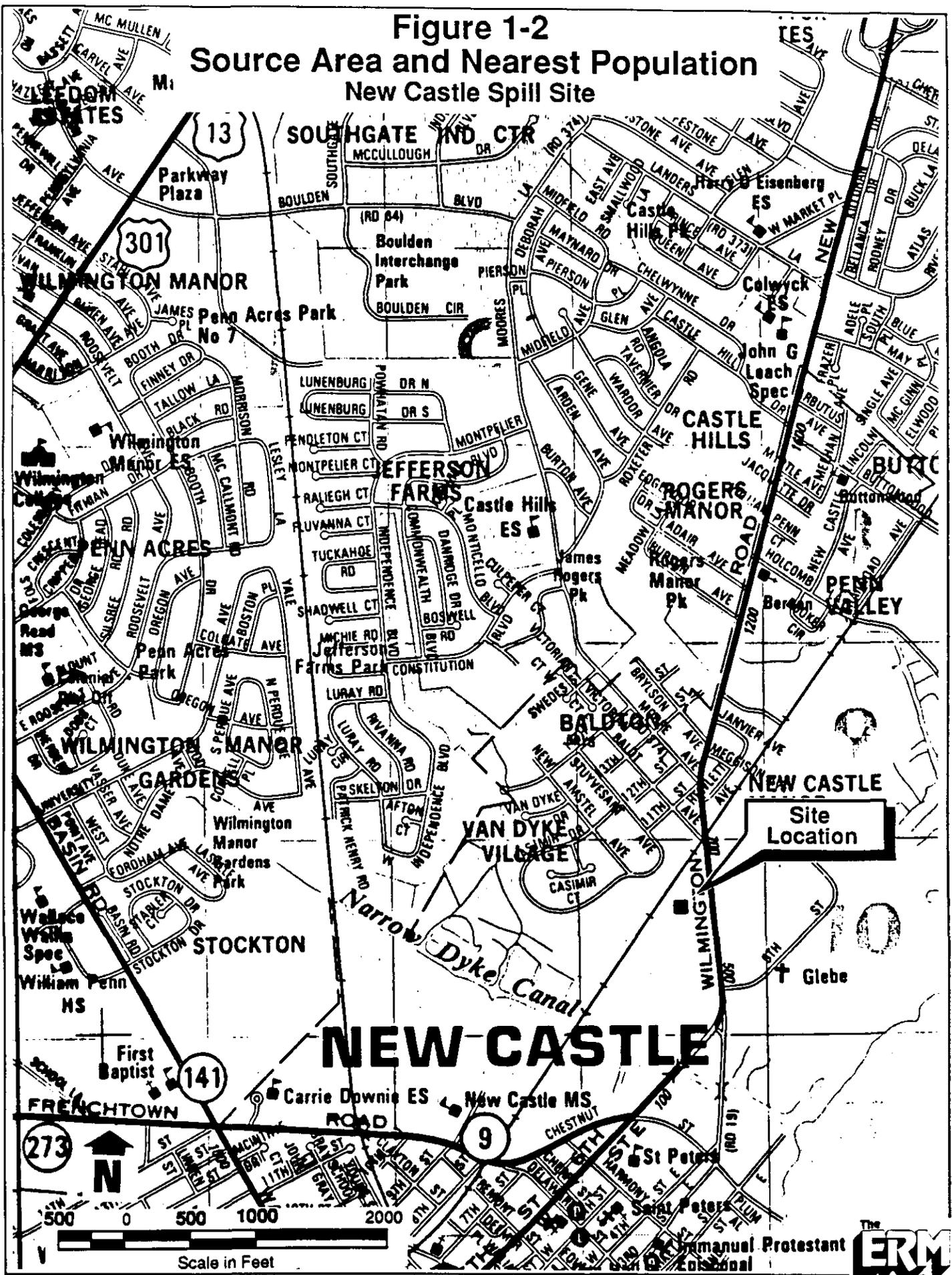
During the summer of 1977, a NCBW&L employee noticed a patch of dead grass on the NCBW&L property. This area of dead grass was located next to the drum storage area on the adjacent NCSS property. A subsequent investigation by Witco detected the presence of tris(2-chloropropyl)phosphate (tris) in the soils beneath the dead grass. The approximate location of the spill area and the nearest residential area are shown on Figure 1-2. The quantity of tris spilled is estimated to be 4-5 drums.

Shortly following the spill, ground water potentially contaminated with tris was pumped from the gallery system and discharged to the adjacent wetlands under the direction of the DNREC. Information provided by the NCBW&L indicated that water was pumped from the gallery system from 13 December 1977 through 31 May 1978 and that the capacity of the gallery system was 618,000 gallons per day (gpd).

### 1.3.3 Ground Water History

The impacted aquifer of concern (i.e., Columbia aquifer) does not supply residents with drinking or bathing water within a 3-mile radius of NCSS. It should be noted that since 1960 the water from this aquifer has had low pH and high iron and manganese levels. The Chicago Bridge and Iron Company is suspected as the source. This aquifer was documented to be of questionable quality prior to the leak at the NCSS facility and has not been used since 1978. The aquifer is currently not used, but is classified as useable (GW-2B).

**Figure 1-2**  
**Source Area and Nearest Population**  
**New Castle Spill Site**



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PW-11 is a municipal well located on-site. However, operation of this well was discontinued after the spill in 1977. The nearest operating water supply well is located approximately 0.7 miles downgradient of the site and is owned by Artesian Water Company. The population served by a municipal or commercial well located in the Columbia aquifer within a three mile radius of the site is zero. No public water wells exist in the Columbia aquifer in the vicinity of the site. The only wells located within a 3-mile radius of NCSS and completed in the Columbia aquifer are monitoring wells associated with other hydrogeological investigations or plant production wells.

#### 1.4 Physical Setting

##### 1.4.1 Demography

Growth patterns within the County of New Castle have followed trends similar to those observed within other areas of the northeast corridor. Since 1970, population trends have shown a 12 percent decrease in population within the City of Wilmington, and a 16.6 percent growth rate within the rest of the county. Since 1980, the average annual rate of population growth within the county has increased to 4,030 persons per year, as compared to 2,245 persons per year between 1970 and 1980. Along with an increased growth in population, the population density within the county has increased from the 1970 estimate of 730 persons per square mile to 851 persons per square mile in 1987. A comparison of census data from 1970 and 1980 indicates a general aging trend within the county and projections suggest this trend will continue.

Population projections from 1985 through 2010 show an anticipated 24.9 percent increase within the county by the year 2010. Population growth is contributed to two primary factors: the expansion of the greater Philadelphia area, and the large number of babies born to the baby-boom generation. Employment within New Castle County is primarily by the manufacturing, trade and service industries.

The Witco property and the City of New Castle are located within the New Castle planning district. The New Castle planning district is the third most populated district within New Castle County. The New Castle area has shown an 18.1 percent increase in population growth since 1970. Much of the growth, development and subsequent employment opportunities within the district may be a direct result of the location of major highways, such as I-95, I-495, I-295, US Route 13, US Route 301, US Route 40 and Delaware Route 273. Both I-95 and I-495 provide access via the Delaware Memorial Bridge to New Jersey. Additionally, the location of rail lines and the Greater Wilmington/New Castle County Airport has provided several opportunities for economic development.

#### 1.4.2 Land Use

The northern one-third of New Castle County, including the Greater Wilmington Area and its associated suburbs, is urbanized. The City of New Castle falls within this land use pattern. Large tracts of undeveloped land remain south of US Route 40 and Route 273. These tracts consist of open fields or swampy areas, or are used for agricultural purposes.

### 1.4.3 Climatology

The climate of New Castle County is typically warm and humid in the summer, and moderately cold in the winter. Annually, the average temperature ranges from a January low of 31.2°F, to a July high of 76°F. Average minimum and maximum temperatures during the period from 1951 to 1980, as recorded at the national Weather Service (NWS) station at Wilmington, Delaware (the closest NOAA weather station) are presented in Table 1-1.

The average annual precipitation for New Castle County, including both rainfall and the water equivalent of melting snow, is 41.38 inches. Precipitation normals during the period 1951 to 1980, as recorded at the NWS station at Wilmington are presented in Table 1-1.

Variations in temperature and precipitation do occur depending on location within the county. For example, of the four weather stations located within New Castle County, the weather station at Wilmington's Porter Reservoir exhibits the lowest average temperature, as well as the highest amount of precipitation (53.3°F and 44.9 inches, respectively). The most likely explanation for these differences may be the higher elevation (274 feet above sea level) of the Porter Reservoir Station as compared to the other weather stations, all of which are at elevations less than 100 feet above sea level.

### 1.4.4 Physiography

The town of New Castle is located in northern Delaware, within the Coastal Plain physiographic province (Figure 1-3). NCSS is

Table 1-1

Mean Monthly Precipitation and Temperature Data at the  
National Weather Service Station  
Wilmington, Delaware

MONTH	MEAN PRECIPITATION (inches)	MEAN TEMPERATURE (Fahrenheit)
January	3.11	31.2
February	2.99	33.2
March	3.87	41.8
April	3.39	52.4
May	3.23	62.2
June	3.51	71.2
July	3.90	76.0
August	4.03	74.8
September	3.59	67.9
October	2.89	56.3
November	3.33	45.6
December	3.54	35.5

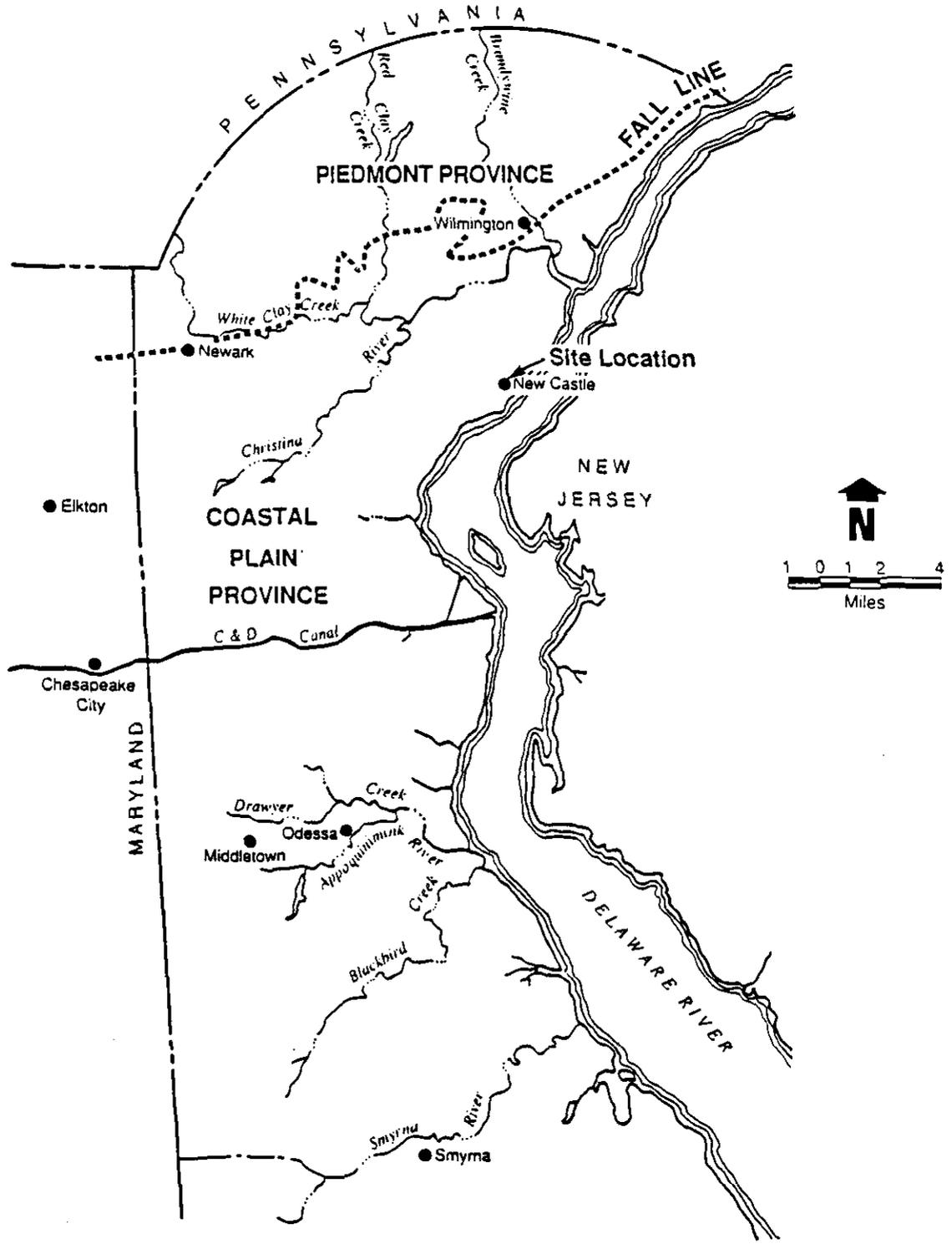
\*Monthly means are determined from climatological data from 1951 through 1980.

SOURCE: National Oceanic and Atmospheric Administration (NOAA)

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# Figure 1-3 Physiographic Province Map New Castle County, Delaware



Source: Modified From Sundstrom and Pickett, 1971.



relatively flat and is located within the Delaware River flood-plain. Elevations within the study area range from 0 to 10 feet above mean sea level. Surface water drainage from the site follows the gently dipping topography to the west-northwest and discharges to a marsh, which drains to the south and ultimately to the Delaware River.

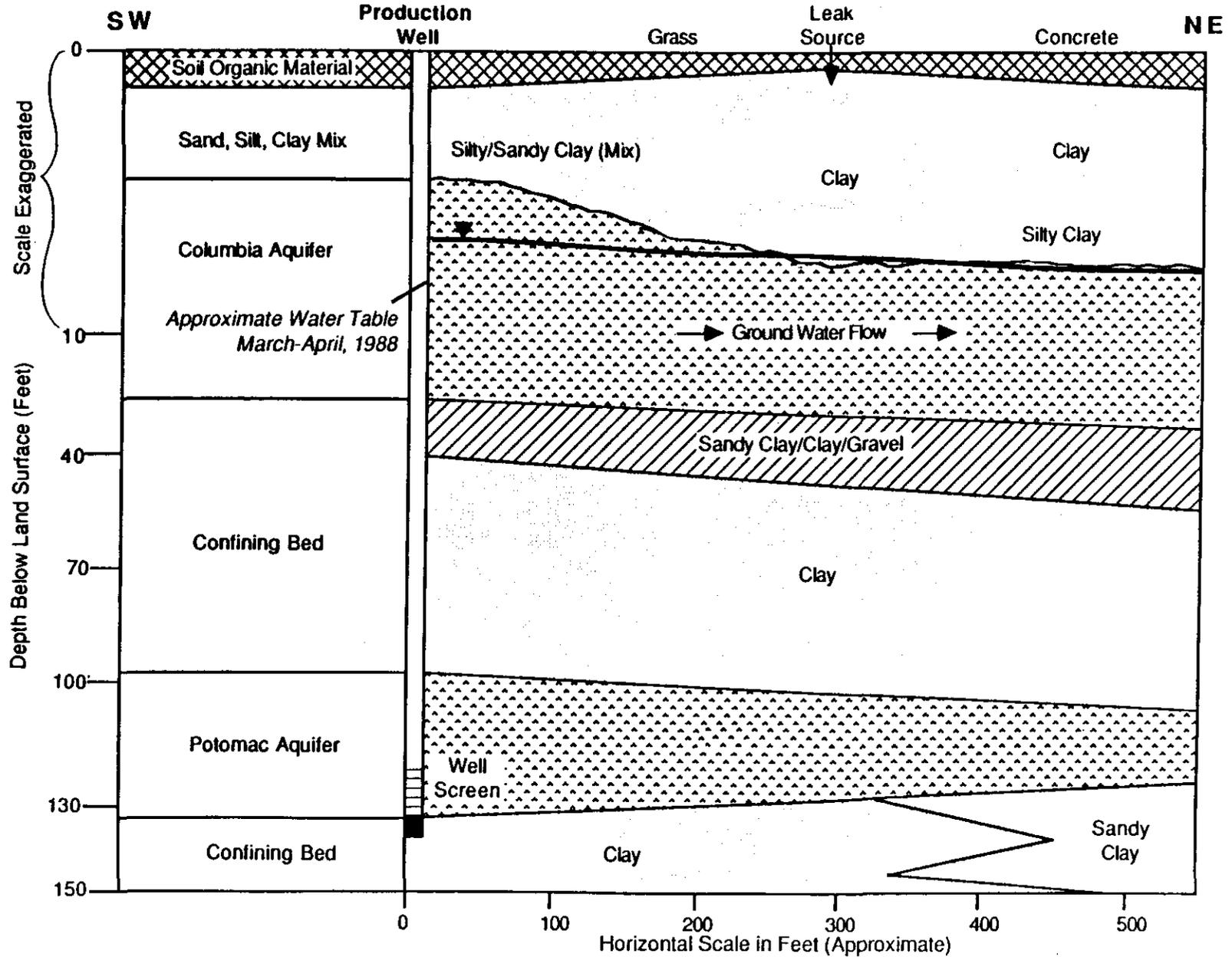
#### 1.4.5 Regional Geology

The study area is underlain by the Pleistocene age sands and gravels of the Columbia Formation (Figure 1-4), which occur in the form of thick channel fillings that form a wedge thickening in a southerly direction. This fluvial depositional formation reached a maximum thickness of 150 feet and covers most of the coastal plain province in Delaware.

Unconformably underlying the surficial deposits are the Cretaceous sands and gravels of the Potomac Formation. This formation consists primarily of discontinuous sand lenses. A thick clay, typical of this formation, is present beneath the NCSS between the Columbia and Potomac aquifers.

The Wilmington Complex, a mix of amphibolites, gabbros, banded gneisses, and some granites, underlies the Potomac Formation. Below this complex is the crystalline basement rock of the Wissahickon Formation which dips generally to the south-southeast at a rate of approximately 89 feet per mile.

### Figure 1-4 Conceptual Model of Hydrostratigraphy New Castle Spill Site



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#### 1.4.6 Regional Hydrogeology

There are two aquifer systems at the site: the shallow, or Columbia aquifer, and the deep, or Potomac aquifer. Deposits of the Columbia aquifer continually discharge to the freshwater non-tidal streams which drain the coastal plain deposits of the Delaware, and to the Delaware Bay and Atlantic Ocean. A tidal fluctuation study conducted by ERM showed no tidal effects upon ground water flow direction. Hydraulic conductivities in the Columbia aquifer range from 15 to 250 ft/day, while transmissivities range from 9,000 to 165,000 gal/day/ft. Storage coefficients range from 0.01 to 0.07. Ground water quality is generally classified as soft and slightly acidic, and typically has a low total dissolved solids (TDS) content.

Regionally, recharge to the underlying Potomac aquifer is generally within its subcrop area via vertical leakage from the overlying Columbia deposits. However, because of the thick clay deposits underlying the site, recharge to the Potomac aquifer in the vicinity of NCSS is not likely. Results of a pump test conducted by ERM in 1986 indicate that the aquifers are not connected in the area of NCSS. Discharge is primarily through pumping for both municipal and industrial purposes. Additional ground water discharge is to streams in the northern portion of the subcrop areas. Transmissivities of the Potomac aquifer range from 3,400 to 63,000 gal/day/ft. Ground water analyses of this aquifer indicate that the dissolved solids consist primarily of iron, sodium, calcium, chloride, and sulfate, with a pH ranging from 5.4 to 8.0.

## 1.5 Previous Investigations

The initial response action to the spill was taken by the DNREC during 1977 after the spill was reported. DNREC enlisted the U.S. EPA to assist in the identification of the presence and toxicity of tris in January 1978. At that time, tris was detected in ground water from the Columbia aquifer at three parts per billion (ppb), or less.

To date, a total of ten field investigations and eight summary reports have been completed for NCSS and the adjacent NCBW&L property. A preliminary assessment has also been conducted of the nearby Chicago Bridge and Iron property, located approximately 1,000 feet east of the NCSS. The documents produced from these investigations are detailed in Section 1 of the Remedial Investigation (RI) report (ERM, 1988).

### 1.5.1 Ground Water

In summary, the shallow ground water of the Columbia aquifer at the site has been sampled twelve times from 24 monitoring wells since 1978. Analyses from the shallow ground water sampling revealed concentrations of tris ranging from none detected to several thousand ug/l have been reported in the shallow aquifer. Shallow ground water has also been sampled for Priority Pollutant List (PPL) volatile organic compounds (VOCs) and semi-volatile organics, with the exception of acrolein, acrylonitrile, and tetrachloro- dibenzo-p-dioxins. Several compounds were reported in the first sampling, but were not found in subsequent samplings. These compounds included ethylbenzene, methylene chloride, tetrachloroethene, and toluene. Only two compounds,

trichlorofluoromethane and trichloroethene (TCE), were consistently reported at concentrations greater than trace or detection limits.

Since 1978, nine ground water samples have been collected from the Potomac aquifer (i.e., NCBW&L Production Well No. 11) with total VOC content ranging from below detection limit for most compounds to 41.1 ug/l of tetrachloroethene. Concentrations of tris in the Potomac aquifer collected in 1978 have been reported at 0.03 ug/l or less. bis(2-Ethylhexyl)phthalate was the only other semi-volatile compound detected, and ranged in concentration from below detection to 274 ug/l. A second sampling reported a none detectable concentration indicating a probable laboratory cross-contamination as the source of the 274 ug/l value.

#### 1.5.2 Soils

The soils associated with NCSS have been sampled three times since 1979. Results of soil samples collected to a depth of 12 feet, in and around the tris spill area, indicate that TCE, di-n-butyl phthalate, and toluene are present at trace levels, while reported concentrations of tris range from less than 50 ug/kg to over 200,000 ug/kg. The phthalate compound is considered suspect because it frequently occurs as a result of laboratory cross-contamination. PCBs, tetrachloroethene, and chloroform were also detected in one sample location.

### 1.5.3 Degree of Hydraulic Interconnection

The absence of a hydraulic connection between the shallow and deep aquifer systems at the site has been demonstrated in the Remedial Investigation Report by regional and site-specific stratigraphy and pump test and water quality data (ERM, 1988).

## 1.6 Environmental Resources Management's (ERM) Investigation

ERM conducted a field investigation at the NCSS during the Spring of 1988. The scope of that investigation and a brief summary of the results are presented in the following subsections.

### 1.6.1 Scope of Work

To provide the additional data necessary to satisfy data gaps, ERM proposed the following program of field activities:

- a well inventory to determine which on-site wells are functional;
- a tidal study to determine the effect, if any, of tidal fluctuations in the Delaware River on the direction and gradient of ground water flow;
- collection of 15 Phase I soil samples from 9 locations (3 soil borings, 5 monitoring wells, and one pumping well) for analysis of Target Compound List (TCL) parameters;

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- installation of five new 2-inch diameter monitoring wells and one 6-inch diameter pumping well to monitor ground water quality and to determine the permeabilities of the clays that separate the upper and lower aquifers;
- collection of Phase I ground water samples from 17 wells for analysis of TCL parameters, tris, Total Organic Carbon (TOC), Chemical Oxygen Demand (COD), pH, iron, and manganese;
- collection of three additional Phase II ground water samples, one submitted for TCE and the other for tris analysis. One additional soil sample was submitted for tris analysis;
- collection of Phase I surface water and sediment samples from 6 locations in the adjacent wetlands area for analysis of tris; and
- collection of Phase II surface water and sediment samples from the same 6 locations per DNREC comments on the Draft RI Report. Phase I surface water samples were analyzed for tris, TCE, and filtered and unfiltered fractions of iron and manganese. Sediment samples were analyzed for tris, TCE, grain size, total organic carbon, and percent moisture.

### 1.6.2 Summary of Analytical Results

Ground water quality data were gathered from 17 monitoring and observation wells between April 18 and 21, 1988 and selected wells on 22 June 1988. The major TCL compounds/constituents reported are presented below at their maximum levels:

Iron	19,400 ug/l
Manganese	5,230 ug/l
Tris	110,000 ug/l
TCE	120 ug/l
1,2-Dichloroethene	11 ug/l
Trichlorofluoromethane	950 ug/l

Surface water data were gathered from 6 sample locations. The maximum reported concentration of tris was 42 ug/l. This sample was collected on 14 March 1988.

Soil data were gathered from 15 soil samples collected from 9 monitoring well borings and soil borings, between March 7 and 18, 1988. The major TCL compounds reported and their maximum concentrations are as follows:

	<u>Concentration</u>	<u>Depth</u>
Tris	11.8 mg/kg	2-4 ft
Fluoranthene	3.6 mg/kg	2-4 ft
Phenanthrene	4.4 mg/kg	2-4 ft
Benzo(b)fluoranthene	2.5 mg/kg	2-4 ft

### 1.6.3 Conclusions of the RI

This section presents the conclusions from the Remedial Investigation for the New Castle Spill Site.

### Columbia Aquifer

The unconfined Columbia aquifer which underlies the New Castle Spill Site is composed primarily of a medium grained sand with an average transmissivity of 60,000 gal/day/ft and approximate saturated thickness of 23.5 feet. In the northern part of the study area, ground water flows in a northerly direction at a rate of 1.0 ft/day, while in the southern part of the study area, ground water flows in a westerly direction toward the marsh at a rate of 0.5 ft/day. Ground water within the study area is not tidally influenced.

The drilling program defined three distinct stratigraphic units across the study area: a surficial layer consisting of a variable sequence of clay, silty clay and silty sand; an intermediate layer (i.e., Columbia aquifer) consisting of medium grained sand; and a very dense, stiff clay layer at an average depth of 30 feet which designates the top of the underlying Potomac Formation. Vertical permeability test results ranged from  $1.48 \times 10^{-8}$  to  $4.83 \times 10^{-8}$  cm/sec. A minimum of 5-feet of this material was encountered in each of the newly installed wells and is considered to be continuous across the study area. Information gathered from other wells within the study area define this clay as the top of an 85-foot-thick sequence of clay, silty clay, silts and sands which serve to isolate the Columbia aquifer from the underlying Upper Potomac aquifer.

### Aquifer Interconnection

Both the Columbia and Upper Potomac aquifers are isolated by a sequence of clay, silty clay, silt and sand that are continuous

throughout the study area. The impermeable nature of this confining clay sequence is reflected in the five Shelby tube samples of this material which yield an average vertical permeability of  $2.87 \times 10^{-8}$  cm/sec. Under static ground water conditions, 160 years are required for the movement of ground water to a depth of 1-foot into this clay. Likewise, movement of ground water to a depth of 10-feet into the clay would require 1,600 years. Additional information supporting a lack of aquifer interconnection includes; pump test information, and water levels in the Upper Potomac aquifer.

The pump test of the upper Potomac aquifer, conducted in April-May 1986 yields data from well PH that indicates a typical confined response to pumping. Additionally, the storage coefficient calculated for the upper Potomac from this test (0.00011) is indicative of a confined system. A final line of evidence, with respect to the April-May pump test, is the stability of the water levels in the Columbia aquifer during the first 12 hours of the test, and prior to the recharge resulting from the ponding of discharge water on the surface. Stability of the water levels from those wells in close proximity to the pumping well (PW-11) demonstrate a lack of interconnection between the Columbia and Potomac aquifers.

The average depth to the top of the confining clay is approximately 30-feet BLS. As evidenced by depth-to-water measurements obtained from well PH, both recently and in 1986, the potentiometric surface of the upper Potomac aquifer extends approximately 15 feet above its confining layer. These artesian conditions are supportive of the clays continuity throughout the study area.

## Environmental Sampling

Several different media, including both on-site soils and ground water, in addition to sediments and surface water from the wetlands, were collected and analyzed as part of the remedial investigation.

### Soils

The occurrence and distribution of tris, which was detected in 9 of 15 soil samples at concentrations ranging from 54 to 11,740 ug/kg, reflects higher concentrations in those soils of the recognized spill source area. Within the spill source area, tris was detected to a depth of 8 feet. However, the mobility of tris is limited both by its preference to adsorb onto the soil matrix underlying the New Castle Spill Site, and by the fact that the area of highest tris concentration in the soils is presently capped by asphalt and concrete. Therefore, additional leaching of tris into the ground water from a "washing effect" by infiltrating rain water is significantly restricted.

TCE was conspicuously absent from all soil samples submitted for analysis as part of this study. It is therefore concluded that the presence of TCE in ground water originates from an upgradient and off-site source and therefore can not be attributed to past activities at the New Castle Spill Site

The trace and non-quantifiable concentrations of Polynuclear Aromatic Hydrocarbons (PAH's) in soils of the spill source area had a tendency to decrease with depth and are likely derived from asphalt paving.

### Ground Water

Detectable and quantifiable concentrations of tris, ranging from 17.1 to 110,000 ug/l, were identified in 7 of 17 wells sampled. The distribution of tris in the Columbia aquifer is consistent with the spill source area, and reflects a reduced mobility by its occurrence primarily in the upper 10-feet of the aquifer. This is evidenced by higher tris concentrations in the "OB" series wells, screened at the top of the Columbia aquifer, in contrast to the "MW" series wells, screened at the base of the same aquifer. In addition to tris, TCE was the other predominant compound identified in the 17 ground water samples collected.

The distribution of TCE, which was detected in 8 of 17 samples, ranged in concentration from 1 to 120 ug/l. As discussed in Section 5.3.1 of the R.I. Report, the absence of this compound in the soil samples submitted for analysis indicate an upgradient and off-site source for TCE. The occurrence and distribution of TCE in the ground water samples suggests that this off-site source may exist either to the south or east of the New Castle Spill Site.

### Wetlands

The NCSS is bordered to the west by wetlands that support a diverse flora and associated wildlife community. Samples collected from within the wetlands possessed quantifiable concentrations of tris ranging from none-detected to 42 ug/l in surface water while wetlands sediments yielded results of

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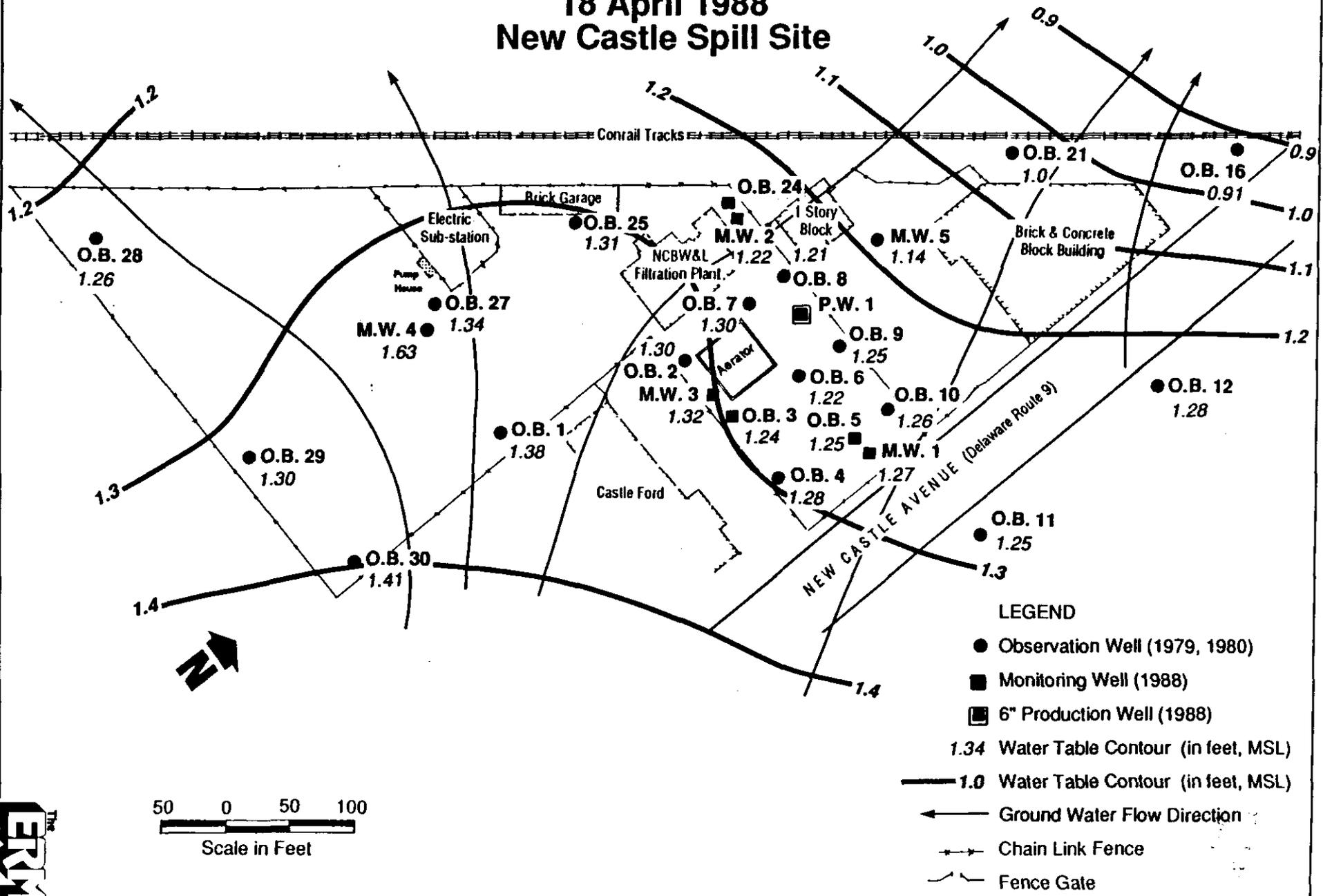
none-detected. Confirmatory sampling conducted in June 1988 yielded order-of-magnitude lower results for surface water, while two sediment samples contained quantifiable tris concentrations of 300 and 402 ug/kg. However, based on investigations conducted as part of this study, it is concluded that potential receptors dwelling within the wetland, such as macroinvertebrates, fish, birds and mammals, are not affected by the New Castle Spill Site.

An investigation of DNREC files to identify potential users of groundwater from the Columbia aquifer within a 2-mile radius north of the New Castle Spill Site, and 1-mile south of the New Castle Spill Site indicate that there are no withdrawals of ground water from the Columbia for either domestic or municipal purposes.

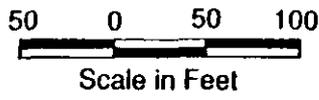
### 1.7 Conceptual Models

The conceptual models describe the flow of ground water below the site in terms of stratigraphy and direction (Figures 1-4 and 1-5). Two aquifers underlie the site: the Columbia and the Potomac. The Columbia aquifer is unconfined and the top of the water table occurred at 5 to 7 feet below grade. The ground water flow velocity of the Columbia aquifer was measured to be 1.0 ft/day with ground water flow direction in the northern part of the site to the north-northwest. In the southern portion of the site, ground water flows to the west at a velocity of 0.5 ft/day (ERM 1989). Within the study area, the direction of a ground water flow in the Potomac aquifer is unknown. Martin (1984) estimates the hydraulic conductivity of the Potomac aquifer to be 25 ft/day. There is no measurable recharge of the Potomac by the Columbia at

**Figure 1-5**  
**Water Table Contour Map, Columbia Aquifer**  
**18 April 1988**  
**New Castle Spill Site**



- LEGEND**
- Observation Well (1979, 1980)
  - Monitoring Well (1988)
  - ▣ 6" Production Well (1988)
  - 1.34 Water Table Contour (in feet, MSL)
  - 1.0 Water Table Contour (in feet, MSL)
  - ← Ground Water Flow Direction
  - +— Chain Link Fence
  - +— Fence Gate



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the site, as demonstrated by the Theis drawdown curves. The Potomac aquifer is confined by two clay beds, the lower being a sandy clay. The Potomac aquifer is currently being used as a drinking water supply.

In the southern portion of the site, the ground water flow direction is to the west and is a possible route for migration of compounds from the site to the marsh, where mixing with surface water might occur. This is made possible by the high water table, which can mobilize water soluble compounds at only a few feet below land surface.

The main factor preventing the mobilization of tris or other compounds, is the high clay content of the soil above the Columbia aquifer. The clay and silty sand matrix promotes sorption of compounds onto soil and prevents them from leaching into the ground water.

#### 1.8 Format of the EA

The format of this report includes the following sections:

- Methodology;
- Indicator Compounds;
- Exposure Assessment;
- Toxicity Evaluation;
- Risk Characterization; and
- Conclusions.

## SECTION 2

### METHODOLOGY

#### 2.1 EPA's Endangerment Assessment Process for CERCLA Sites

This section provides a broad overview of the CERCLA Endangerment Assessment (EA) process. The discussion is not intended to be a comprehensive guide to preparing risk assessments. EPA has proposed guidelines for the preparation of EAs in the Endangerment Assessment Handbook (US EPA, 1985a), Superfund Public Health Evaluation Manual (US EPA, 1986a), Exposure Assessment Handbook (US EPA, 1988), and Toxicology Handbook (US EPA, 1986b).

An EA is normally conducted after the completion of a Remedial Investigation (RI). The RI determines the nature and extent of contamination at a site, and its results form the data base from which potential exposures can be determined and risks assessed. In addition, the RI defines whether or not the present conditions at the site are at steady state.

There are four evaluations which must be completed in a CERCLA EA:

1. Identification of indicator compounds, which are used to represent the potential risks posed by carcinogenic and noncarcinogenic compounds at the site;

2. Exposure evaluation, which includes the calculation of doses to potentially exposed human and/or non-human populations;
3. Toxicity evaluation of the potential carcinogenicity and noncarcinogenic effects of site indicator compounds; and
4. Characterization of the risks to human populations and/or the environment.

## 2.2 Indicator Compounds

For the purpose of an endangerment assessment, indicator compounds are selected on a site-specific basis. These are generally the compounds that are most prevalent and represent the majority of risk posed by the site.

The selection and ranking of indicator chemicals follows the procedure outlined in the Superfund Public Health Evaluation Manual (US EPA, 1986a). As part of the indicator compound selection process, toxicological information about each compound was compiled using Appendix C of the Superfund Public Health Evaluation Manual (US EPA, 1986a). A range and representative concentration for each compound was calculated for each appropriate medium. This information includes the following:

1. toxicologic class: potential carcinogens (PC) or noncarcinogens (NC);
2. severity-of-effect ratings value for noncarcinogens;

3. weight-of-evidence ratings for carcinogens; and
4. toxicity constants for the various environmental media.

Data used in the selection of indicator chemicals were subjected to comprehensive quality assurance and quality control review. Cambridge Analytical, Inc. analyzed the samples from the the New Castle Spill Site Operable Unit RI. ERM's quality control and quality assurance procedures, including chain of custody documentation, split samples, replicate analyses, sample spiking with an internal standard, routine instrument calibration, methodology (extraction) blanks, and adherence to recommended sample holding times and storage temperatures, were also implemented.

The site-related compounds identified at the New Castle Spill Site were subdivided into potential carcinogens and noncarcinogens. An indicator score (IS), which is the product (CT) of the compound concentration (C) and the toxicity constant (T), was calculated for each medium and then summed to yield a total indicator score per compound. The compounds were then ranked numerically based upon decreasing indicator scores. The top-scoring compounds (based on IS values) were then re-evaluated based upon frequency of detection, water solubility, vapor pressure, Henry's law constant, and soil organic carbon partition coefficient ( $K_{OC}$ ) to determine the final indicator compounds. This re-evaluation has a direct relationship to the IS value but selectively eliminates those compounds which are degradation products, have similar physical or chemical properties, or have comparable half-lives in the various environmental media.

### 2.3 Exposure Evaluation

The purpose of an exposure evaluation is to determine the possible intake of each indicator compound by a potentially exposed population and/or the environment. The modes of contaminant transport, leading from the sources on the site to a point of potential exposure, are defined. Concentrations of the indicator compounds are determined in each medium at a point which a population may be exposed (i.e., exposure point concentration). A potentially exposed population is then defined, and possible doses are determined. Finally, the possible intake resulting from the potential exposure is calculated.

The sources of contamination at the site are given in the RI. The exposure evaluation determines the migration of contaminants from the site to potentially exposed populations through the following tasks:

- Evaluating fate and transport processes for the indicator compounds;
- Establishing exposure scenarios for each medium;
- Determining possible exposures to potentially affected populations; and,
- Calculating doses and resultant intakes.

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### 2.3.1 Evaluating Fate and Transport Processes for the Indicator Compounds

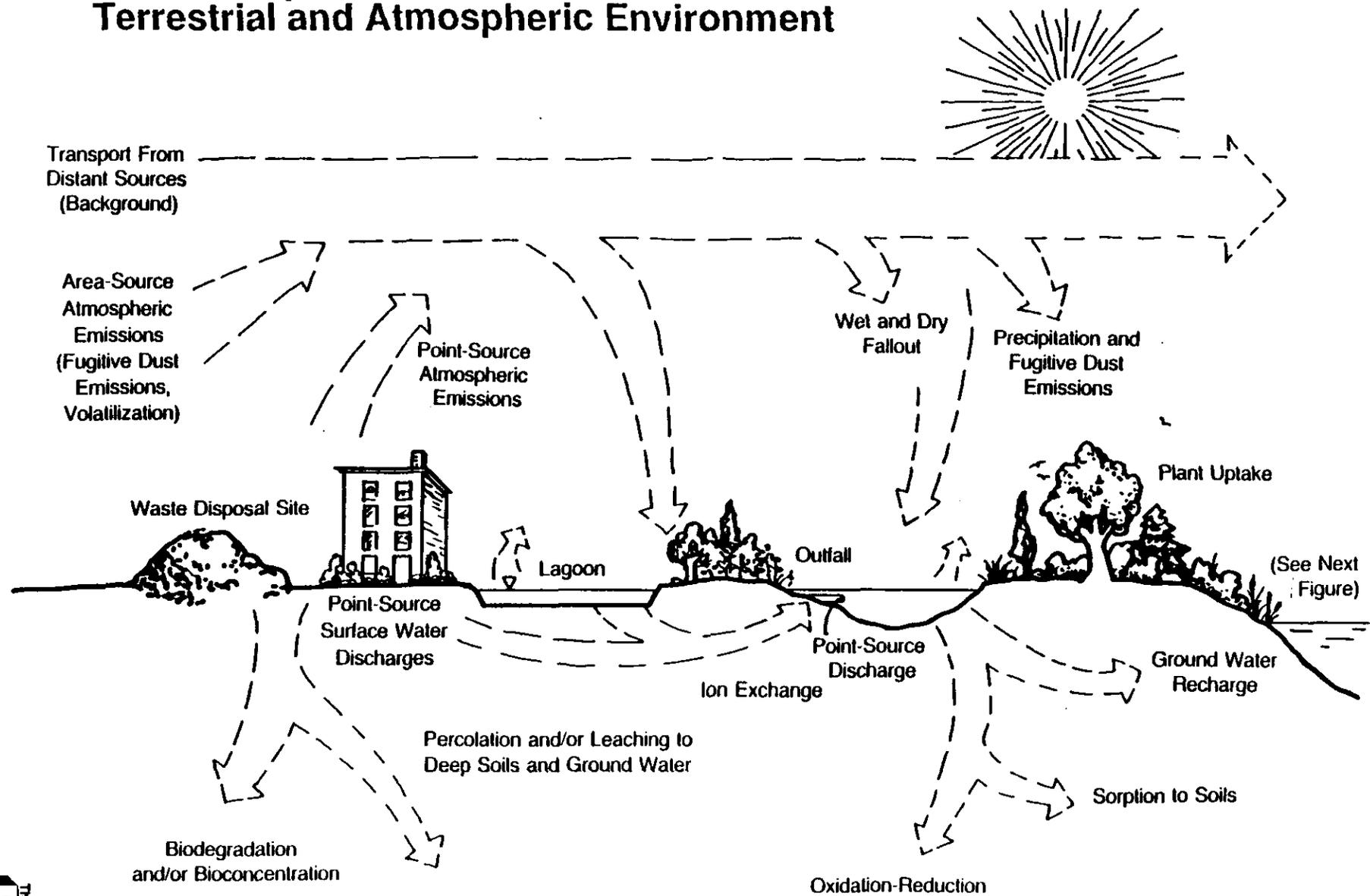
The first step in the analysis of exposure is to evaluate the important fate and transport processes for the indicator compounds in a qualitative manner. This is done so that the potential for releases from sources of contamination can be evaluated. This analysis can also identify any significant intermedia transport routes that may need to be evaluated in detail later, in fate and transport modeling. Examples of the fate and transport processes of compounds in the terrestrial, atmospheric, and aquatic environments are presented in Figures 2-1 and 2-2.

Examples of the environmental fates of the indicator compounds include sorption by soils and sediments, volatilization into the atmosphere, photochemical degradation, and bioaccumulation. Physical and chemical constants such as solubility and octanol-water partition coefficients are tabulated so that their importance in affecting fate and mobility of the contaminants can be evaluated.

### 2.3.2 Establishing Exposure Scenarios for Each Medium

An exposure scenario qualitatively establishes the connection between a source of a contaminant and a human population. The mode of exposure to the population, such as inhalation, ingestion, or dermal contact, is identified as part of the exposure scenarios. Exposure scenarios are determined by integrating information from the RI with knowledge about potentially exposed populations and their likely behavior.

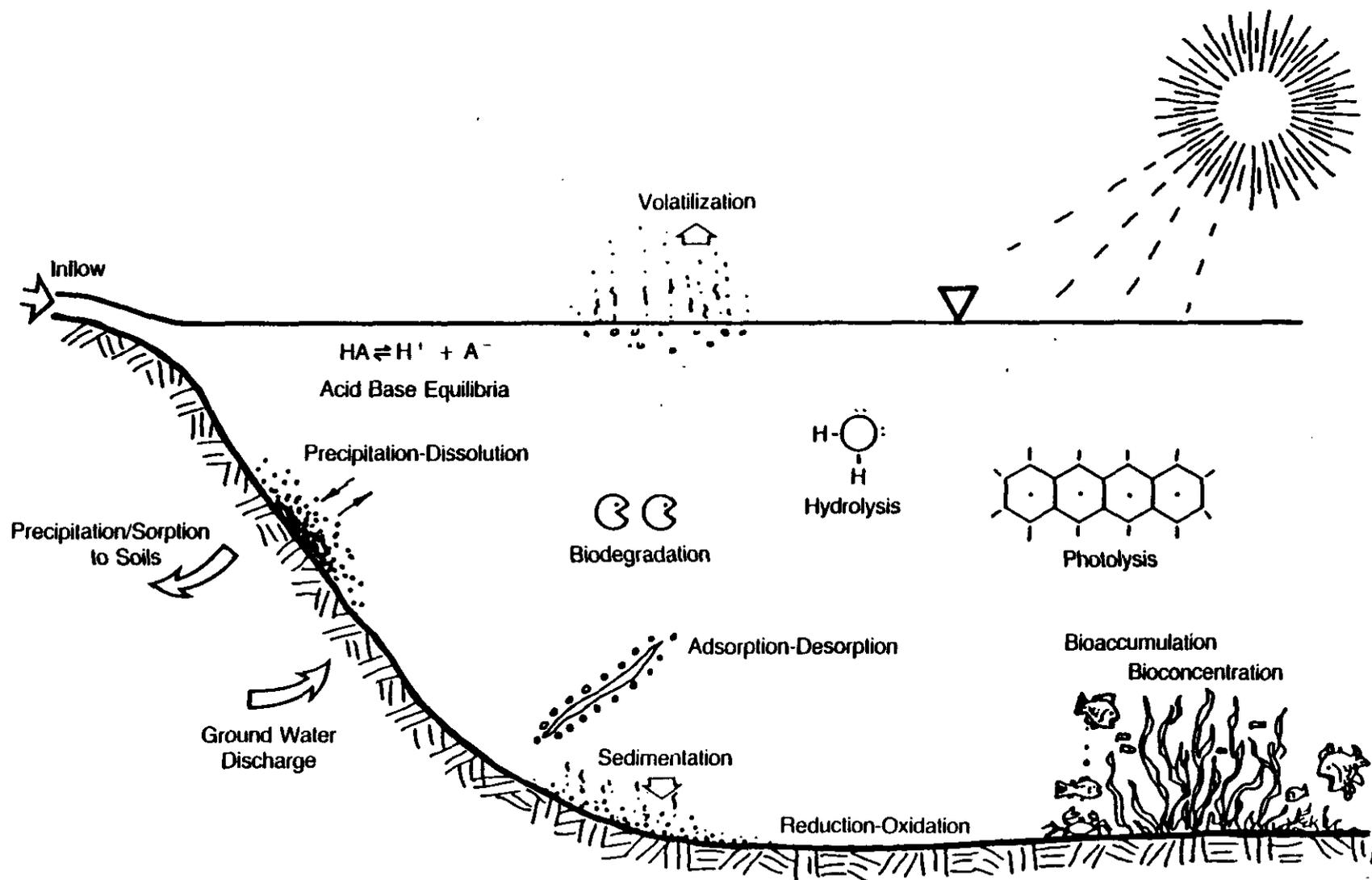
# Figure 2-1 Fate and Transport Processes of Chemicals in the Terrestrial and Atmospheric Environment



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**Figure 2-2**  
**Fate and Transport Processes of**  
**Chemicals in the Aquatic Environment**



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### 2.3.3 Determining Exposures to Potentially Affected Populations

The next step is the quantitative determination of the exposure concentrations at the potential points of contact with human populations. This step may be quite complicated; it requires knowledge of the contaminant source and its behavior in, and effect on, the environment between the site and any potentially exposed populations. The exposed populations for each medium may also be different, as would be the case if the direction of ground water flow were opposite to that of the prevailing wind.

If the transporting medium can be treated as steady-state, monitoring data may be used to quantify exposure concentrations. If no data are available or if transient, increasing concentrations are suspected, models may be used to predict concentrations.

Ground water contaminant transport through advection and dispersion is normally described in the RI. Transport in such other media as surface water and the atmosphere is not normally evaluated in the RI, and modeling assessments are often required to determine exposures. Many factors, including the fate processes reviewed previously, are considered when selecting the most appropriate model.

### 2.3.4 Calculation of Doses to and Possible Intakes by Potentially Exposed Populations

Once exposure concentrations in all media have been determined, the resultant doses and intakes to potentially exposed

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populations are calculated. Dose is defined as the amount of compound contacting body boundaries (skin, lungs, or gastrointestinal tract), and intake is the amount of chemical absorbed by the body. To calculate dose and intake, several factors must be considered:

- the amount of contaminated medium that contacts internal or external body surface during each exposure event;
- the amount of contaminant absorbed during each exposure event; and
- the frequency of each exposure event.

Doses and intakes are normally calculated together since they are very similar. Short-term (maximum) and long-term (average) doses are calculated in the same manner. First, for each exposure pathway under consideration, a dose per event is developed. This value quantifies the amount of contaminant contacted during each exposure event. "Event" may have different meanings depending on the nature of the scenario under consideration (e.g., each day's inhalation of contaminated air constitutes an inhalation exposure event). The quantity of contaminant absorbed per event (intake) is calculated by considering the concentration of contaminant in the medium in which exposure occurs, the rate of contact with the medium (inhalation rate, ingestion rate, etc.), and the duration of each event.

Event-based intake values are converted to final intake values by multiplying the dose per event by the frequency of exposure events over the time frame being considered. Subchronic (short-term) exposure is based on the number of exposure events

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that occur during the short-term time frame using maximum contaminant concentrations in the media to define dosage. Subchronic exposure values are intended to represent 10- to 90-day exposures. Chronic (long-term) exposures are based on the number of events that occur within an assumed 70-year lifetime using average contaminant concentrations in the media to define dosage.

Estimates of daily intakes of contaminants are necessary to assess risk. Daily intake estimates are expressed in terms of mass of contaminant per unit of body mass per day. They are derived by dividing average daily exposures by an appropriate average body mass--a 70 kg adult, for example.

For carcinogens, the CDI values are used to assess carcinogenic risk. For compounds with noncarcinogenic effects, both SDIs and CDIs are used to evaluate acute and chronic effects.

In the New Castle Spill Site assessment, three routes of exposure are applicable: ingestion of contaminants, inhalation of volatilized contaminants, and dermal exposure to contaminants. Calculations were made for each exposure mechanism according to the Superfund Exposure Manual (US EPA, 1988).

#### 2.3.4.1 Inhalation Exposure

Potential inhalation intakes are estimated based on the number of hours in each event, the inhalation rate of the exposed individual during the event, and the concentration of contaminant in the air breathed. The formula for calculating event-based dosage is:

$$\text{IEX} = \text{D} \times \text{I} \times \text{C} \times \text{RF},$$

where

IEX = estimated inhalation intake (mass of contaminant per event)

D = duration of an exposure event (hours per event)

I = average inhalation rate of exposed persons (cubic meters per hour)

C = contaminant air concentration throughout the exposure period (milligrams per cubic meter of contaminated air)

RF= retention factor of inhaled compound, i.e., the fraction of the inhaled concentration that is absorbed into the bloodstream (assumed to be an average of 0.50 for most compounds)

Subchronic (short-term) exposure resulting from inhalation is calculated using the maximum contaminant air concentration. Chronic (long-term) exposure is based on the average concentration.

#### 2.3.4.2 Dermal Exposure

Dermal intake is determined by the concentration of hazardous substance in a contaminated medium that is contacted, the body surface area contacted, the duration of the contact, the flux,

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and the absorbed fraction. For exposure to contaminated water, dermal intake per event is calculated as follows:

$$DEX = D \times A \times C \times \text{Flux} \times \text{ABS}$$

where

DEX = estimated dermal intake per event (mass of contaminant per event)

D = duration of an exposure event (hours per event)

A = skin surface area available for contact (cm<sup>2</sup>)

C = contaminant concentration in water (weight fraction)

Flux = flux rate of water across skin (mass/cm<sup>2</sup>/hr)

ABS = amount of contaminant absorbed

For exposure to contaminated soil, dermal intake per event is calculated as:

$$DEX = f_w \times A \times \text{DA} \times f_a \times M$$

where

f<sub>w</sub> = weight fraction of chemical substance in soil (unitless)

A = skin surface area exposed per event (cm<sup>2</sup>/event)

DA = dust adherence (mg/cm<sup>2</sup>)

$f_a$  = the fraction of a chemical absorbed through the skin

M = the degree to which the soil matrix impedes absorption, expressed as a percentage.

Possible subchronic intake resulting from each dermal exposure event is calculated using the maximum (short-term) contaminant concentrations in water. Chronic intake is based on average (long-term) contaminant concentrations.

#### 2.3.4.3 Ingestion Exposure

Potential intake resulting from ingestion of soil-borne or water-borne contaminants is determined by multiplying the concentration of the contaminant in the soil or water by the amount of soil or water ingested per day and the degree of absorption (assumed to be one hundred percent).

#### 2.4 Toxicity Evaluation

The selected indicator compounds are subjected to toxicity evaluation to develop a data base of quantitative toxicity indices against which intakes can be compared during the risk characterization evaluation.

This evaluation presents summaries of health effects data, pharmacokinetics and metabolism, toxic and carcinogenic effects, and applicable and relevant standards available for the indicator chemicals. Because of its major impact on the risk evaluation,

the procedures used for classifying animal and human carcinogens by both the EPA and the International Agency for Research on Cancer (IARC) of the World Health Organization, and the attendant uncertainties, will be presented.

Evaluations of carcinogenicity basically involve two steps: (1) the identification of potential carcinogens among the contaminants present at the site, and (2) the quantitative determination of their carcinogenic potency.

Evidence of possible carcinogenicity in humans comes primarily from long-term animal tests and epidemiological investigations. Results from these studies are supplemented with information from short-term tests, pharmacokinetic studies, comparative metabolism studies, structure-activity relationships, and other relevant information sources.

For judging the qualitative evidence of carcinogenicity, EPA and IARC have adopted a policy of "weight-of-evidence", meaning that the quality and adequacy of all relevant data on responses induced by a possible carcinogen using different procedures will be considered. There are three major steps in determining the weight-of-evidence for carcinogenicity:

1. Characterization of the evidence from human studies and from animal studies individually,
2. Combination of the two types of studies into a final indication of overall weight-of-evidence for human carcinogenicity, and

3. Evaluation of all supportive information to determine if the overall weight-of-evidence should be modified.

Further details concerning the classification systems of EPA and IARC, and on the use of these data in the endangerment assessment process are presented in Appendix A.

The second phase in carcinogen assessment involves the quantification of risk. Experimental studies of carcinogenic effects that utilize the low exposure levels usually encountered in the environment generally are not feasible. Therefore, various mathematical models have to be used for extrapolation from the high doses used in animal bioassays down to the doses involved with exposure to ambient environmental concentrations. Since the resolution power of animal studies, for example, is not adequate for precise elaboration of the dose-response curve, extrapolating from a high dose to a low dose introduces a level of uncertainty which may amount to orders of magnitude. Given the recognized differences in carcinogenic response between species, and between strains of the same species, it is clear that additional uncertainties will be introduced when quantitative extrapolations, as from rodents to humans, are made. Of the various proposed models for quantitative extrapolation, EPA recommends a linearized multistage model: "In the absence of adequate information to the contrary, the linearized multistage model will be employed" (Federal Register, Guidelines for Carcinogen Risk Assessment, 24 September 1986). The linearized multistage model assumes linearity at low doses. Alternative models do not assume a linear relationship and in general are much less conservative. There is no biologically sound basis for choosing one model over another. However, when applied to the same data, the various models can produce a wide range of risk

estimates; the model recommended by EPA usually produces the highest estimates of risk. Moreover, this model does not provide a best estimate of risk, but rather an upper-bound probability that the risk will be less than the calculated value 95 percent of the time.

## 2.5 Risk Characterization

The risks to potentially exposed populations from exposure and subsequent intakes of the indicator compounds are characterized in three tasks:

1. Comparison with potentially Applicable or Relevant and Appropriate Requirements (ARARs),
2. Calculation of Noncarcinogenic Hazard Indices, and
3. Calculation of Carcinogenic Risk.

### 2.5.1 Comparison with Potentially Applicable or Relevant and Appropriate Requirements

In this section, the exposure point concentrations of all contaminants are compared to potentially applicable or relevant and appropriate requirements (ARARs) standards as defined by the National Contingency Plan (NCP). At present, EPA considers drinking water maximum contaminant levels (MCLs), national ambient air quality standards (NAAQS), and federally approved state water quality standards developed under the Clean Water Act to be potentially applicable or relevant and appropriate requirements.

### 2.5.2 Calculation of Noncarcinogenic Hazard Index

The Hazard Index method is used for assessing the overall potential for noncarcinogenic effects posed by multiple chemicals. The Hazard Index calculates a safety margin, a factor by which the acceptable intake exceeds the estimated exposure level. This approach assumes that multiple subthreshold exposures could result in an adverse effect and that the magnitude of the adverse effect will be proportional to the sum of the ratios of the subthreshold exposures to acceptable exposures. This relationship can be expressed as:

$$\text{Hazard Index} = E_1/AL_1 + E_2/AL_2 + \dots + E_i/AL_i,$$

where:

$E_i$  = Exposure level (or intake) for the  $i^{\text{th}}$  contaminant

$AL_i$  = Acceptable level (or intake) for the  $i^{\text{th}}$  contaminant

For a single contaminant, there may be a potential adverse health effect when the hazard index exceeds unity, although because the "acceptable level" itself incorporates a large margin of safety (safety factor), it is possible that no toxic effects may occur even if the "acceptable level" is exceeded. For multiple chemical exposures, hazard indices, if summed, may result in an overall hazard index that exceeds one even if no single chemical exceeds its acceptable level. However, the assumption of additivity should be made only for compounds that produce the same toxic effect by the same mechanisms of action.

US EPA has developed some preliminary information regarding Acceptable Intakes for Subchronic Exposures (AISs) and Reference Doses (RfD) (US EPA, 1986a). Where these are available, they are used as acceptable levels for subchronic and chronic exposures, respectively. When unavailable, these intakes may be calculated using approved US EPA methodology from well-designed and conducted toxicology studies on experimental animals.

### 2.5.3 Calculation of Carcinogenic Risk

For potential carcinogens, risks are estimated as probabilities. The carcinogenic potency factor, which is the upper 95% confidence limit of the probability of a carcinogenic response per unit intake over a lifetime of exposure, converts estimated CDIs directly to incremental risk values. This is not the only methodology to calculate risks, but it is likely to be an upper bound. In general, because only relatively low CDIs are likely to result from environmental exposures, the EPA methodology assumes that the exposure will be in the linear portion of the dose-response curve. Based on this assumption, the slope of the dose-response curve is equivalent to the carcinogenic potency factor, and the risk is directly proportional to the CDI at low levels of exposure. The low-dose carcinogenic risk equation is:

$$\text{Risk} = \text{CDI} \times \text{Carcinogenic Potency Factor (CPF)}$$

The carcinogenic risks posed by each carcinogen are summed for each receptor population (i.e., children age 2-6, children age 6-12, and adults). The carcinogenic risk for each receptor population is then weighted, and finally, the weighted results are added to yield a lifetime weighted carcinogenic risk.

## 2.6 Uncertainty

US EPA employs a great deal of conservatism in the process that it proposes to describe human cancer risks. US EPA gives animal test evidence stronger weight than IARC does in determining the strength of evidence that a substance is a human carcinogen. In this process, US EPA includes benign liver tumors in its estimations. Additionally, US EPA suggests the use of the linear multistage dose-response model to predict human cancer risk at low doses. This model is more conservative than other standard dose-response models. It forces linearity on the dose-response curve, even if the experimental data are clearly nonlinear, and it uses statistical upper confidence limits on risk rather than most likely estimates. Furthermore, US EPA's procedure for interspecies extrapolation uses a very conservative procedure based on relative body surface area rather than body weight.

US EPA is conservative in calculating intakes over a lifetime, for instance, in determining the amount of air that is breathed or water consumed. US EPA is also conservative in estimating the amount of chemical absorbed by the body. These combined factors lead to a very high level of conservatism in the overall process, with the effect of overestimating risks. The scenarios developed as part of this assessment for the New Castle Spill Site EA follow this philosophy as well, leading to a probable overall overestimation of risk.

## SECTION 3

### INDICATOR COMPOUNDS

#### 3.1 Selection of Contaminants of Concern

The numerous contaminants identified in the Remedial Investigation are composed of a diverse group of compounds with widely disparate physicochemical, environmental, and toxicological properties. The extent of contamination widely varied in concentration and occurrence throughout the New Castle Spill Site. Thus, some contaminants represent a greater potential risk to human health or the environment than others because of differences in toxicity, capacity to migrate to receptors, and likelihood of exposure concentrations at levels high enough to pose human health risks. It is neither necessary nor practical to evaluate all contaminants in terms of transport, exposure, and attendant health or environmental risk in order to effectively address endangerment. Rather, a selective identification of contaminants of concern is undertaken to focus effort on a limited set of compounds which represent the major hazards associated with a particular site. Selection of indicator compounds was performed in accordance to procedures outlined in the Endangerment Assessment Manual (US EPA, 1985) and described in detail in the Superfund Public Health Evaluation Manual (US EPA, 1986a). The detailed methodology of this process was presented in Section 2.

### 3.2 Indicator Compound Selection

During the indicator compound selection process, a maximum and average concentration was determined for each chemical evaluated. Toxicological information consisting of potential carcinogenic (PC) or noncarcinogenic (NC) classification, and toxicity constants for water, soil, and air were obtained from Appendix C of the Superfund Public Health Evaluation Manual (U.S. EPA, 1986a). The five worksheets used to develop CTs (media concentrations (C) multiplied by toxicity constants (TCs) and indicator score (IS) values are presented in Appendix B and described as follows.

Worksheet 1 presents a summary of site-related monitoring data. Worksheet 2 presents a summary of medium-specific toxicity constants for each compound detected at the New Castle Spill Site. Worksheet 3 ranks carcinogenic compounds, and Worksheet 4 ranks noncarcinogenic compounds. Worksheet 5 presents the list of thirteen contaminants of concern and their ranking as carcinogens and noncarcinogens, and lists other physical properties of the chemicals. The last column of Worksheet 5 shows whether the contaminant has been chosen as an indicator compound for the site. The following are the indicator compounds chosen for the New Castle Spill Site:

- 1) tris (2-Chloropropyl) phosphate (tris);
- 2) Trichloroethene (TCE); and
- 3) trans-1,2-Dichloroethene

Table 3-1 lists the compounds detected at the site and the justification for their selection. Both TCE and

**TABLE 3-1  
NEW CASTLE SPILL SITE INDICATOR CHEMICALS  
JUSTIFICATION FOR SELECTION**

<b>CHEMICAL</b>	<b>DESIGNATED</b>	<b>RATIONALE</b>
trans-1,2-Dichloroethene	YES	Ranked high among noncarcinogens
Trichloroethene	YES	Exceeds standards, ranked high
1,2-Dichlorobenzene	NO	Ranks low, infrequently detected, meets MCLG
Iron	NO	Iron located in a non-drinking water aquifer
Manganese	NO	Detected at levels below Federal AWQC
Fluorene	NO	Infrequently detected at low levels
Ethylbenzene	NO	Ranks low, infrequently detected
Acenaphthylene	NO	Infrequently detected
Isophorone	NO	Infrequently detected
Fluoranthene	NO	Infrequently detected
Anthracene	NO	Infrequently detected at low levels
Pyrene	NO	Representative compound already chosen
Benz(a)anthracene	NO	Ranks low
Benzo(a)pyrene	NO	Infrequently detected, mid-ranked
Indeno(1,2,3-cd)pyrene	NO	Infrequently detected
Chrysene	NO	Representative compound chosen
Carbon Disulfide	NO	Ranked high, but too infrequently detected
tris(2-Chloropropyl)phosphate	YES	Frequently detected at high levels
Dibenzofuran	NO	Infrequently detected
Naphthalene	NO	Infrequently detected at low levels
2-Methylnaphthalene	NO	Infrequently detected at low levels
Benzo(b)fluoranthene	NO	Ranked high, but with estimated data

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trans-1,2-dichloroethene were chosen for their high IS rankings, as well as their frequency of detection. TCE ranks high as a carcinogen and trans-1,2-dichloroethene ranks high as a noncarcinogen. Although there are no toxicity constants available in the Superfund Public Health Evaluation Manual for tris, its frequency of occurrence, concentration, and relation to past site activities justifies its choice as an indicator compound. Of the three, only TCE is currently considered a "probable" human carcinogen by EPA.

### 3.3 Discussion of the Classification of Chemicals

A general discussion of the chemical classes and several representative compounds detected during the RI is presented here to emphasize the importance of examining physical/chemical parameters when selecting indicator compounds.

Both TCE and its degradation product, trans-1,2-dichloroethene, are classified as volatile organics. While TCE is relatively volatile, with a vapor pressure of 58 mm Hg, trans-1,2-dichloroethene is quite volatile, with a vapor pressure of 324 mm Hg. Both compounds are purgeable organics.

Tris is a semivolatile with a vapor pressure of less than 2 mm Hg. Its solubility in water at 30°C is 0.11 wt.% or 1,100 mg/L. Other frequently detected semivolatiles include chrysene, anthracene, pyrene, fluoranthene, and phenanthrene. All are in the class of polyaromatic hydrocarbons (PAHs) which are base-neutral extractable organics. While these PAHs are frequently detected in high concentrations, their EPA-approved

toxicity constants and locations on the site make them less of a potential hazard than the selected indicator compounds.

The inorganics detected, iron and manganese, were frequently found at high concentrations. They are often present in soils at high levels (Connor and Shacklette, 1975), and they do not volatilize easily but tend to adsorb onto soil particles. As a result, they are not transported easily to the air or into ground water, and they were not considered as representative indicator compounds. These compounds' concentrations are also representative of the native soils in the region.

## SECTION 4

### EXPOSURE ASSESSMENT

#### 4.1 General Information

The purpose of an exposure evaluation is presented in Section 2. The three major components are as follows:

- Evaluating environmental fate and transport of the indicator compounds;
- Establishing exposure scenarios;
- Determining possible exposures to potentially affected populations, and
- Calculating doses and resultant intakes.

#### 4.2 Environmental Fate and Transport

The three indicator compounds chosen for the New Castle Spill Site behave differently in each of the three types of environmental media being considered: surface water, ground water and soil. In general, TCE and trans-1,2-dichloroethene tend to be more mobile in the environment than tris, since they tend not to absorb onto soils, as tris does. These two compounds also tend to be less persistent in the environment, because they hydrolyze and volatilize more quickly than tris. Complete fate

and transport profiles for the indicator compounds appear in Appendix C. Table 4-1 shows the relative importance of processes influencing fate and transport for the indicator compounds.

It is assumed that compounds present at the site may be transported both off site and to the marsh by ground water movement. Tris may be transported by surface water runoff as well along the railroad tracks to the marsh, as evidenced by existing drainage patterns leading from the site. It is also assumed that some remaining tris contamination is present in the marsh as a result of the 1978 pumping of site ground water. This assumption is made based upon tris' lower vapor pressure (i.e., low volatility) and tendency to sorb, resistance to hydrolysis and strong phosphate bonds. Table 4-2 details the physical properties of the indicator compounds that help determine their environmental fate and transport.

#### 4.3 Exposure Scenarios for the Environmental Media

The primary exposure pathways for the indicator compounds are influenced by the geology and hydrology of the site as well as the chemical properties of the indicator compounds. These factors interact to define the various routes by which the compounds originating at the site could affect potentially exposed populations. These routes are presented in detail in Table 4-3 and summarized as follows:

<u>Medium</u>	<u>Exposure Route</u>
Air	- None
Surface Water	- Dermal Contact

**TABLE 4-1**  
**RELATIVE IMPORTANCE OF PROCESSES INFLUENCING FATE**  
**OF THE INDICATOR COMPOUNDS FOR THE NEW CASTLE SPILL SITE**

COMPOUND	SORPTION	VOLATILIZATION	BIODEGRADATION	PHOTOLYSIS	HYDROLYSIS	BIOACCUMULATION	OXIDATION
Trichloroethene	NO	YES	YES	NO	YES	NO	UNCLEAR
trans-1,2-Dichloroethene	NO	YES	NO	NO	YES	NO	UNCLEAR
tris(2-Chloropropyl)phosphate	YES	NO	UNCLEAR	UNCLEAR	NO	NO	UNCLEAR

**REFERENCES:**

MILLS, W.B., ET AL, 1982  
 CALLAHAN, M.A. ET AL, 1979

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**TABLE 4-2  
PHYSICAL PROPERTIES OF THE INDICATOR COMPOUNDS  
FOR THE NEW CASTLE SPILL SITE**

PROPERTY	trans-1,2-Dichloroethene	Trichloroethene	tris(2-Chloro propyl)phosphate
MOLECULAR WEIGHT, g	9.69E+01	1.31E+02	328.00
MELTING POINT, °C		-7.30E+01	
BOILING POINT, °C	6.00E+01	8.70E+01	
DENSITY, g/mL	1.26E+00	1.46E+00	1.29
FLASH POINT, °C	3.90E+01		218.00
<b>PARTITION COEFFICIENT</b>			
WATER SOLUBILITY, mg/L(25°C)	6.30E+04	1.10E+03	0.11 wt % or 1100 mg/L
OCTANOL WATER, K <sub>ow</sub>	3.02E+00	2.40E+02	199 *
SEDIMENT WATER, K <sub>oc</sub>	5.90E+01	1.26E+02	92.7 **
MICROORGANISM WATER, K <sub>b</sub> [(ug/g)/(mg/L)]	4.80E+01	9.70E+01	
<b>VOLATILIZATION COEFFICIENTS</b>			
HENRY'S LAW COEFFICIENTS (atm-m <sup>3</sup> /mol)	6.56E-03	9.10E-03	
VAPOR PRESSURE, mmHG (25°C)	2.65E+02	5.79E+01	<2
REAERATION RATE RATIO K <sub>v</sub> /k <sub>vo</sub>	6.01E-01	5.50E-01	

Source: Superfund Health Evaluation Manual, USEPA 1986  
Aquatic Fate Process Data for Organic Priority Pollutants, Mabey et al., 1982.

\* From EPA comment package dated March 1989

\*\* From SPHEM, 1986       $\log K_{oc} = (-0.55 \times \text{Log solubility}) + 3.64$

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**TABLE 4-3  
EXPOSURE PATHWAYS FOR  
NEW CASTLE SPILL SITE**

Scenario	Transport Media	Source	Release Mechanism	Exposure Point	Exposure Route	Selected for Analysis
<b>Present, existing</b>	<i>Air</i>	<i>Contaminated soil</i>	<i>Volatilization</i>	<i>On-site or Off-site</i>	<i>Inhalation</i>	<i>No - site covered by cement over majority of area</i>
			<i>Fugitive Dust</i>	<i>On-site or Off-site</i>	<i>Inhalation</i>	<i>No - site covered by cement over majority of area</i>
		<i>Contaminated surface water</i>	<i>Volatilization</i>	<i>On -site or Off-site</i>	<i>Inhalation</i>	<i>No - Tris not volatile, other indicators present in low concentrations</i>
	<i>Surface Water</i>	<i>Contaminated groundwater</i>	<i>Ground Water Discharge</i>	<i>Adjacent marshland</i>	<i>Dermal Contact</i>	<i>Yes - Marsh is near residential area</i>
		<i>Ponded water</i>	<i>Surface Runoff</i>	<i>Adjacent Marshland</i>	<i>Dermal Contact</i>	<i>Yes - Ponded area near tracks runs off to marsh area</i>
	<i>Soil/Sediments</i>	<i>Contaminated ground water</i>	<i>Groundwater Transport, Adsorption</i>	<i>Adjacent Marshland</i>	<i>Dermal Contact</i>	<i>Yes - Marsh soil is exposed in dry weather includes incidental ingestion</i>
<i>PICA Behavior</i>					<i>No- Behavior not likely</i>	
<i>Spill</i>		<i>Adsorption</i>	<i>On-Site</i>	<i>Dermal Contact</i>	<i>No - Site covered by cement over majority of area</i>	
<b>Hypothetical Future use</b>	<i>Ground Water</i>	<i>Contaminated soil</i>	<i>Leaching, Transport</i>	<i>Local Residence</i>	<i>Dermal contact</i>	<i>Yes - Future use of Columbia aquifer</i>
			<i>Leaching, Transport</i>	<i>Local Residence</i>	<i>Ingestion</i>	<i>Yes - Future use of Columbia aquifer</i>
			<i>Leaching, Transport, Volatilization</i>	<i>Local Residence</i>	<i>Inhalation</i>	<i>Yes - Future use of Columbia aquifer</i>

*Italics represents change in table*

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- Ground Water\*            - Dermal Contact while Bathing            1/2  
                             - Ingestion of Drinking Water            200  
                             - Inhalation while Bathing

\*Evaluated only as a hypothetical scenario

#### 4.3.1 Air

Since the indicator compounds are either relatively nonvolatile, or present in soil beneath the concrete pad, air is not considered a significant transport medium.

#### 4.3.2 Ground Water

Under the current, existing conditions scenario, ground water transports compounds located on site to the marsh area where it is able to mix with the surface water. Compounds can be exposed to the population via surface water contact. The Columbia aquifer does not presently supply residents with drinking or bathing water.

An exposed population is not available for assessment of exposure via residential use of ground water under the current, existing conditions scenario because 1) drinking water is supplied by municipal wells, 2) private or residential wells in the Columbia aquifer do not exist as evidence in the well inventory, 3) the Potomac aquifer is the source of regional potable water, and 4) the closest operating municipal well is completed in the Potomac aquifer and located approximately 0.7 miles downgradient. Based

on the well inventory performed during the RI, no municipal wells are installed in the Columbia aquifer.

The Columbia aquifer has been classified as GW-2B by U.S. EPA and pump test results do indicate that this aquifer is very productive. Therefore, a hypothetical, future-use ground water scenario consisting of a well installed at the property boundary was employed to determine the hazard and/or risk to a population who might use the Columbia aquifer downgradient of NCSS as a water supply. This scenario is highly unlikely since residents currently franchise to municipal water supply but are not restricted from drilling private wells.

Drinking water and bathing exposure routes through Potomac aquifer use are not analyzed because of the lack of localized recharge from the Columbia aquifer to the Potomac aquifer beneath it. There is a 80- to 90-foot clay unit separating the two aquifers that prevents recharge. Thus, migration of contaminants from the Columbia aquifer into the Potomac aquifer is unlikely.

#### 4.3.3 Surface Water

Surface water is an exposure medium to the extent that people in the residential area may come into contact with the water of the marshland. This scenario occurs chiefly as children play in the area, but it is also assumed that adults may come into contact with the surface water. The marsh lies only a few yards from the residences. There is a clear path of access for children in the form of a large break in the tall grass surrounding the marsh. It is only a few yards further to marsh water, which is present

12 months of the year. This site thus represents an attractive year-round play area for local children.

Exposure to contaminants from surface water contact is based upon supposed presence of tris in the marsh area due to past ground water pumping into the marsh and of other indicator compounds owing to ground water discharge and surface water movement from the site area to the marsh area. Figure 1-5 shows estimated ground water movement from the south end of the site to the marsh. In the case of ground water movement as a pathway, ground water is assumed to mix with the surface water after discharge to the marsh. In the case of surface water movement to the marsh it is observed that ponded water at the railroad tracks drains in the direction of the marsh.

The quantity of on-site contaminants that can travel to the marsh via ground water discharge is presently unknown. Similarly, the amount of mixing between the discharged ground water and surface water is also unknown. Information regarding both variables will help determine what level of contaminants are potentially available to expose the population. The scenarios described are possible occurrences given estimated geologic and hydrologic characteristics of the area.

#### 4.3.4 Soil/Sediments

Soil is an exposure medium similar to surface water. Children and adults may come into contact with soil in the area of the marsh, or near the railroad tracks. This contact also includes incidental ingestion through casual contact with soil on hands and fingers. The exposure concentrations used to calculate acute

hazard from soil contamination are from monitoring well MW-5. MW-5 data is being used to represent the worst possible soil concentrations for the study area. An average of all soil data across the site is used to calculate the chronic hazard. Soil can become contaminated through the adsorption of contaminants onto soil particles after ground water transport, or directly, after a spill of a contaminant.

#### 4.4 Exposures to Potentially Affected Populations

The potentially affected populations consist of the adults and children living in the residential area to the west of the site. These populations are assumed to be exposed in different ways and to be affected differently by exposure. Routes of exposure concerning contaminant intakes and dosage are analyzed for each of three populations: Adult, Child Age 6-12, and Child Age 2-6. The routes of exposure for the different populations are outlined in Table 4-4.

The hypothetical, future-use ground water scenario involves an exposed family who could possibly use the Columbia aquifer in the vicinity of NCSS as a potable water supply. Routes of exposure concerning intakes and dosages are analyzed for adults, child age 6-12 and child age 2-6. The routes of exposure for the hypothetical population are also outlined in Table 4-4.

#### 4.5 Calculations of Dosages and Resultant Intakes

The amount of contaminants that populations are exposed to depends on habits of the population. Assumptions behind these habits are presented in Table 4-5 for the different routes of

**TABLE 4-4  
 ROUTES OF EXPOSURE  
 USED IN CALCULATION OF INTAKES  
 NEW CASTLE SPILL SITE**

SCENARIO	POPULATION	EXPOSURE TYPE	EXPOSURE ROUTE	
<b>Present, existing</b>	Child 2-6	Dermal	Playing in soil of marshland and near rail-road tracks - includes incidental ingestion  Surface water contact	
	Child 6-12	Dermal	Playing in soil of marshland and near rail-road tracks - includes incidental ingestion  Surface water contact	
	Adult	Dermal	Surface water contact  Casual contact with soil	
	<b>Hypothetical Future use</b>	Child 2-6	Inhalation	Exposure while bathing
		Child 6-12		
		Adult	Ingestion	Drinking water
		Dermal	Contact while bathing	

**TABLE 4-5  
CHARACTERISTICS OF SUBCHRONIC/CHRONIC EXPOSURE SCENARIOS  
NEW CASTLE SPILL SITE**

Scenario	Route of Exposure	Medium	Activity	Population	Subchronic Exposure Characteristics	Chronic Exposure Characteristics
<b>Present, existing</b>	Dermal	Soil	Play	Child Age 6-12	One exposure events (hands only) at maximum concentration <i>includes 50 mg incidental ingestion</i>	One exposure event (hands only) per day, 150 days per year, at average concentration <i>includes 50 mg incidental ingestion</i>
				Child Age 2-6	One exposure events (hands only) at maximum concentration <i>includes 100 mg incidental ingestion</i>	One exposure event (hands only) per day, 150 days per year, at average concentration <i>includes 100 mg incidental ingestion</i>
		Surface Water	Casual Contact	Child Age 2-6 Child Age 6-12 Adult	One hours of exposure (20% of body) at maximum concentration	One hour of exposure (20% of body), 150 days a year (Child 6-12), or 5 days a year (Adult) at average concentration
<b>Hypothetical future use</b>	Dermal	Ground Water	Bathing	Child Age 6-12 Child Age 2-6 Adult	Twenty minutes of exposure over 80% of the body at maximum concentration concentration	Twenty minutes of exposure over 80% of the body at average concentration concentration
	Inhalation	Ground Water	Bathing	Child Age 6-12 Child Age 2-6 Adult	Thirty minutes exposure at maximum concentration	Thirty minutes exposure at average concentration
	Ingestion	Ground Water	Drinking Water	Child Age 6-12 Child Age 2-6 Adult	Ingestion of 1L (children) or 2L (adults) each day at maximum concentration	Ingestion of 1L (children) or 2L (adults) each day at average concentration

*Italics includes changes to table.*

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exposure; standard parameters used for calculation of dosage and intake are presented in Table 4-6.

The methodology used for calculation of intakes of contaminants from the aforementioned routes is presented in Section 2. The resulting doses and intakes at chronic and subchronic exposure levels for the actual and hypothetical scenarios are presented in Table 4-7 and Table 4-8, respectively. Sample calculations for subchronic and chronic intakes are presented in Appendix D.

#### 4.6 Environmental Exposure Scenarios and Receptors

The potential environmental exposure pathways and receptors (or population) for the New Castle Spill Site are presented in Table 4-9. The macroinvertebrate population has been exposed to the indicator compounds through the surface water and sediments in the marshland adjacent to the site. Although the macroinvertebrate population does not appear to be affected by compounds detected at the site, potential exposure pathway and receptor populations were identified. The exposure pathway might be ingestion of the macroinvertebrae. Qualitatively, populations could include fish (bottom feeders up through game fish), fish-eating birds (i.e., mammals, wading birds, ducks, herons, hawks) and potentially man. Figure 4-1 depicts a possible food cycle from surface water and sediments to man. The mammal identified during the environmental assessment was limited to Muskrat, which is a herbivore.

**TABLE 4-6  
STANDARD PARAMETERS FOR CALCULATION OF DOSAGE AND INTAKE  
NEW CASTLE SPILL SITE**

		Adult	Child Age 6-12	Child Age 2-6
<b>PHYSICAL CHARACTERISTICS</b>				
Average Body Weight	(a)	70 kg	29 kg	16 kg
Average Skin Surface Area	(a),(e)	18,150 cm <sup>2</sup>	10,470 cm <sup>2</sup>	6980 cm <sup>2</sup>
<b>ACTIVITY CHARACTERISTICS</b>				
Amount of Water Ingested Daily	(a)	2 liters	1 liter	1 liter
Amount of Air Breathed Daily	(d)	20 m <sup>3</sup>	11 m <sup>3</sup>	6 m <sup>3</sup>
Duration of Soil Contact	(d)	1 event	1 event	1 event
Frequency of Soil Contact	(d)	5 days/year	150 days/year	150 days/year
Percentage of Skin Surface Area Contacted by Soils	(d)	20%	20%	20%
Skin Absorption Rate of Compounds in Soil	(c)	6%	12%	12%
Incidental Soil Ingestion (per event)	(c)	50 mg	50 mg	100 mg
Frequency of Surface Water Contact (Casual)	(d)	5 days/year	150 days/year	150 days/year
Duration of Surface Water Contact (Casual)	(d)	1 event	1 event	1 event
Percentage of Skin Surface Area Immersed	(a)	20%	20%	20%
Percentage of Surface Area Immersed while Bathing	(a)	80%	80%	80%
Length of Exposure while Bathing	(b)	20 min.	20 min.	20 min.
Amount of Air Breathed while Bathing	(a)	0.83 cu m/hr	0.46 cu m/hr	0.25 cu m/hr
Length of Additional Exposure after Bathing	(b)	10 min.	10 min.	10 min.
Volume of Showerstall	(b)	3 cu m	3 cu m	3 cu m
Volume of Bathroom	(b)	10 cu m	10 cu m	10 cu m
Volume of Water used in Showering	(b)	200 L	200 L	200 L
Absorption via Inhalation (%)		50	50	50
Absorption via Ingestion (%)		100	100	100
<b>MATERIAL CHARACTERISTICS</b>				
Dust Adherence (Potting Soil)	(a)	0.51 mg/cm <sup>2</sup>	0.51 mg/cm <sup>2</sup>	0.51 mg/cm <sup>2</sup>
Soil Matrix Effect	(c)	15%	15%	15%
Mass Flux Rate (water-based)	(a)	0.5 mg/cm <sup>2</sup> /hr	0.5 mg/cm <sup>2</sup> /hr	0.5 mg/cm <sup>2</sup> /hr

a - Superfund Exposure Assessment Manual, 1986

b - K.G. Symms, "An Approximation of the Inhalation Exposure to Volatile Synthetic Organic Chemicals from Showering with Contaminated Household Water," Paper to be presented at the Symposium of American College of Toxicologists, Nov. 15, 1986

c - J.K. Hawley, "Assessment of Health Risk from Exposure to Contaminated Soil," Risk Analysis, Vol. 5, No. 4, 1985

d - ERM Staff Professional Judgement

e - Anderson et al, 1984

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**TABLE 4-7**  
**CALCULATION OF SUBCHRONIC AND CHRONIC DAILY INTAKES**  
**FOR NEW CASTLE SPILL SITE**  
**(all exposure point concentrations in ppm)**

**PRESENT, EXISTING CONDITIONS**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Maximum Exposure Point Concentration	Subchronic Daily Intake mg/kg/day	Average Exposure Point Concentration	Chronic Daily Intake mg/kg/day
Adults	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	ND	0.00E+00	ND	0.00E+00
			Trichloroethene	ND	0.00E+00	ND	0.00E+00
			tris(2-Chloropropyl)phosphate	4.20E-02	1.09E-06	1.10E-02	3.89E-09
	Soils	Dermal Contact	trans-1,2-Dichloroethene	ND	0.00E+00	ND	0.00E+00
			Trichloroethene	ND	0.00E+00	ND	0.00E+00
			tris(2-Chloropropyl)phosphate	1.17E+01	1.12E-05	1.64E+00	2.13E-08
Child Age 6-12	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	ND	0.00E+00	ND	0.00E+00
			Trichloroethene	ND	0.00E+00	ND	0.00E+00
			tris(2-Chloropropyl)phosphate	4.20E-02	1.52E-06	1.10E-02	1.63E-07
	Soils	<i>Dermal Contact *</i>	trans-1,2-Dichloroethene	ND	0.00E+00	ND	0.00E+00
			Trichloroethene	ND	0.00E+00	ND	0.00E+00
			tris(2-Chloropropyl)phosphate	1.17E+01	2.81E-05	1.64E+00	1.61E-06
Child Age 2-6	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	ND	0.00E+00	ND	0.00E+00
			Trichloroethene	ND	0.00E+00	ND	0.00E+00
			tris(2-Chloropropyl)phosphate	4.20E-02	1.83E-06	1.10E-02	1.97E-07
	Soils	<i>Dermal Contact **</i>	trans-1,2-Dichloroethene	ND	0.00E+00	ND	0.00E+00
			Trichloroethene	ND	0.00E+00	ND	0.00E+00
			tris(2-Chloropropyl)phosphate	1.17E+01	8.28E-05	1.64E+00	4.76E-06

\* included incidental ingestion of 50 mg

\*\* included incidental ingestion of 100 mg

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**TABLE 4-8**  
**CALCULATION OF SUBCHRONIC AND CHRONIC DAILY INTAKES**  
**FOR NEW CASTLE SPILL SITE**  
(all exposure point concentrations in ppm)  
**HYPOTHETICAL FUTURE-USE SCENARIO**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Maximum Exposure Point Concentration	Subchronic Daily Intake mg/kg/day	Average Exposure Point Concentration	Chronic Daily Intake mg/kg/day		
Adults	Ground Water	Dermal Contact	trans-1,2-Dichloroethene	1.10E-02	3.78E-07	2.00E-03	6.84E-08		
			Bathing	Trichloroethene	1.20E-01	4.12E-06	1.80E-02	6.16E-07	
				tris(2-Chloropropyl)phosphate	1.10E+02	3.78E-03	9.00E+00	3.08E-04	
		Ingestion	Drinking Water	trans-1,2-Dichloroethene	1.10E-02	3.15E-04	2.00E-03	5.72E-05	
					Trichloroethene	1.20E-01	3.43E-03	1.80E-02	5.15E-04
					tris(2-Chloropropyl)phosphate	1.10E+02	3.15E+00	9.00E+00	2.57E-01
		Inhalation	Bathing	trans-1,2-Dichloroethene	1.10E-02	4.68E-04	2.00E-03	1.70E-04	
					Trichloroethene	1.20E-01	5.10E-03	1.80E-02	1.53E-03
					tris(2-Chloropropyl)phosphate	1.10E+02	4.68E+00	9.00E+00	7.65E-01
Child Age 6-12	Ground Water	Dermal Contact	trans-1,2-Dichloroethene	1.10E-02	5.23E-07	2.00E-03	9.54E-08		
			Bathing	Trichloroethene	1.20E-01	5.70E-06	1.80E-02	8.59E-07	
				tris(2-Chloropropyl)phosphate	1.10E+02	5.23E-03	9.00E+00	4.29E-04	
		Ingestion	Drinking Water	trans-1,2-Dichloroethene	1.10E-02	3.80E-04	2.00E-03	6.90E-05	
					Trichloroethene	1.20E-01	4.14E-03	1.80E-02	6.21E-04
					tris(2-Chloropropyl)phosphate	1.10E+02	3.80E+00	9.00E+00	3.11E-01
		Inhalation	Bathing	trans-1,2-Dichloroethene	1.10E-02	6.27E-04	2.00E-03	2.28E-04	
					Trichloroethene	1.20E-01	6.84E-03	1.80E-02	2.05E-03
					tris(2-Chloropropyl)phosphate	1.10E+02	6.27E+00	9.00E+00	1.03E+00
Child Age 2-6	Ground Water	Dermal Contact	trans-1,2-Dichloroethene	1.10E-02	6.32E-07	2.00E-03	1.15E-07		
			Bathing	Trichloroethene	1.20E-01	6.89E-06	1.80E-02	1.04E-06	
				tris(2-Chloropropyl)phosphate	1.10E+02	6.32E-03	9.00E+00	5.18E-04	
		Ingestion	Drinking Water	trans-1,2-Dichloroethene	1.10E-02	6.88E-04	2.00E-03	1.25E-04	
					Trichloroethene	1.20E-01	7.50E-03	1.80E-02	1.13E-03
					tris(2-Chloropropyl)phosphate	1.10E+02	6.88E+00	9.00E+00	5.63E-01
		Inhalation	Bathing	trans-1,2-Dichloroethene	1.10E-02	6.16E-04	2.00E-03	2.24E-04	
					Trichloroethene	1.20E-01	6.72E-03	1.80E-02	2.02E-03
					tris(2-Chloropropyl)phosphate	1.10E+02	6.16E+00	9.00E+00	1.01E+00

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TABLE 4-9

POTENTIAL ENVIRONMENTAL EXPOSURE PATHWAYS  
NEW CASTLE SPILL SITE

MEDIUM	SOURCE	RELEASE MECHANISM	EXPOSURE POINT	EXPOSURE ROUTE	POPULATION	QUALITATIVE POTENTIAL AS PATHWAY **
Macroinvertebrae	Contaminated Surface Water and Sediments	Leaching and Transport	Adjacent Marshland	Ingestion	Bottomfeeders - fish	Low
					Game Fish	Low
		Bioconcentration			Fish-Eating Birds *	Low
					Mammals (muskrats)	Low
Game Fish	Macroinvertebrae	Bioconcentration	Marshlands	Ingestion	Humans	Low
Game Birds	Macroinvertebrae Fish	Bioconcentration	Marshlands	Ingestion	Humans	Low

\*Includes wading birds, ducks, herons, hawks

\*\*Low potential to biomagnify in the food chain because of the low bioconcentration factors for these compounds

BCF, L/kg (US EPA, 1986)

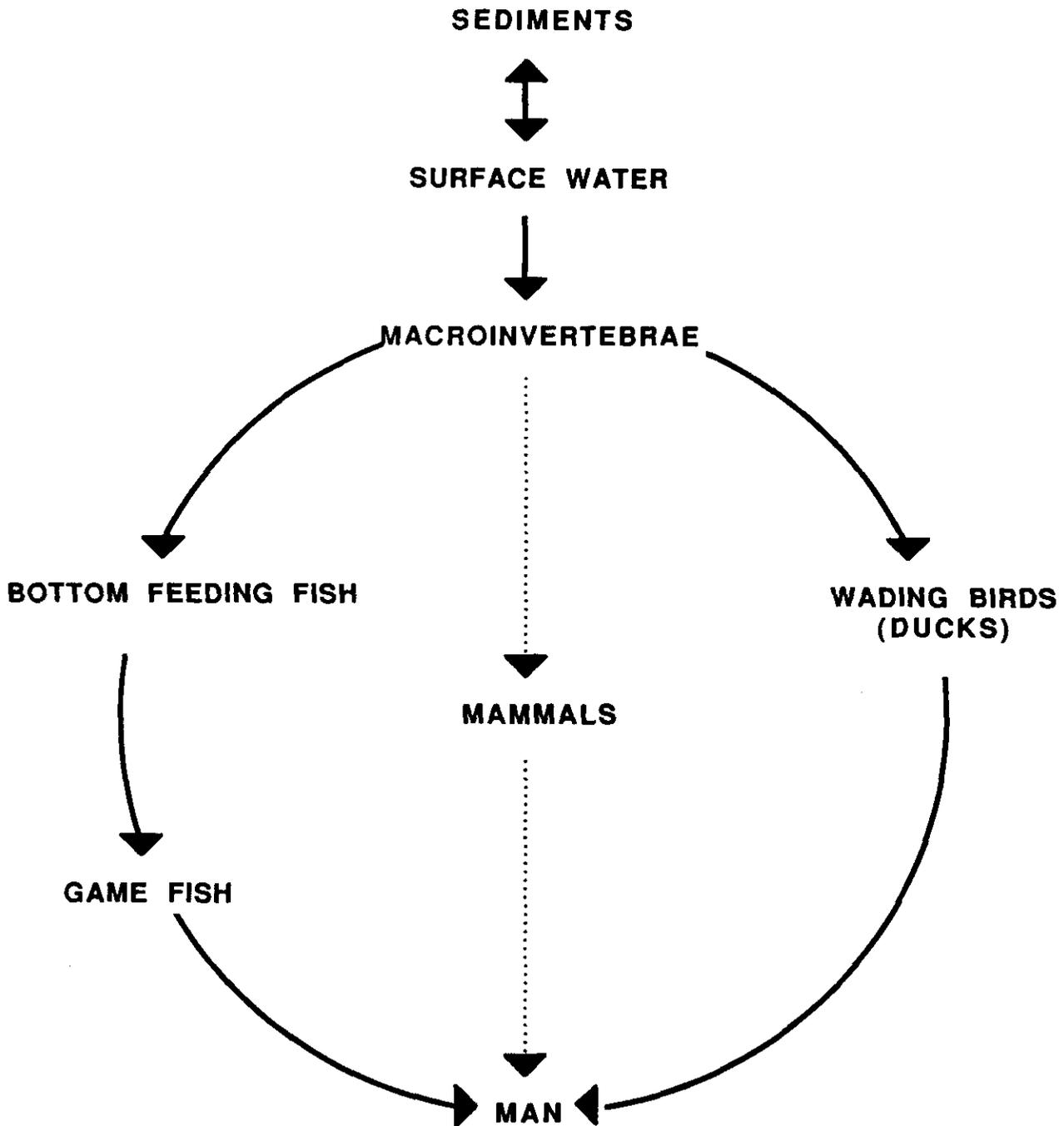
trans-1,2-Dichloroethene	1.6
Trichloroethene	10.6
tris-(2-Chloropropyl)phosphate	2.7***

\*\*\*Based on tris(2,3-Dibromopropyl)phosphate

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FIGURE 4-1  
POSSIBLE FOOD CYCLE FOR  
NEW CASTLE SPILL SITE



—————▶ Possible Pathways  
.....▶ No Evidence

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## SECTION 5

### TOXICITY EVALUATION

#### 5.1 General Information

The toxicity evaluation of the indicator compounds selected for the New Castle Spill Site is conducted to develop a data base of quantitative toxicity indices against which exposure point intakes can be compared in the risk characterization of the site. The methodology for this toxicity evaluation is discussed in Section 2. A detailed discussion of US EPA's weight-of-evidence classification system is presented in Appendix A.

Trichloroethene is classified as a "probable" human carcinogen by US EPA. However, the International Agency for Research on Cancer (IARC) considers the data insufficient to determine whether TCE is a "probable" or a "possible" human carcinogen. trans-1,2-Dichloroethene is classified as a noncarcinogen by US EPA and IARC. Neither US EPA nor IARC has classified tris as a carcinogen or noncarcinogen. Based on available test data, tris is evaluated as a noncarcinogen in this assessment. Table 5-1 presents the relevant quantitative indices of toxicity for the indicator compounds that will be used in risk characterization. Based on the available data from animal studies, a reference dose (RfD) value has been determined for tris. The detailed methodology used to calculate this value is presented in Section 2.

Table 5-1

RELEVANT QUANTITATIVE INDICES OF TOXICITY FOR INDICATOR COMPOUNDS  
NEW CASTLE SPILL SITE

COMPOUND	ORAL AIS (mg/kg/day)	ORAL RfD (mg/kg/day)	ORAL CPF (1/(mg/kg/day))	INHALATION CPF (1/(mg/kg/day))
trans-1,2-Dichloroethene	2E-01 (a)	2E-02 (b)	NA	NA
Trichloroethene	NA	NA	1.10E-02 (b)	1.3E-02 (b)
Tris(2-chloropropyl)phospate	1.25E +00 (c)	1.25E-01 (c)	NA	NA

NA - Not applicable

(a) - The RfD for this compound is based on a subchronic study.

Removal of a safety factor of 10 (extrapolation of subchronic to chronic) yields the AIS.

(b) - Values obtained from on-line IRIS data base

(c) - Calculated by ERM (see Appendix E for derivation)

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The toxicity data presented below are summarized from US EPA Health Effects Assessment documents, the product safety information sheet for tris, and the Kirk-Othmer Concise Encyclopedia of Chemical Technology. A detailed toxicology profile for each indicator is presented in Appendix F. The major health effects resulting from exposure to indicator compounds are discussed below. However, the concentrations at which toxic effects occur are generally orders of magnitude higher than environmental concentrations of those compounds. The potential ARARs identified for each indicator compound are presented in Appendix F as well.

## 5.2 trans-1,2-Dichloroethene

trans-1,2-Dichloroethene toxicity has not been well studied in animals or humans. Exposure to high vapor concentrations of trans-1,2-dichloroethene causes nausea, vomiting, weakness, tremor, and cramps in humans, but these effects reverse following removal of the source of trans-1,2-dichloroethene. Exposure to vapors can also produce anesthetic and narcotic effects. Chronic exposure to low (500-1000 ppm) levels of trans-1,2-dichloroethene in animals resulted in no observable changes in pathology, clinical chemistry or organ and body weights (ACGIH 1986, Documentation of the TLVs and BEIs).

## 5.3 Trichloroethene

In humans, trichloroethene was once used medically for its anesthetic and analgesic properties. Acute exposures to extremely high inhaled concentrations of trichloroethene (e.g.,

15,000 ppm) are known to elicit cardiac arrhythmias. Chronic exposure to levels of trichloroethene ranging from 40-200 ppm has been reported to induce neurotoxic (toxic to nerves or the nervous system) symptoms such as involuntary muscular movements, sleep disturbances, and psychotic episodes. There is a great variation between individuals in tolerance to the neurotoxic effects of trichloroethene (ACGIH 1986).

#### 5.4 tris(2-chloropropyl)phosphate

The Acceptable Daily intake for Chronic exposure (AIC) or reference dose (RfD) of 0.125 mg/kg/day for tris(2-chloropropyl)-phosphate was calculated from a rat subchronic study described in the Product Safety Information Sheet supplied by Stauffer Chemical Company. The RfD for humans was calculated to be equal to the No-Observable-Adverse-Effect Level (NOAEL) for an experimental animal divided by appropriate safety factors. In a toxicology study, the NOAEL is the dose at which the animal does not receive any toxic effects from the chemical in question. The RfD is calculated in Appendix E of this document and is based on the following study.

A three month toxicology study was performed on male and female rats receiving daily dietary concentrations of tris at 800, 2,500, 7,500 and 20,000 ppm. An increase in relative and absolute liver weight is observed in male rats at all dose levels and in female rats receiving 7,500 or 20,000 ppm tris in the diet. This type of change is not judged to be an adverse or toxic effect of tris exposure. Toxic effects were observed in the female rats receiving the highest dose and in male rats at both the 7,500 and 20,000 ppm dose levels. These effects included

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mild cortical tubular degenerative changes in the kidney and mild histopathological changes in the liver (only in animals receiving 20,000 ppm). Very mild hypoplasia of the sternal bone marrow and very mild thyroid follicular hyperplasia was observed in the female rats receiving the highest dose of tris.

The toxicity of tris(2-chloropropyl)phosphate has not been well studied in animals and has not been examined in humans. The acute oral LD50 is 2,800 mg/kg for female rats and 4,200 mg/kg for male rats. Extensive studies have been performed which indicate that tris(2-chloropropyl)phosphate is not a mutagen. No animal studies have been conducted using chronic exposures. (Stauffer Chemical Product Safety Sheet for Fyrol PCF - Flame Retardant).

## SECTION 6

### RISK CHARACTERIZATION

#### 6.1 General Information

This section assesses the potential risks to human health and the environment associated with exposure to the various indicator chemicals under the No-Action Alternative. The potential risks of exposure to carcinogens and noncarcinogens are assessed by comparing the following:

- 1) current exposure point concentrations with potentially applicable or relevant and appropriate requirements (ARARs),
- 2) current subchronic doses with acceptable subchronic intakes for noncarcinogenic effects,
- 3) current chronic doses with acceptable reference doses (RfDs) for noncarcinogenic effects, and
- 4) calculated risks with guideline risks for potential carcinogens.

A discussion of uncertainties encountered in the endangerment assessment process is included in this section to provide some perspective in interpreting the results of the assessment.

6.2 Comparison to Potentially Applicable or Relevant  
and Appropriate Requirements

Standards and guidelines for contaminants of concern are determined by considering the exposure pathways operating at the site. The following is a summary of the standards and guidelines that are potentially applicable to the media at NCSS.

<u>Medium</u>	<u>Potential ARARs</u>
Soil	- None (only PCBs and dioxin by U.S. EPA)
Sediment	- None
Air	- Not applicable since no air releases exist
Surface Water	- U.S. EPA Ambient Water Quality Criteria - DNREC Water Quality Standards for Streams: Amended 23 December 1985
Ground Water	- U.S. EPA Maximum Contaminant Levels (MCLs) - DNREC Regulations Governing Drinking Water Standard: Revised 5 May 1982

- DNREC Regulations Governing the Control of Water Pollution: Amended 23 June 1983
- Ground Water discharges as surface water (see Surface Water)

Wetlands

- U.S. Army Corp of Engineers

The potential Delaware ARARs for NCSS are presented in Table 6-1. A comparison of detected levels of compounds in ground water and surface water to potential ARARs is presented in Table 6-2.

For many of the contaminants related to the NCSS, promulgated standards and guidelines do not exist. The compounds that meet all of their potential ARARs are trans-1,2-dichloroethene and 1,2-dichlorobenzene. The maximum and average values of TCE exceed its MCL; however, only the maximum detected value exceeds its ambient water quality criteria (AWQC) for protection of human health for ingestion of water and fish and for ingestion of fish only. The maximum and average values of iron exceed its AWQC for protection of human health for ingestion of water and fish and for protection of aquatic life-chronic. The maximum and average values of manganese exceed its AWQC for protection of human health for ingestion of water and fish and for ingestion of fish only. The iron and manganese levels detected in the ground water exceeded Delaware secondary MCLs. However, these levels were not above levels found in typical Coastal Plain aquifer systems.

TABLE 6-1

POTENTIAL DELAWARE ARARs FOR  
NEW CASTLE SPILL SITE

LAW/REGULATION/CODE	ARAR TYPE		
	AMBIENT/ CHEMICAL-SPECIFIC	ACTION- SPECIFIC	LOCATION- SPECIFIC
Regulations Governing the Construction of Water Wells		X	
Water Quality Standards for Streams (amended 12/23/85)	X		
Regulations Governing the Control of Water Pollution	X	X	
The Wetlands Act (Chapter 66)			X
Wetland Regulations			X
Subaqueous Lands (Chapter 72)			X
Regulations Governing the Use of Public Subaqueous Lands			X
Delaware Environmental Protection Act			
Regulations Governing the Control of Air Pollution	X	X	
Title 16, Section 122, Delaware Code			X
Regulations Governing Drinking Water Standards	X		
Delaware River Basin Commission Rules and Regulations		X	X
Coastal Zone Act			X
Delaware Hazardous Waste Rules and Regulations (RCRA)	X	X	
Delaware Solid Waste Disposal Regulations		X	
Preservation of Historic Places (36 CFR 800)			X

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**Table 6-2**  
**NEW CASTLE SPILL SITE**  
**COMPARISON WITH APPLICABLE OR RELEVANT**  
**AND APPROPRIATE REQUIREMENTS**

(all values in mg/L)

CHEMICAL	GROUND WATER (mg/L)		US EPA MCLs	US EPA MCLGs	DNREC STANDARDS*	SURFACE WATER (mg/L)		EPA AMBIENT FRESHWATER QUALITY CRITERIA				
	MAX	AVE				MAX	AVE	ingestion of fish	ingestion of fish and water	protection of aquatic life - acute	protection of aquatic life - chronic	
trans-1,2-dichloroethene	0.011	0.001		0.070					8.07E - 02	2.70E - 03	1.16E + 01	not available
Trichloroethene	0.120	0.018	0.005								4.50E + 01	2.19E + 01
1,2-Dichlorobenzene	0.002	0.0003	0.6200								2.50E - 01	5.00E + 02
Carbon Disulfide	0.015	0.001										1.00E + 00
Iron	19.400	12.600			0.300						1.00E + 00	
Manganese	5.240	1.494			0.050						1.50E + 00	
tris(2-Chloropropyl)phosphate	110.000	9.000				0.042	0.011					
Naphthalene	0.008	0.002									2.30E + 00	6.20E - 01
Phenanthrene	0.005	0.001							2.80E - 06	3.11E - 05		

\* DNREC standards defer to US EPA standards if blank

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### 6.3 Calculation of Noncarcinogenic Hazard

As described in Section 2, the hazard index is the ratio of the expected potential dose to acceptable exposure levels. Values of less than unity (one) indicate that no hazard exists. The assessment of the noncarcinogenic hazard for the site and marsh area (i.e., present, existing conditions scenario) is shown in Table 6-3. Both chronic (repeated) and subchronic (short-term) exposures are calculated to attain hazard indices. Subchronic (acute) exposure occurs from a large tris concentration in soils (>100 ppm) near the railroad tracks. Chronic exposure results from the movement of tris into the marsh area, and coming into contact with the local population through repeated dermal exposure to surface water and marsh sediments. The levels for the upper bound chronic hazard indices (present, existing conditions scenario) are less than one.

For consistency with SPHEM guidance and EPA Region III practice, worst-case (or conservative) upper bound estimates of exposure and risk are calculated. The calculations are based on subchronic exposures (i.e., maximum concentrations) combined with the chronic reference doses and reflect the conservative assumption that some individuals may be chronically exposed to the highest concentrations. The upper bound worst-case hazard index for the present, existing conditions is  $1.45 \times 10^{-4}$ , which is four orders of magnitude lower than EPA's guideline of one.

The assessment of the noncarcinogenic hazard for the hypothetical, future-use scenario (e.g., family using Columbia aquifer near the site for potable water) is shown in Table 6-4. Both chronic and subchronic exposures are calculated to obtain

**TABLE 6-3  
NEW CASTLE SPILL SITE  
ASSESSMENT OF NONCARCINOGENIC HAZARD INDICES  
UPPER BOUND REASONABLE CASE ESTIMATE**

**PRESENT, EXISTING CONDITIONS**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Subchronic Intake (mg/kg/day)	AIS (mg/kg/day)	Subchronic Hazard Index	Chronic Intake (mg/kg/day)	RfD (AIC) (mg/kg/day)	Chronic Hazard Index
Adults	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.09E-06	1.25E+00	8.72E-07	3.89E-09	1.25E-01	3.11E-08
	Soils	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.12E-05	1.25E+00	8.96E-06	2.13E-08	1.25E-01	1.70E-07
<b>Total Subchronic Hazard Index =</b>				<b>9.83E-06</b>			<b>Total = 2.02E-07</b>		
Children 6-12	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.52E-06	1.25E+00	1.22E-06	1.63E-07	1.25E-01	1.30E-06
	Soils	<i>Dermal Contact*</i>	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	2.81E-05	1.25E+00	2.25E-05	1.61E-06	1.25E-01	1.29E-05
<b>Total Subchronic Hazard Index =</b>				<b>2.37E-05</b>			<b>Total = 1.42E-05</b>		
Children 2-6	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.83E-06	1.25E+00	1.46E-06	1.97E-07	1.25E-01	1.58E-06
	Soils	<i>Dermal Contact**</i>	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	8.28E-05	1.25E+00	6.62E-05	4.76E-06	1.25E-01	3.81E-05
<b>Total Subchronic Hazard Index =</b>				<b>6.77E-05</b>			<b>Total = 3.97E-05</b>		

**Lifetime-Weighted  
Subchronic Hazard = 1.45E-05**

**Lifetime-Weighted  
Chronic Hazard = 3.76E-06**

*Bold Italics indicates a change to the table.  
\* Included incidental ingestion of 50 mg  
\*\* Included incidental ingestion of 100 mg*

**LIFETIME WEIGHTED = CHRONIC INTAKE x RfD and represents the upper bound reasonable case**

NA - Not applicable

**SAMPLE CALCULATION**

tris(2-chloropropyl)phosphate in adults; dermal contact with surface water

$$\begin{aligned} \text{Chronic hazard index} &= \text{Chronic daily intake/RfD} \\ &= 0.0000000389 / 0.125 \\ &= 0.0000000311 \end{aligned}$$

Total chronic hazard index = sum of all exposure routes for all indicators.

$$\text{Lifetime-weighted chronic hazard} = \{\text{total chronic adults} \cdot (58/68)\} + \{\text{total chronic child 6-12} \cdot (6/68)\} + \{\text{total chronic child 2-6} \cdot (4/68)\}$$

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**TABLE 6-3 Continued  
NEW CASTLE SPILL SITE  
ASSESSMENT OF NONCARCINOGENIC HAZARD INDICES  
UPPER BOUND WORST CASE ESTIMATE**

**PRESENT, EXISTING CONDITIONS**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Subchronic Intake (mg/kg/day)	AIS (mg/kg/day)	Subchronic Hazard Index	Subchronic Intake (mg/kg/day)	RfD (AIC) (mg/kg/day)	Chronic Hazard Index
Adults	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.09E-06	1.25E+00	8.72E-07	1.09E-06	1.25E-01	8.72E-06
	Soils	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.12E-05	1.25E+00	8.96E-06	1.12E-05	1.25E-01	8.96E-05
				<i>Total Subchronic Hazard Index=</i>		<i>9.83E-06</i>	<i>Total Chronic Hazard Index=</i>	<i>9.83E-05</i>	
Children 6-12	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.52E-06	1.25E+00	1.22E-06	1.52E-06	1.25E-01	1.22E-05
	Soils	<i>Dermal Contact*</i>	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	2.81E-05	1.25E+00	2.25E-05	2.81E-05	1.25E-01	2.25E-04
				<i>Total Subchronic Hazard Index=</i>		<i>2.37E-05</i>	<i>Total Chronic Hazard Index=</i>	<i>2.37E-04</i>	
Children 2-6	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	1.83E-06	1.25E+00	1.46E-06	1.83E-06	1.25E-01	1.46E-05
	Soils	<i>Dermal Contact**</i>	trans-1,2-Dichloroethene	0.00E+00	2.00E-01	0.00E+00	0.00E+00	2.00E-02	0.00E+00
			Trichloroethene	0.00E+00	NA	0.00E+00	0.00E+00	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	8.28E-05	1.25E+00	6.62E-05	8.28E-05	1.25E-01	6.62E-04
				<i>Total Subchronic Hazard Index=</i>		<i>6.77E-05</i>	<i>Total Chronic Hazard Index=</i>	<i>6.77E-04</i>	

**Upper Bound Worst  
Case Estimate of  
Chronic Hazard = 1.45E-04**

*Italics indicates a change to the table.*

\* Included incidental ingestion of 50 mg

\*\* Included incidental ingestion of 100 mg

*Upper bound worst case estimate of chronic hazard index is based on the maximum concentration detected times the RfD.*

NA - Not applicable

**SAMPLE CALCULATION**

tris(2-chloropropyl)phosphate in adults; dermal contact with surface water

Chronic hazard Index = Subchronic daily intake/RfD

$$= 0.00000109 / 0.125$$

$$= 0.00000872$$

Total chronic hazard index = sum of all exposure routes for all indicators.

$$\text{Lifetime-weighted chronic hazard} = \{\text{total chronic adults} \cdot (58/68)\} + \{\text{total chronic child 6-12} \cdot (6/68)\} + \{\text{total chronic child 2-6} \cdot (4/68)\}$$

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**TABLE 6-4  
NEW CASTLE SPILL SITE  
ASSESSMENT OF NONCARCINOGENIC HAZARD INDICES  
UPPER BOUND REASONABLE CASE ESTIMATE**

**HYPOTHETICAL FUTURE-USE SCENARIO**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Subchronic Intake		Subchronic Hazard Index	Chronic Intake (mg/kg/day)	RID (AIC) (mg/kg/day)	Chronic Hazard Index	
				(mg/kg/day)	AIS (mg/kg/day)					
Adults	Ground water	Dermal Contact	trans-1,2-Dichloroethene	3.78E-07	2.00E-01	1.89E-06	6.84E-08	1.00E-02	6.84E-06	
			Trichloroethene	4.12E-06	NA	0.00E+00	6.16E-07	NA	0.00E+00	
			tris(2-Chloropropyl)phosphate	3.78E-03	1.25E+00	3.02E-03	3.08E-04	1.25E-01	2.46E-03	
		Ingestion	trans-1,2-Dichloroethene	3.15E-04	2.00E-01	1.58E-03	5.72E-05	1.00E-02	5.72E-03	
			Trichloroethene	3.43E-03	NA	0.00E+00	5.15E-04	NA	0.00E+00	
			tris(2-Chloropropyl)phosphate	3.15E+00	1.25E+00	2.52E+00	2.57E-01	1.25E-01	2.06E+00	
	Inhalation	trans-1,2-Dichloroethene	4.68E-04	2.00E-01	2.34E-03	1.70E-04	1.00E-02	1.70E-02		
		Trichloroethene	5.10E-03	NA	0.00E+00	1.53E-03	NA	0.00E+00		
		tris(2-Chloropropyl)phosphate	4.68E+00	1.25E+00	3.74E+00	7.65E-01	1.25E-01	6.12E+00		
					<b>Total =</b>		<b>6.27E+00</b>	<b>Total =</b>		<b>8.20E+00</b>
	Children 6-12	Ground water	Dermal Contact	trans-1,2-Dichloroethene	5.23E-07	2.00E-01	2.62E-06	9.54E-08	1.00E-02	9.54E-06
				Trichloroethene	5.70E-06	NA	0.00E+00	8.59E-07	NA	0.00E+00
tris(2-Chloropropyl)phosphate				5.23E-03	1.25E+00	4.18E-03	4.29E-04	1.25E-01	3.43E-03	
Ingestion			trans-1,2-Dichloroethene	3.80E-04	2.00E-01	0.00E+00	6.90E-05	1.00E-02	6.90E-03	
			Trichloroethene	4.14E-03	NA	0.00E+00	6.21E-04	NA	0.00E+00	
			tris(2-Chloropropyl)phosphate	3.80E+00	1.25E+00	3.04E+00	3.11E-01	1.25E-01	2.49E+00	
Inhalation		trans-1,2-Dichloroethene	6.27E-04	2.00E-01	3.14E-03	2.28E-04	1.00E-02	2.28E-02		
		Trichloroethene	6.84E-03	NA	0.00E+00	2.05E-03	NA	0.00E+00		
		tris(2-Chloropropyl)phosphate	6.27E+00	1.25E+00	5.02E+00	1.03E+00	1.25E-01	8.24E+00		
				<b>Total =</b>		<b>8.06E+00</b>	<b>Total =</b>		<b>1.08E+01</b>	
Children 2-6		Ground water	Dermal Contact	trans-1,2-Dichloroethene	6.32E-07	2.00E-01	3.16E-06	1.15E-07	1.00E-02	1.15E-05
				Trichloroethene	6.89E-06	NA	0.00E+00	1.04E-06	NA	0.00E+00
	tris(2-Chloropropyl)phosphate			6.32E-03	1.25E+00	5.06E-03	5.18E-04	1.25E-01	4.14E-03	
	Ingestion		trans-1,2-Dichloroethene	6.88E-04	2.00E-01	0.00E+00	6.25E-05	1.00E-02	6.25E-03	
			Trichloroethene	7.50E-03	NA	0.00E+00	1.13E-03	NA	0.00E+00	
			tris(2-Chloropropyl)phosphate	6.88E+00	1.25E+00	5.50E+00	5.63E-01	1.25E-01	4.50E+00	
	Inhalation	trans-1,2-Dichloroethene	6.18E-04	2.00E-01	3.44E-03	2.24E-04	2.00E-02	1.12E-02		
		Trichloroethene	6.72E-03	NA	0.00E+00	2.02E-03	NA	0.00E+00		
		tris(2-Chloropropyl)phosphate	6.18E+00	1.25E+00	5.50E+00	1.01E+00	1.25E-01	8.08E+00		
					<b>Total =</b>		<b>1.10E+01</b>	<b>Total =</b>		<b>1.26E+01</b>

*Bold Italics indicates a change to the table.*

NA - Not applicable

**Lifetime-weighted**

**Subchronic Hazard = 6.71E+00**

**Lifetime-weighted**

**Chronic Hazard = 8.69E+00**

**LIFETIME WEIGHTED = CHRONIC INTAKE x RiD and represents the upper bound reasonable case**

**SAMPLE CALCULATION**

tris(2-chloropropyl)phosphate in adults; dermal contact with ground water

Chronic hazard index = Chronic daily intake/RiD

= 0.000308/0.125

= 0.00246

trans-1,2-Dichloroethene = 3.82E-03

Trichloroethene = 0.00E+00

tris(2-Chloropropyl)phosphate = 6.70E+00

trans-1,2-Dichloroethene = 2.30E-02

Trichloroethene = 0.00E+00

tris(2-Chloropropyl)phosphate = 8.66E+00

Total chronic hazard index = sum of all exposure routes for all indicators.

Lifetime-weighted Chronic Hazard = {Total chronic adults \*(58/68)} + {total chronic child 6-12 \*(6/68)} + {total chronic child 2-6 \*(4/68)}

AR301076



Regional Review: No  
 Contaminants: ~~not volatile~~ tris (2 chloro propyl)  
 Site Type: Multi-Source Groundwater  
 Site Name: New Castle Spill Site, New Castle, DE  
 Date: 9/28/89  
 Source Control: No  
 EPA Region: III

Role of Comments In Remedy Selection: State 2 Public 2 PRP 2

Lead: RI/FS RS RD RP RA RP

Cost: \$466,147.00  
 Site Area: ~~not~~ approximately 6 acres

Waste Volume: ~~not~~ unknown  
 Treated Volume: ~~not~~ unknown

MAJOR CONTAMINANT(S)	CONTAMINANT LEVELS	CLEANUP GOALS	BASELINE RISK	RISK WITH CLEANUP GOAL
GW: <del>tris</del> phosphate	Up to: 110,000 ug/l	<del>4.4 mg/l?</del>	$1 \times 10^{-6}$	<del>Not available</del>
TCE	120 ug/l		<del><math>1 \times 10^{-7}</math></del>	Not applicable based on
1,2 - Dichloro-ethane	120 ug/l		0.0	
Sub surface soil:				
Tris	11.8 mg/kg		0.0	not applicable
Fluoranthene	3.6 mg/kg			
Phenanthrene	4.4 mg/kg			
Benzo(b)fluoranthene	2.5 mg/kg			

**DESCRIPTION OF SITE**

The site is a former manufacturing plant of the Witco Corporation, which produced ~~materials~~ used in the production of plastic ~~bars~~ ~~with catalyst~~. Raw materials and product were stored at ~~the~~ in 55 gallon drums at this facility. ~~Leakage of drums~~ resulted in groundwater and soil contamination with tris (2 chloro propyl) phosphate, a flame retardant. TCE is present in the groundwater but appears to be ~~from~~ ~~of~~ source up gradient of the fac

**DESCRIPTION OF SITE REMEDY**

Monitored natural attenuation of tris. Institutional controls restricting well development in the upper ~~of~~ Columbia aquifer.

AR301077

**TABLE 6-4 Continued  
NEW CASTLE SPILL SITE  
ASSESSMENT OF NONCARCINOGENIC HAZARD INDICES  
UPPER BOUND WORST CASE ESTIMATE**

**HYPOTHETICAL FUTURE-USE SCENARIO**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Subchronic Intake	AIS	Subchronic Hazard Index	Subchronic Intake	RfD (AIC)	Upper Bound Chronic
				(mg/kg/day)	(mg/kg/day)		(mg/kg/day)	(mg/kg/day)	Hazard Index
Adults	Ground water	Dermal Contact	trans-1,2-Dichloroethene	3.78E-07	2.00E-01	1.89E-06	3.78E-07	1.00E-02	3.78E-05
			Trichloroethene	4.12E-06	NA	0.00E+00	4.12E-06	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	3.78E-03	1.25E+00	3.02E-03	3.78E-03	1.25E-01	3.02E-02
		Ingestion	trans-1,2-Dichloroethene	3.15E-04	2.00E-01	1.58E-03	3.15E-04	1.00E-02	3.15E-02
			Trichloroethene	3.43E-03	NA	0.00E+00	3.43E-03	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	3.15E+00	1.25E+00	2.52E+00	3.15E+00	1.25E-01	2.52E+01
		Inhalation	trans-1,2-Dichloroethene	4.68E-04	2.00E-01	2.34E-03	4.68E-04	1.00E-02	4.68E-02
			Trichloroethene	5.10E-03	NA	0.00E+00	5.10E-03	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	4.68E+00	1.25E+00	3.74E+00	4.68E+00	1.25E-01	3.74E+01
								<b>Total Subchronic Hazard Index=</b>	<b>6.27E+00</b>
Children 6-12	Ground water	Dermal Contact	trans-1,2-Dichloroethene	5.23E-07	2.00E-01	2.62E-06	5.23E-07	1.00E-02	5.23E-05
			Trichloroethene	5.70E-06	NA	0.00E+00	5.70E-06	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	5.23E-03	1.25E+00	4.18E-03	5.23E-03	1.25E-01	4.18E-02
		Ingestion	trans-1,2-Dichloroethene	3.80E-04	2.00E-01	0.00E+00	3.80E-04	1.00E-02	3.80E-02
			Trichloroethene	4.14E-03	NA	0.00E+00	4.14E-03	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	3.80E+00	1.25E+00	3.04E+00	3.80E+00	1.25E-01	3.04E+01
		Inhalation	trans-1,2-Dichloroethene	6.27E-04	2.00E-01	3.14E-03	6.27E-04	1.00E-02	6.27E-02
			Trichloroethene	6.84E-03	NA	0.00E+00	6.84E-03	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	6.27E+00	1.25E+00	5.02E+00	6.27E+00	1.25E-01	5.02E+01
								<b>Total Subchronic Hazard Index=</b>	<b>8.06E+00</b>
Children 2-6	Ground water	Dermal Contact	trans-1,2-Dichloroethene	6.32E-07	2.00E-01	3.16E-06	6.32E-07	1.00E-02	6.32E-05
			Trichloroethene	8.89E-06	NA	0.00E+00	8.89E-06	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	6.32E-03	1.25E+00	5.06E-03	6.32E-03	1.25E-01	5.06E-02
		Ingestion	trans-1,2-Dichloroethene	8.88E-04	2.00E-01	0.00E+00	8.88E-04	1.00E-02	8.88E-02
			Trichloroethene	7.50E-03	NA	0.00E+00	7.50E-03	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	8.88E+00	1.25E+00	5.50E+00	8.88E+00	1.25E-01	5.50E+01
		Inhalation	trans-1,2-Dichloroethene	6.16E-04	2.00E-01	3.44E-03	6.16E-04	2.00E-02	3.08E-02
			Trichloroethene	6.72E-03	NA	0.00E+00	6.72E-03	NA	0.00E+00
			tris(2-Chloropropyl)phosphate	6.16E+00	1.25E+00	5.50E+00	6.16E+00	1.25E-01	4.93E+01
								<b>Total Subchronic Hazard Index=</b>	<b>1.10E+01</b>

*Italics indicates a change to the table.*

**Lifetime-weighted Subchronic Hazard = 6.71E+00**      **Upper Bound Worst Case Estimate of Chronic Hazard = 6.68E+01**

Upper bound worst case estimate of chronic hazard index is based on the maximum concentration detected times the RfD.

NA - Not applicable

**SAMPLE CALCULATION**

tris(2-chloropropyl)phosphate in adults; dermal contact with ground water  
 Chronic hazard Index = Subchronic daily intake/RfD  
 = 0.00378/0.125  
 = 0.0302

trans-1,2-Dichloroethene = 3.82E-03      trans-1,2-Dichloroethene = 8.16E-02  
 Trichloroethene = 0.00E+00      Trichloroethene = 0.00E+00  
 tris(2-Chloropropyl)phosphate = 6.70E+00      tris(2-Chloropropyl)phosphate = 6.67E+01

Total chronic hazard index = sum of all exposure routes for all indicators.

Lifetime-weighted Chronic Hazard = {Total chronic adults \*(58/68)}+{total chronic child 6-12 \*(6/68)}+{total chronic child 2-6 \*(4/68)}

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hazard indices. The lifetime-weighted (i.e., reasonable case) subchronic and chronic hazard indices exceeded one when the hazard indices for the indicator compounds were summed. When these indices exceed one, the hazard index is recalculated based on target organ effects. Compounds, which affect the same target organ, are summed. When the indices are recalculated as in Table 6-4, only the lifetime-weighted subchronic and chronic exposure to tris concentrations in ground water still exceeded one. That is, only exposure to the tris detected in ground water may pose a hazard.

When an upper bound worst-case hazard index was calculated, this hypothetical chronic index exceeded one. Again, only the tris levels exceeded the guideline.

#### 6.4 Calculation of Carcinogenic Risk

An assessment of potential carcinogenic risks for the present, existing conditions at the site and marsh area is presented in Table 6-5. Only chronic intakes are used to calculate carcinogenic risk. The site risk under the present, existing conditions scenario is approximated as zero. The upper bound worst-case risk was also approximated at zero.

An assessment of potential carcinogenic risks for the hypothetical, future-use scenario at the site is presented in Table 6-6. Again, only chronic intakes are used to calculate carcinogenic risks. The lifetime-weighted average (i.e., upper bound reasonable case) from exposure to potential carcinogens detected at NCSS is  $3 \times 10^{-5}$ . U.S. EPA's guidelines for carcinogenic risks at hazardous waste sites are  $1 \times 10^{-4}$  to

**TABLE 6-5  
NEW CASTLE SPILL SITE  
ASSESSMENT OF CARCINOGENIC RISK  
UPPER BOUND REASONABLE CASE ESTIMATE**

**PRESENT, EXISTING CONDITIONS**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Chronic Intake (mg/kg/day)	CPF 1/(mg/kg/day)	Route/Compound Specific Risk
Adults	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	0E+00
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	3.89E-09	NA	
	Soils	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	2.13E-08	NA	
Children 6-12	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	0E+00
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	1.63E-07	NA	
	Soils	<b><i>Dermal Contact *</i></b>	trans-1,2-Dichloroethene	0.00E+00	NA	
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	1.61E-06	NA	
Child Age 2-6	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	NA
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	1.97E-07	NA	
	Soils	<b><i>Dermal Contact**</i></b>	trans-1,2-Dichloroethene	0.00E+00	NA	
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	4.76E-06	NA	

Lifetime Weighted = 0E+00

**Bold Italics indicates a change in the table.**

**\* Included incidental ingestion of 50 mg**

**\*\* Included incidental ingestion of 100 mg**

**LIFETIME WEIGHTED = CHRONIC INTAKE x CPF and represents the upper bound reasonable case**

NA - Not applicable

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**TABLE 6-5 Continued  
NEW CASTLE SPILL SITE  
ASSESSMENT OF CARCINOGENIC RISK  
UPPER BOUND WORST CASE ESTIMATE**

**PRESENT, EXISTING CONDITIONS**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Subchronic Intake (mg/kg/day)	CPF 1/(mg/kg/day)	Route/Compound Specific Risk
Adults	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	0E+00
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	1.09E-06	NA	
	Soils	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	0E+00
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	1.12E-05	NA	
Children 6-12	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	0E+00
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	1.52E-06	NA	
	Soils	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	0E+00
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	2.81E-05	NA	
Child Age 2-6	Surface Water	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	NA
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	1.83E-06	NA	
	Soils	Dermal Contact	trans-1,2-Dichloroethene	0.00E+00	NA	0E+00
			Trichloroethene	0.00E+00	1.10E-02	
			Tris(2-chloropropyl)phosphate	8.28E-05	NA	

NA - Not applicable

Lifetime Weighted Average = 0E+00

Upper bound worst case estimate of carcinogenic risk is based on the maximum concentration detected times the CPF.

AR301081



**TABLE 6-6  
NEW CASTLE SPILL SITE  
ASSESSMENT OF CARCINOGENIC RISK  
UPPER BOUND REASONABLE CASE ESTIMATE**

**HYPOTHETICAL FUTURE-USE SCENARIO**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Chronic Intake (mg/kg/day)	CPF 1/(mg/kg/day)	Route/Compound Specific Risk
Adults	Ground water	Dermal Contact	trans-1,2-Dichloroethene	6.84E-08	NA	7E-09
			Trichloroethene	6.18E-07	1.10E-02	
			Tris(2-chloropropyl)phosphate	3.08E-04	NA	
		Ingestion	trans-1,2-Dichloroethene	5.72E-05	NA	6E-06
			Trichloroethene	5.15E-04	1.10E-02	
			Tris(2-chloropropyl)phosphate	2.57E-01	NA	
		Inhalation	trans-1,2-Dichloroethene	1.70E-04	NA	2E-05
			Trichloroethene	1.53E-03	1.30E-02	
			Tris(2-chloropropyl)phosphate	7.65E-01	NA	
			<b>TOTAL-</b>		<b>3E-05</b>	
Children 6-12	Ground water	Dermal Contact	trans-1,2-Dichloroethene	9.54E-08	NA	9E-09
			Trichloroethene	8.59E-07	1.10E-02	
			Tris(2-chloropropyl)phosphate	4.29E-04	NA	
		Ingestion	trans-1,2-Dichloroethene	6.90E-05	NA	7E-06
			Trichloroethene	6.21E-04	1.10E-02	
			Tris(2-chloropropyl)phosphate	3.11E-01	NA	
		Inhalation	trans-1,2-Dichloroethene	2.28E-04	NA	3E-05
			Trichloroethene	2.05E-03	1.30E-02	
			Tris(2-chloropropyl)phosphate	1.03E+00	NA	
			<b>TOTAL-</b>		<b>3E-05</b>	
Child Age 2-6	Ground water	Dermal Contact	trans-1,2-Dichloroethene	1.15E-07	NA	1E-08
			Trichloroethene	1.04E-06	1.10E-02	
			Tris(2-chloropropyl)phosphate	5.18E-04	NA	
		Ingestion	trans-1,2-Dichloroethene	1.25E-04	NA	1E-05
			Trichloroethene	1.13E-03	1.10E-02	
			Tris(2-chloropropyl)phosphate	5.63E-01	NA	
		Inhalation	trans-1,2-Dichloroethene	2.24E-04	NA	3E-05
			Trichloroethene	2.02E-03	1.30E-02	
			Tris(2-chloropropyl)phosphate	1.01E+00	NA	
			<b>TOTAL-</b>		<b>4E-05</b>	

*Bold italics indicates a change to the table.*

Lifetime Weighted = **3E-05**

NA - Not applicable

**LIFETIME WEIGHTED = CHRONIC INTAKE x CPF and represents an upper bound reasonable case.**

Sample calculation adults exposed to trichloroethene in ground water

Route/Compound specific risk = chronic daily intake \* carcinogenic potency factor

= 0.000000616/0.011 or 0.000000007

Lifetime Weighted Average = {(TCR adults)\*(58/68)}+{(TCRchild 6-12)\*(6/68)}+{(TCRchild 2-6)\*(4/68)}

where TCR is Total Carcinogenic Risk

AR301082



**TABLE 6-6 Continued  
NEW CASTLE SPILL SITE  
ASSESSMENT OF CARCINOGENIC RISK  
UPPER BOUND WORST CASE ESTIMATE**

**HYPOTHETICAL FUTURE-USE SCENARIO**

Exposed Population	Exposure Media	Route of Exposure	Indicator Compound	Subchronic Intake (mg/kg/day)	CPF 1/(mg/kg/day)	Route/Compound Specific Risk
Adults	Ground water	Dermal Contact	trans-1,2-Dichloroethene	3.78E-07	NA	5E-08
			Trichloroethene	4.12E-06	1.10E-02	
			Tris(2-chloropropyl)phosphate	3.78E-03	NA	
		Ingestion	trans-1,2-Dichloroethene	3.15E-04	NA	4E-05
			Trichloroethene	3.43E-03	1.10E-02	
			Tris(2-chloropropyl)phosphate	3.15E+00	NA	
		Inhalation	trans-1,2-Dichloroethene	4.68E-04	NA	7E-05
			Trichloroethene	5.10E-03	1.30E-02	
			Tris(2-chloropropyl)phosphate	4.68E+00	NA	
		<b>TOTAL-</b>				
Children 6-12	Ground water	Dermal Contact	trans-1,2-Dichloroethene	5.23E-07	NA	6E-08
			Trichloroethene	5.70E-06	1.10E-02	
			Tris(2-chloropropyl)phosphate	5.23E-03	NA	
		Ingestion	trans-1,2-Dichloroethene	3.80E-04	NA	5E-05
			Trichloroethene	4.14E-03	1.10E-02	
			Tris(2-chloropropyl)phosphate	3.80E+00	NA	
		Inhalation	trans-1,2-Dichloroethene	6.27E-04	NA	9E-05
			Trichloroethene	6.84E-03	1.30E-02	
			Tris(2-chloropropyl)phosphate	6.27E+00	NA	
		<b>TOTAL-</b>				
Child Age 2-6	Ground water	Dermal Contact	trans-1,2-Dichloroethene	6.32E-07	NA	8E-08
			Trichloroethene	6.89E-06	1.10E-02	
			Tris(2-chloropropyl)phosphate	6.32E-03	NA	
		Ingestion	trans-1,2-Dichloroethene	6.88E-04	NA	8E-05
			Trichloroethene	7.50E-03	1.10E-02	
			Tris(2-chloropropyl)phosphate	6.88E+00	NA	
		Inhalation	trans-1,2-Dichloroethene	6.16E-04	NA	9E-05
			Trichloroethene	6.72E-03	1.30E-02	
			Tris(2-chloropropyl)phosphate	6.16E+00	NA	
		<b>TOTAL-</b>				

NA - Not applicable

Upper Bound Estimate of Risk = 1E-04

Upper bound worst case estimate of carcinogenic risk is the maximum concentration detected times the CPF.

Sample calculation adults exposed to trichloroethene in ground water

Route/Compound specific risk = chronic daily intake \* carcinogenic potency factor

= 0.00000412/0.011 or 0.0000005

Lifetime Weighted Average = {(TCR adults)\*(58/68)}+{(TCRchild 6-12)\*(6/68)}+{(TCRchild 2-6)\*(4/68)}

where TCR is Total Carcinogenic Risk

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$1 \times 10^{-7}$ . The calculated reasonable-case risk for NCSS is within U.S. EPA's guidelines.

An upper bound estimate of cancer risk can be determined using the subchronic exposures (i.e., maximum concentrations) and the carcinogenic potency factors (CPF). The CPF is expressed as the lifetime cancer risk per mg/kg body weight/day. This factor is equivalent to  $q_1^*$  (i.e., slope of the line) when it is based on animal study data evaluated by the multistage model. This factor is an estimated upper 95 percent confidence limit of the carcinogenic potency of the chemical. That is, only 5 percent chance exists that the probability of response could be greater than the estimated value of the basis of the experimental data used. Predicted risk may overestimate the actual risk at a site but this method is used so that the carcinogenic risk is not underestimated.

The calculated upper bound worst-case carcinogenic risk is  $1 \times 10^{-4}$  for the hypothetical future-use scenario. This value is equal to the upper limit of U.S. EPA's recommended range of  $1 \times 10^{-4}$  to  $1 \times 10^{-7}$  for hazardous waste sites. For perspective, this calculated value that represents the worst possible exposure conditions is very conservative, and may have overestimated the risk by up to 95 percent.

#### 6.5 Risk Perspective

No additional lifetime risk is expected to result from living in an existing or future residential area adjacent to NCSS. It is important to note that U.S. EPA's methodology for calculating cancer risk is based upon a set of conservative assumptions and

does not provide an accurate estimate of risk, but rather a probability that the risk will not exceed the derived estimate. The uncertainty inherent in U.S. EPA's methodology is described in Section 2.

The lifetime risk of cancer from all causes is 0.20 to 0.25. That is, approximately 20 to 25 percent of all people develop cancer in their lifetimes. No estimated additional lifetime risk of cancer from living near NCSS is expected to occur.

#### 6.6 Environmental Assessment

According to the Delaware State Wetland Map for New Castle County (Photograph No. 35-8) and ERM's (1989) wetlands delineation, the New Castle Spill Site is located to the northeast of emergent and forested wetland habitats. ERM's investigation (1989) describes a well-established wetland ecosystem supporting diverse populations of biota. Complete scientific listings of the biota encountered during the wetlands survey are contained in Section 4.6 of the Remedial Investigation (ERM 1989).

Two series of surface water and sediment sampling at six sampling stations in the aforementioned wetland were conducted. The initial sampling effort (April 1988) reported non-detectable levels of tris(2-chloropropyl)phosphate (tris) in all six sediment samples. During the initial sampling, tris was detected in surface water at four of the six sampling stations with levels between 22.4 and 42 ug/l. The maximum tris concentration was reported for station WS-1, located closest to the proposed source area. A Phase II sampling effort was conducted in November of 1988; sediment and water samples were collected at the same six

stations as the April 1988 effort. In sediments, 402 ug/l and 300 ug/l of tris were reported for stations SD-3 and SD-4, respectively. Surface water samples revealed low levels of tris at stations WS-1, WS-2, WS-3, and WS-4. The concentration of tris detected ranged from 1.15 ug/l to 5.37 ug/l.

The Phase II sampling effort included an analysis for trichloroethene (TCE) at the six sampling stations. TCE was not detected in any of the six surface water samples. An estimated TCE value of 3 ug/kg was reported for the sediments collected at station SD-1; all other sampling locations were characterized by non-detectable TCE levels.

Based on the volatility and moderate water solubility of tris, appreciable water column concentrations would not be anticipated for this compound. Although there is a diminutive quantity of information available on the sorptive behavior of tris, its calculated  $K_{OC}$  (i.e., 9) suggests sorption might not be a major fate process. Considerable sorptive capacity would explain the levels of tris detected at stations SD-3 and SD-4 during the second sampling effort. There is a paucity of data available regarding the ecotoxic properties of tris. Although definitive data is lacking, a comparison of the tris levels detected in the wetlands with toxicity information for similar compounds (e.g., tris(2,3-bromopropyl)phosphate) suggests the tris concentrations detected are not of the magnitude to elicit toxicity in aquatic species (i.e., rainbow trout, sac fry,  $LC_{50} = 240$  ug/l; fingerlings -  $LC_{50} = 1,450$  ug/l), (Verschuere 1983). The U.S. EPA (1986) has not evaluated tris; therefore, no water quality criteria are available for a comparison.

Trichloroethene was not reported in the wetlands water samples and was estimated once in sediments at 3 ug/kg. The infrequent detection of TCE in the wetlands indicate its presence is suspect and does not pose a hazard to the biota occurring in the wetlands.

A comparison of the surface water concentrations to Ambient Water Quality Criteria (AWQC) and toxicity values is presented in Table 6-7. The measured surface water concentrations do not exceed either the AWQC or listed toxicity value for the compounds. Sediment criteria are not available at this time for a comparison with actual sediment values.

In summary, the low levels of tris detected and the infrequent detection of TCE suggest no imminent threat to the biotic component of the wetlands ecosystem exists from the contaminants migrating off the New Castle Spill Site.

**TABLE 6-7**

**COMPARISON OF SURFACE WATER  
CONCENTRATIONS TO AWQC  
AND TOXICITY VALUES**

COMPOUND	MAXIMUM SURFACE WATER CONCENTRATION (mg/L)	AWQC (mg/L)		TOXICITY VALUE * (mg/L)
		ACUTE	CHRONIC	
trans-1,2-Dichloroethene	Not Analyzed	11.6	-	None Listed
Trichloroethene	Not Detected	45	21.9	40.7 **
tris(2-Chloropropyl)phosphate	0.042	None Established		0.240 ***

AWQC = Ambient Water Quality Criteria (US EPA, 1986)

\*Most sensitive aquatic species listed in Verschueren, 1983

\*\*96 hour LC50 (flow-through test) - *Pimephales promelas* Rafinesque (fathead minnows)

\*\*\*No toxicity values or AWQC for tris(2-chloropropyl)phosphate

Based on more toxic compound - tris(2,3-dibromopropyl)phosphate

96 hour LC50 (static test) - rainbow trout sac fry

- fingerling (1.45 ppm)

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## SECTION 7

### CONCLUSIONS

The Endangerment Assessment (EA) for the New Castle Spill Site has examined the existing data, identified compounds of concern, evaluated potential exposure pathways, and approximated potential risks or hazards to human and environmental receptors. Risks and hazards calculated in this report are representative of a present maximum, which should theoretically decrease with time. In addition, the risks calculated in this assessment are derived from the worst-case assumption under hypothetical future-use conditions. The methods employed, as recommended by U.S. EPA, are highly conservative and may result in either over-or-under-estimation of the potential risk depending on the underlying assumption (as discussed in Sections 2.6 and 6.5).

An exposed population was not available for assessment of potential exposure to ground water via residential use under present, existing conditions scenario because 1) drinking water is supplied by municipal or commercial means, 2) private wells in Columbia aquifer in the downgradient direction do not exist, 3) closest municipal well is located approximately 0.7 miles downgradient, and 4) there are no users of the Columbia aquifer since the Potomac aquifer (regional potable aquifer) is available. Therefore, a hypothetical, future-use ground water scenario involving adults, children 6 to 12 years, and children 2 to 6

years and consisting of a well installed in the Columbia aquifer at the property boundary was employed to determine the hazard or risk to a population using ground water leaving the NCSS property.

Upper bound reasonable case and worst case estimates were made for each exposure scenario. The reasonable case is based on realistic exposure durations, frequencies, and pathways; while the worst case assumes that an individual may be chronically exposed to the highest concentrations detected at the site. This type of estimate means a 95 percent probability exists that the risk may be overestimated with a 5 percent chance of underestimating the risk.

All of the carcinogenic risks calculated were within U.S. EPA's range. Table 7-1 is a summary of the calculated carcinogenic risks and noncarcinogenic hazard indices for the New Castle Spill Site. The risk/hazard from present conditions (i.e., exposure to surface water and sediments) were orders of magnitude below EPA's guidelines. That is, exposure by a population to compounds detected in the surface water or sediments does not threaten human health or the environment. Only the subchronic and chronic hazard indices for the hypothetical case exceeded U.S. EPA's guidelines of one. However, a population is not currently exposed to the Columbia aquifer and although these calculated intakes exceed one, in reality, exposure to these intakes may never occur.

The conclusions of the environmental assessment are that there is 1) no exceedance of ambient water quality criteria by measured surface water concentrations, 2) no exceedance of the toxicity

**TABLE 7 - 1  
SUMMARY OF CALCULATED CARCINOGENIC RISKS  
AND NONCARCINOGENIC HAZARD INDICES FOR  
THE NEW CASTLE SPILL SITE**

<u>PRESENT, EXISTING CONDITIONS*</u>		<u>Upper bound Reasonable Case</u>	<u>Upper Bound Worst Case</u>	<u>US EPA'S RECOMMENDED GUIDELINE</u>
<b>CARCINOGENIC RISK</b>		~ 0E+00	~ 0E+00	1E-04 to 1E-07
<b>NONCARCINOGENIC HAZARD INDEX</b>	Subchronic	1.45E-05	1.45E-05	1
	Chronic	3.76E-06	1.45E-04	1
<u>HYPOTHETICAL FUTURE-USE SCENARIO**</u>		<u>Upper bound Reasonable Case</u>	<u>Upper Bound Worst Case</u>	<u>US EPA'S RECOMMENDED GUIDELINE</u>
<b>CARCINOGENIC RISK</b>		3E-05	1E-04	1E-04 to 1E-07
<b>NONCARCINOGENIC HAZARD INDEX</b>	Subchronic	<b>6.71E+00</b>	<b>6.71E+00</b>	1
	Chronic	<b>8.89E+00</b>	<b>6.68E+01</b>	1

Bold value indicates an exceedance of US EPA's guideline

Upper bound reasonable case represents the average concentration detected times either the RfD or CPF.

Upper bound worst case represents the maximum concentration detected times either the RfD or CPF.

~ = approximate

\* - Surface water and soils exposure

\*\* - Ground water exposure

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value for the most sensitive aquatic species tested, 3) no imminent threat to the biotic component of the wetlands ecosystem from contaminants migrating off the New Castle Spill Site, and 4) low qualitative potential for bioaccumulation of these compounds in the aquatic species and less potential for biomagnification of the food chain to a human population.

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## ACRONYMS

US EPA	United States Environmental Protection Agency
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act
SUPERFUND	Synonym for CERCLA
IS	Indicator Score
CT	Concentration times the Toxicity Constant for each Medium
ERM	Environmental Resources Management, Inc.
PC	Potential Carcinogen
NC	Noncarcinogen
IARC	International Agency for Research on Cancer
CAG	Carcinogen Assessment Group US EPA
OSHA	Occupational Safety and Health Administration
TWA	Time Weighted Average
NTP	National Toxicology Program
ACGIH	American Conference of Governmental Industrial Hygienists
MCL	Maximum Contaminant Level
TLV	Threshold Limit Value
RFD	Reference Dose (=AIC, acceptable intake chronic)
ADI	Allowable Daily Intake
SDI	Subchronic Daily Intake
CDI	Chronic Daily Intake
NCP	National Oil and Hazardous Substances Pollution Contingency Program
NAAQS	National Ambient Air Quality Standards
AIS	Acceptable Intake Subchronic
CPF	Carcinogenic Potency Factor
IC	Indicator Compound
WHO	World Health Organization
DNREC	Delaware Natural Resources and Environmental Control
NCSS	New Castle Spill Site
HENRY'S LAW CONSTANT	The mass of a slightly soluble gas that dissolves in a definite mass of a liquid at a given temperature
Koc	A measure of the tendency for organics to be absorbed by soil or sediment
Kow	A measure of how a chemical is distributed at equilibrium between octanol and water
Kb	A measure of the partitioning of a chemical between microorganisms and water in the water column
TOXICOKINETICS	Quantitation of the time course of chemical absorption, distribution, biotransformation, and excretion OR kinetics of the chemical
TERATOGENIC	Of, or relating to, or causing developmental malfunctions and monstrosities

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APPENDIX A  
US EPA MODIFICATION TO THE IARC CLASSIFICATION  
OF CARCINOGENS

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## IARC CLASSIFICATION SYSTEM

The International Agency for Research on Cancer (IARC) initiated a research program in 1971 to evaluate the carcinogenic risk of chemicals to humans. In 1982, IARC developed a system for categorization of carcinogens based on the strength of evidence for carcinogenicity. IARC does not assess the relevance of experimental laboratory animal data to human risk. IARC's system is in sharp contrast to the EPA categorization system which was adapted from the 1982 IARC system and is a basic element of the risk assessment process. The EPA categorization system differs from the IARC system in that it stresses the weight-of-evidence approach which incorporates the balancing of positive and negative studies. During January 1987, IARC revised its categorization system resulting in changes that incorporates some new features of the EPA system, but digress from it in other ways.

The IARC categorization system is based on a definition of chemical carcinogenesis as the induction by chemicals of neoplasms that are not usually observed, of neoplasms that are commonly observed, and/or of more neoplasms than are usually found.

The evidence for carcinogenicity in humans by IARC can be derived from three types of studies:

1. Case reports of individual cancer patients which include a history of exposure to the chemical in question.
2. Descriptive epidemiological studies.
3. Analytical epidemiological studies (case control and cohort).

The degrees of evidence for carcinogenicity in studies of humans by IARC are defined as:

1. Sufficient evidence of carcinogenicity, which indicates that there is a casual relationship between the agent and human cancer.
2. Limited evidence of carcinogenicity, which indicates that a casual interpretation is credible, but that alternative explanations, such as change, bias, or confounding could not be adequately excluded.

## EPA CLASSIFICATION SYSTEM

EPA (Fed Register, 1986) has made the following modifications of the IARC (IARC, 1982) approach to classifying human and animal studies. For human studies:

1. "The observation of a statistically significant association between an agent and life-threatening benign tumors in humans is included in the evaluations of risk to humans."
2. "A 'no-data available' classification is added."
3. "A "no evidence of carcinogenicity" classification is added. This classification indicates that no association was found between exposure and increased risk of cancer in well-conducted, well-designed, independent analytical epidemiologic studies."

For animal studies:

1. An increased incidence of combined benign and malignant tumors will be considered to provide sufficient evidence of carcinogenicity if the other criteria defining the "sufficient" category of evidence are met.
2. A statement that increased incidence of benign tumors alone provides "limited" evidence of carcinogenicity is added.
3. Under specific circumstances, such as the production of neoplasms that occur with high spontaneous background incidence, the evidence may be decreased to "limited" if warranted by specific information available on the agent.
4. A "no data available" classification has been added.
5. A "no evidence of carcinogenicity" classification is also added.

Agents that are judged to be in the EPA weight-of-evidence stratification Groups A and B are to be regarded as suitable for quantitative risk assessments. The appropriateness of quantifying the risks from agents in Group C, specifically agents that are at the boundary of Group C and D, would be judged on a case-by-case basis. Agents that are judged to be in Groups D and E should generally not be evaluated as carcinogens using quantitative risk assessments.

Evidence of carcinogenicity from human studies comes from three main sources:

1. Case reports of individual cancer patients who were exposed to the agent(s).
2. Descriptive epidemiological studies.
3. Analytical epidemiologic (case control and cohort) studies.

Three criteria must be met before a causal association can be inferred between exposure and cancer in humans:

1. There is no identified bias which can explain the association.
2. The possibility of confounding has been considered and ruled out as explaining the association.
3. The association is unlikely to be due to chance.

The weight-of-evidence for carcinogenicity from studies in humans can be categorized by:

- a. Sufficient evidence of carcinogenicity, which indicates that there is a causal relationship between the agent and human cancer.
- b. Limited evidence of carcinogenicity, which indicates that a causal interpretation is credible, but that alternative explanations such as change, bias, or confounding, could not be adequately excluded.
- c. Inadequate evidence.
  - i. There were few pertinent data, or
  - ii. The available studies, while showing evidence of association, did not exclude chance, bias or confounding.
4. No evidence.
5. No data available.

Assessments of weight-of-evidence for carcinogenicity from studies in experimental animals are classified into five groups:

1. Sufficient evidence of carcinogenicity, which indicates an increased incidence of malignant tumors or combined malignant and benign tumors:
  - a. In multiple species or strains; or
  - b. In multiple experiments (preferably with different routes of administration or using different dose levels); or
  - c. To an unusual degree in a single experiment with regard to incidence, site or type of tumor, or age at onset.
2. Limited evidence of carcinogenicity.
  - a. Studies involve a single species, strain, or experiment; or
  - b. The experiments are restricted by inadequate dose levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or
  - c. An increase in the incidence of benign tumors only.
3. Inadequate evidence.
4. No evidence of carcinogenicity.
5. No data.

The categorization of overall evidence of carcinogenicity is subdivided into five groups.

Group A: Human carcinogens are used only when there is sufficient evidence from epidemiologic studies to support the causal association between exposure to agent(s) and cancer.

Group B: Probable human carcinogens include agents for which the evidence of human carcinogenicity from epidemiologic studies ranges from almost "sufficient" to "inadequate." B1 is reserved for agents for which there is at least limited evidence of carcinogenicity to humans from epidemiologic studies. The agents for which there is inadequate evidence from human studies or no data from epidemiologic studies, but sufficient

evidence exists from animal studies, would usually be classified as B2.

Group C: Possible human carcinogens are used for agents with limited evidence of carcinogenicity in animals in the absence of human data. It includes a wide variety of evidence:

- a. Definitive malignant tumor response in a single well-conducted study,
- b. Marginal tumor responses in studies having inadequate design or reporting,
- c. Benign but not malignant tumors with an agent showing no response in a variety of short-term tests for mutagenicity, and
- d. Marginal responses in a tissue known to have a high and variable background rate.

Group D: Not classified is used for agent(s) with inadequate human or animal evidence of carcinogenicity or for which no data are available.

Group E: No evidence of carcinogenicity for humans is used for agents that show no evidence of carcinogenicity in at least two adequate animal studies in different species or in both adequate epidemiologic and animal studies.

The text for the general weight-of-evidence discussion is taken from proposed guidelines for carcinogen risk assessment (Fed. Reg. 1986).

The EPA Carcinogen Assessment Group (CAG) has evaluated more than fifty chemicals as suspect human carcinogens and developed relative carcinogenic potency factors for each chemical. The ranking of potency indices is subjected to the uncertainty of comparing different routes of exposure and a number of different species. These indices are based on estimates of low dose risk using linear multistage extrapolation from the observed range. Thus, these indices are not valid when compared to potencies in the experimental or observational range, especially if linearity does not exist in this range.

3. Inadequate evidence, which applies to both positive and negative evidence, indicates that one of two conditions prevailed: a) there were few pertinent data, b) the available studies, while showing evidence of association, did not exclude change, bias, or confounding.
4. No evidence, which applies when several adequate studies were available which do not show evidence of carcinogenicity.

The assessment of evidence of carcinogenicity from studies in experimental animals by IARC are defined as:

1. Sufficient evidence of carcinogenicity, which indicates that there is an increased incidence of malignant tumors: a) in multiple species or strains, b) in multiple experiments, or c) to an unusual degree with regard to incidence, site, type of tumor, or age of onset. Chemicals for which there is sufficient evidence of carcinogenicity in humans are judged by IARC to present a carcinogenic risk to humans.
2. Limited evidence of carcinogenicity, which means that the data suggest a carcinogenic effect but are limited because of some type of inadequacy in experimental design.
3. Inadequate evidence, which indicates that because of major qualitative or quantitative limitations, the study cannot be interpreted as showing either the presence or absence of a carcinogenic effect.
4. No evidence of carcinogenicity applies when several adequate studies show that the chemical does not induce cancer.

The new IARC categories are listed below:

<u>IARC Category</u>		<u>IARC Titles</u>
1	Sufficient evidence from epidemiological studies	Known human carcinogen
2A	Sufficient animal evidence Evidence of human carcinogenicity is at least limited evidence from epidemiological studies	Probable human carcinogen
2B	Sufficient animal evidence and inadequate evidence from human studies	Possible human carcinogen

APPENDIX B  
WORKSHEETS FOR INDICATOR COMPOUND  
SELECTION PROCESS

AR301104



WORKSHEET 1.  
 SCORING FOR INDICATOR COMPOUND SELECTION:  
 CONCENTRATIONS IN VARIOUS ENVIRONMENTAL MEDIA.

NAME OF SITE: New Castle Spill Site  
 DATE PREPARED: 16-Nov-88  
 ANALYST: TAS

CHEMICAL	GROUND WATER (mg/L)			SURFACE WATER (mg/L)			SUBSURFACE SOIL (mg/kg)		
	MIN	MAX	AVE	MIN	MAX	AVE	MIN	MAX	AVE
trans-1,2-Dichloroethene	ND	0.011	0.001						
Trichloroethene	ND	0.120	0.018						
1,2-Dichlorobenzene	ND	0.002	0.000						
Ethylbenzene							ND	0.011	0.001
Benz(a)anthracene							ND	1.300	0.112
Benzo(b)fluoranthene							ND	2.500	0.220
Benzo(a)pyrene							ND	1.200	0.092
Indeno(1,2,3-cd)pyrene							ND	0.100	0.014
Chrysene							ND	1.800	0.158
Carbon Disulfide	ND	0.015	0.001				ND	0.006	0.001
bis(2-Ethylhexyl)phthalate							ND	0.038	0.004
2-Butanone							ND	0.047	0.005
Iron - u	ND	19.400	12.600						
Manganese - u	ND	5.240	1.494						
Acenaphthylene - u							ND	0.580	0.073
Isophorone - u							ND	0.079	0.010
Fluoranthene - u							ND	3.600	0.312
Anthracene - u							ND	1.100	0.077
Pyrene - u							ND	2.400	0.210
tris (2-Chloropropyl)phosphate - u	ND	110.000	9.000	ND	0.042	0.011	ND	11.740	1.640
Dibenzofuran - u							ND	0.470	0.059
Naphthalene - u	ND	0.008	0.002				ND	0.390	0.049
2-Methylnaphthalene - u							ND	0.042	0.005
Phenanthrene - u	ND	0.005	0.001				ND	4.400	0.348
Fluorene - u							ND	0.580	0.002

u - Toxicity constants not specified in Appendix C of Superfund Public Health Evaluation Manual (US EPA, 1986a)

ND - Not Detected

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WORKSHEET 2.  
 SCORING FOR INDICATOR COMPOUND SELECTION:  
 TOXICITY INFORMATION.

NAME OF SITE: New Castle Spill Site  
 DATE PREPARED: 16-Nov-88  
 ANALYST: TAS

CHEMICAL	TOXICOLOGIC CLASS	EPA RATING VALUE		WATER	SOIL	AIR
		ORAL	INHALATION	T.C.	T.C.	T.C.
trans-1,2-Dichloroethene	NC	5	5	5.29E-02	2.65E-06	5.29E-01
Trichloroethene	PC	B2	B2	4.29E-03	2.14E-07	4.29E-02
Trichloroethene	NC	5	4	1.05E+00	5.26E-05	2.96E+01
1,2-Dichlorobenzene	NC	4	5	5.19E-02	2.60E-06	3.61E-01
Ethylbenzene	NC	4	4	1.10E-02	5.52E-07	1.10E-01
Benzo(a)anthracene	PC	B2	B2	5.81E-01	2.91E-05	5.81E+00
Benzo(b)fluoranthene	PC	B2	B2	1.43E+01	7.00E-04	1.43E+02
Benzo(a)pyrene	PC	B2	B2	4.55E+00	2.28E-04	4.55E+01
Benzo(a)pyrene	NC	8	6	2.67E+01	1.33E-03	1.91E+01
Indeno(1,2,3-cd)pyrene	PC	C	C	1.42E+01	7.00E-04	1.43E+02
Chrysene	PC	B2	B2			
Carbon Disulfide	NC	7	7	4.24E-01	2.12E-05	4.24E+00
bis(2-Ethylhexyl)phthalate	PC	B2	B2	5.71E-04	2.66E-08	5.71E-03
2-Butanone	NC	10	10	7.75E-03	3.87E-07	7.75E-02

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WORKSHEET 3  
 SCORING FOR INDICATOR COMPOUND SELECTION:  
 CALCULATION OF CT AND IS VALUES FOR CARCINOGENIC EFFECTS.

NAME OF SITE: New Castle Spill Site  
 DATE PREPARED: 18-Nov-88  
 ANALYST: TAS

CHEMICAL	GROUND WATER CT		SURFACE WATER CT		SUBSURFACE SOIL CT		IS VALUE		TENATIVE RANK	
	MAX	ME	MAX	ME	MAX	ME	MAX	ME	MAX	ME
Benzo(b)fluoranthene					1.75E-03	1.54E-04	1.75E-03	1.54E-04	1	1
Trichloroethene	5.15E-04	7.72E-05					5.15E-04	7.72E-05	2	2
Benzo(a)pyrene					2.74E-04	2.10E-05	2.74E-04	2.10E-05	3	3
Indeno(1,2,3-cd)pyrene					7.00E-05	9.80E-06	7.00E-05	9.80E-06	4	4
Benzo(a)anthracene					3.78E-05	3.28E-06	3.78E-05	3.28E-06	5	5
bis(2-Ethylhexyl)phthalate					1.09E-09	1.26E-10	1.09E-09	1.26E-10	6	6
Chrysene									7	7

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WORKSHEET 4  
 SCORING FOR INDICATOR COMPOUND SELECTION:  
 CALCULATION OF CT AND IS VALUES FOR NONCARCINOGENIC EFFECTS.

NAME OF SITE: New Castle Spill Site  
 DATE PREPARED: 16-Nov-98  
 ANALYST: TAS

CHEMICAL	GROUND WATER CT		SURFACE WATER CT		SUBSURFACE SOIL CT		IS VALUE		TENATIVE RANK	
	MAX	ME	MAX	ME	MAX	ME	MAX	ME	MAX	ME
Trichloroethene	1.26E-01	1.89E-02					1.26E-01	1.89E-02	1	1
Carbon Disulfide	6.36E-03	4.24E-04			1.27E-07	2.12E-08	6.36E-03	4.24E-04	2	2
Benzo(a)pyrene					1.60E-03	1.22E-04	1.60E-03	1.22E-04	3	3
trans-1,2-Dichloroethene	5.82E-04	5.29E-05					5.82E-04	5.29E-05	4	4
1,2-Dichlorobenzene	1.04E-04	1.56E-05					1.04E-04	1.56E-05	5	5
2-Butanone					1.82E-08	1.94E-09	1.82E-08	1.94E-09	6	6
Ethylbenzene					6.07E-09	5.52E-10	6.07E-09	5.52E-10	7	7

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WORKSHEET 5.  
 SCORING FOR INDICATOR CHEMICAL SELECTION:  
 EVALUATION OF EXPOSURE FACTORS AND FINAL CHEMICAL SELECTION.

NAME OF SITE: New Castle Spill Site  
 DATE PREPARED: 18-Nov-88  
 ANALYST: TAS

CHEMICAL	IS VALUE		RANKING		WATER SOLUBILITY	VAPOR PRESSURE	HENRY'S LAW CONSTANT	Koc	HALF-LIFE (DAYS)				IC	
	AVE PC	AVE NC	PC	NC	(mg/L)	(mm HG)	(atm-m <sup>3</sup> /mole)		SOIL	AIR	SW	GW		
trans-1,2-Dichloroethene		5.29E-05		4	6.30E+04	2.65E+02	6.56E-03	59		2.10	-	1.00	6.00	yes
Trichloroethene	7.72E-05	1.89E-02	2	1	1.10E+03	5.79E+01	9.10E-03	128		3.70	-	1.00	90.00	yes
1,2-Dichlorobenzene		1.56E-05		5	1.00E+02	1.00E+00	1.93E-03	1700		26.00	-	1.50	8.50	
Ethylbenzene		5.52E-10		7	1.52E+02	7.00E+00	6.43E-03	1100		1.46	-	1.50	7.50	
Benz(a)anthracene	3.26E-06			5	5.70E-03	2.20E-06	1.16E-06	1380000		5.50	-	1.00	5.00	
Benzo(b)fluoranthene	1.54E-04			1	1.40E-02	5.00E-07	1.19E-05	550000		5.50	-	1.00	2.00	
Benzo(a)pyrene	2.10E-05	1.22E-04	3	3	1.20E-03	5.60E-06	1.55E-06	5500000	420.00	480.00	1.00	6.00	0.40	
Indeno(1,2,3-cd)pyrene	9.80E-08			4	5.30E-04	1.00E-10	6.86E-06	1800000		5.50	-	0.0208	2.08	
Chrysene	0.00E+00			7	1.80E-03	6.30E-06	1.05E-06	200000		5.50	-	0.20	-	
Carbon Disulfide		4.24E-04		2	2.94E+03	3.60E+02	1.23E-02	54						
bis(2-Ethylhexyl)phthalate	1.26E-10	1.94E-09	6	6										
2-Butanone														
tris(2-Chloropropyl)phosphate														yes

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APPENDIX C  
FATE AND TRANSPORT PROFILES OF  
THE INDICATOR COMPOUNDS

## Trans-1,2-DICHLOROETHENE

### General:

trans-1,2-Dichloroethene is a colorless liquid with an ether-like odor. It has many uses as a solvent, in rubber manufacturing, as a refrigerant, and as an additive to dye and lacquer solutions. It is also used as a constituent of perfumes and thermoplastics. trans-1,2-Dichloroethene is only slightly soluble in water. When its water solubility is exceeded, this chemical will sink in a column of water.

### Fate and Transport:

Volatilization appears to be the major transport process for trans-1,2-dichloroethene in surface water and soils. The volatilization half-life in surface water is reported to be 22 minutes. Once in the troposphere, the chemical is attacked at the double bond by hydroxyl radicals to form formic acid, hydrochloric acid, and carbon monoxide. The tropospheric half-life of trans-1,2-dichloroethene, based on its rate of reaction with hydroxyl radicals, is probably less than one day. Based on its rate of oxidation in the troposphere, little or no trans-1,2-dichloroethene would be expected to migrate into the stratosphere; thus photolysis is probably a minor fate process in the atmosphere. Photolysis does not appear to be an important fate process in the terrestrial or aquatic environments. Oxidation and hydrolysis in the aquatic environment do not appear to be significant. Based on its  $K_{OC}$ , trans-1,2-dichloroethene probably does not adsorb to soils and sediments to any extent. Based on its octanol/water partition coefficient ( $K_{OW}$ ), this compound probably does not bioaccumulate. trans-1,2-Dichloroethene does biodegrade in the environment, especially under anaerobic conditions. However, rates of degradation are probably slow; therefore, biodegradation is probably not an important fate process.

### Summary:

The major environmental fate and transport process for trans-1,2-dichloroethene is volatilization from surface water and soils to the troposphere with subsequent attack by hydroxyl radicals. In groundwater and subsurface soils, trans-1,2-dichloroethene will infiltrate and migrate with the ground water flow.

### References:

Callahan, M.A., et al., 1979; Verschueren, K., 1983; Vogel, T.M., et al., 1987.

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## TRICHLOROETHENE

### General:

Trichloroethene (TCE) is ubiquitous in the environment, although it is not naturally occurring. Widely used as a solvent in industrial degreasing of metals, TCE has minor uses in fumigant mixtures, inhalation anesthesia, and decaffeination of coffee. TCE is a highly volatile unsaturated aliphatic hydrocarbon with a relatively high water solubility. From its density, any TCE in excess of its water solubility would sink to the bottom of the water.

### Fate and Transport:

Volatilization of TCE in the environment is its most important fate process. Its laboratory half-life is reported to be 21 minutes. Once the compound enters the troposphere, high temperatures and UV radiation promote rapid degradation ( $t_{1/2} = 4$  days) to hydrochloric acid (HCl), dichloroacetyl chloride, phosgene, carbon monoxide, and hexachlorobutadiene. The overall half-life of TCE in surface water and air is 1-90 days and 4 days, respectively. Limited laboratory studies on the adsorption of TCE onto soils and sediments indicate that TCE does not adsorb to a great extent to pure clays (<5 percent adsorption). Thus, adsorption will not be considered as a major fate process. TCE does not significantly bioaccumulate in the environment as seen by bioconcentration factors of  $10^{-17}$  for bluegills, with a half-life in tissue of less than 1 day. Higher mammals, including man, can degrade TCE to chlorinated acetic acids. Under anaerobic conditions, TCE can degrade to carbon dioxide in subsurface environments. However, biodegradation/biotransformation is considered of minor significance as an environmental fate process.

### Summary:

The major environmental transport process for TCE is volatilization from surface water and soils to the atmosphere. In ground water and subsurface soils, TCE will infiltrate and migrate with the ground water flow.

### References:

Callahan, M.A. et al., 1979; Mills, W.B. et al., 1982, U.S. EPA, 1985 ; Schuller, T.A., 1983; Wilson and Wilson, 1985.

## TRIS(2-CHLOROPROPYL)PHOSPHATE

### General:

Tris(2-chloropropyl)phosphate has many synonyms of which the most common is tris. Tris is a clear, colorless liquid. It is most commonly used as an industrial flame retardant for textiles. Generally, tris and its related compounds are not very water soluble. When its water solubility is exceeded, excess tris will sink.

### Fate and Transport:

Little information on the environmental fate and transport of tris is available. The behavior of tris in the environment is inferred from information available for the structurally related compounds tris(2,3-dibromopropyl)phosphate and tris(2-chloroisopropyl)phosphate. Based on its vapor pressure, tris will volatilize from surface waters and surface soils. Tris has a low reactivity with water and bases; thus, hydrolysis is not considered an important environmental fate process. One study indicates that tris does not bioaccumulate in organisms. Tris has a reported bioconcentration factor of about 3. Little is known about the sorptive behavior of tris. However, because of its low water solubility and its large Kow, tris will probably sorb in significant amounts to clay and other soil material. There is little or no information in the literature regarding oxidation, photolysis, or biodegradation of tris in the environment. Thus, no conclusions as to the importance of these processes can be made at this time.

### Summary:

The major environmental fate process for tris is sorption in soils, with volatilization from surface soils and surface waters as a minor fate process.

### References:

Isnard, P. and S. Lambert, 1988; Grayson, M., 1985.

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APPENDIX D  
SAMPLE CALCULATIONS

**TABLE D-1  
STANDARD INTAKE EQUATIONS  
EXAMPLE CALCULATIONS FOR EXPOSURE  
OF CHILDREN 6-12 TO tri(2-CHLOROPROPYL)PHOSPHATE**

Dermal Contact

1. Exposure to surface water

Present,  
existing

Subchronic Intake

$$= [\text{max}] \cdot \% \text{SAW} \cdot \text{SS} \cdot \text{MF} \cdot \text{DSW} \cdot (1/\text{BW}) \cdot (1 \text{ L}/1000000 \text{ mg})$$

$$= \{0.042 \text{ mg/L} \cdot 0.20 \cdot (10470 \text{ sq cm}) \cdot (0.5 \text{ mg/sq cm/hr})$$

$$\cdot 1 \text{ event} \cdot (1/29 \text{ kg}) \cdot (1 \text{ L}/1000000 \text{ mg})\}$$

$$= 1.52\text{E-}06 \text{ mg/kg/day}$$

Chronic intake

$$= [\text{ave}] \cdot \% \text{SAW} \cdot \text{SS} \cdot \text{MF} \cdot \text{SW} \cdot \text{DSW} \cdot (1/\text{BW}) \cdot (1 \text{ L}/1000000 \text{ mg})$$

$$= \{0.011 \text{ mg/L} \cdot 0.20 \cdot (10470 \text{ sq cm}) \cdot (0.5 \text{ mg/sq cm/hr})$$

$$\cdot 1 \text{ hr} \cdot (150 \text{ days}/365 \text{ days}) \cdot (1/29 \text{ kg}) \cdot (1 \text{ L}/1000000 \text{ mg})\}$$

$$= 1.63\text{E-}07 \text{ mg/kg/day}$$

2. Exposure to soils

Present,  
existing

Subchronic Intake

$$= [\text{max}] \cdot (1/\text{BW}) \cdot (1 \text{ kg}/1000000 \text{ mg}) \cdot \text{DSC} \cdot \{(\% \text{SAS} \cdot \text{SS} \cdot \text{DA}$$

$$\cdot \text{SAR} \cdot \text{ME}) + 1\}$$

$$= 11.7 \text{ mg/kg} \cdot (1/29 \text{ kg}) \cdot (1 \text{ kg}/1000000 \text{ mg}) \cdot 3 \text{ hrs} \cdot \{(0.20 \cdot$$

$$10470 \text{ sq cm} \cdot (0.51 \text{ mg/sq cm}) \cdot 0.12 \cdot 0.15\} + 50 \text{ mg}\}$$

$$= 2.81\text{E-}05 \text{ mg/kg/day}$$

Chronic intake

$$= [\text{ave}] \cdot (1/\text{BW}) \cdot (1 \text{ kg}/1000000 \text{ mg}) \cdot \text{SW} \cdot \text{DSC} \cdot \{(\% \text{SAS} \cdot \text{SS}$$

$$\cdot \text{DA} \cdot \text{SAR} \cdot \text{ME}) + 1\}$$

$$= 1.64 \text{ mg/kg} \cdot (1/29 \text{ kg}) \cdot (1 \text{ kg}/1000000 \text{ mg}) \cdot 1 \text{ hr/day} \cdot$$

$$(150 \text{ day}/365 \text{ days}) \cdot \{(0.20 \cdot 10470 \text{ sq cm} \cdot (0.51 \text{ mg/sq cm})$$

$$\cdot 0.12 \cdot 0.15\} + 50 \text{ mg}\}$$

$$= 1.61\text{E-}06 \text{ mg/kg/day}$$

3. Exposure to ground water while bathing

HYPOTHETICAL

Future use

Subchronic Intake

$$= [\text{max}] \cdot \% \text{SB} \cdot \text{SS} \cdot \text{MF} \cdot (1/\text{BW}) \cdot \text{TB} \cdot (1 \text{ L}/1000000 \text{ mg})$$

$$= 110 \text{ mg/L} \cdot 0.80 \cdot 10470 \text{ sq cm} \cdot (0.5 \text{ mg/sq cm/hr}) \cdot$$

$$(1/29 \text{ kg}) \cdot 0.33 \text{ hr} \cdot (1 \text{ L}/1000000 \text{ mg})$$

$$= 5.23\text{E-}03 \text{ mg/kg/day}$$

Chronic intake

$$= [\text{ave}] \cdot \% \text{SB} \cdot \text{SS} \cdot \text{MF} \cdot (1/\text{BW}) \cdot \text{TB} \cdot (1 \text{ L}/1000000 \text{ mg})$$

$$= 9.0 \text{ mg/L} \cdot 0.80 \cdot 10470 \text{ sq cm} \cdot (0.5 \text{ mg/sq cm/hr}) \cdot$$

$$(1/29 \text{ kg}) \cdot 0.33 \text{ hr} \cdot (1 \text{ L}/1000000 \text{ mg})$$

$$= 4.29\text{E-}04 \text{ mg/kg/day}$$

Inhalation

Inhalation while bathing

HYPOTHETICAL

Future use

Subchronic Intake

$$= [\text{max}] \cdot \{((\text{VW} \cdot \text{IR} \cdot \text{TB}) / (2 \cdot \text{VS})) + ((\text{IR} \cdot \text{AE} \cdot \text{VW}) / (\text{VB}))\} \cdot (1/\text{BW}) \cdot \% \text{INH}$$

$$= 110 \text{ mg/L} \cdot \{((200 \text{ L} \cdot 0.46 \text{ c m/hr} \cdot 0.33 \text{ hr}) / (2 \cdot 3 \text{ c m})) +$$

$$((0.46 \text{ c m/hr}) \cdot 0.166 \text{ hr} \cdot 200 \text{ L}) / (10 \text{ c m})\} \cdot 1/29 \text{ kg} \cdot 0.5$$

$$= 6.27\text{E+}00 \text{ mg/kg/day}$$

Chronic intake

$$= [\text{ave}] \cdot \{((\text{VW} \cdot \text{IR} \cdot \text{TB}) / (2 \cdot \text{VS})) + ((\text{IR} \cdot \text{AE} \cdot \text{VW}) / (\text{VB}))\} \cdot (1/\text{BW}) \cdot \% \text{INH}$$

$$= 9 \text{ mg/L} \cdot \{((200 \text{ L} \cdot 0.46 \text{ c m/hr} \cdot 0.33 \text{ hr}) / (2 \cdot 3 \text{ c m})) +$$

$$((0.46 \text{ c m/hr}) \cdot 0.166 \text{ hr} \cdot 200 \text{ L}) / (10 \text{ c m})\} \cdot 1/29 \text{ kg} \cdot 0.5$$

$$= 1.03\text{E+}00 \text{ mg/kg/day}$$

Ingestion

Drinking water exposure

HYPOTHETICAL

Future use

Subchronic Intake

$$= [\text{max}] \cdot \text{W} \cdot (1/\text{BW}) \cdot \% \text{ING}$$

$$= 110 \text{ mg/L} \cdot 1 \text{ L} \cdot (1/29 \text{ kg}) \cdot 1$$

$$= 3.80\text{E+}00 \text{ mg/kg/day}$$

Chronic intake

$$= [\text{ave}] \cdot \text{W} \cdot (1/\text{BW}) \cdot \% \text{ING}$$

$$= 9 \text{ mg/L} \cdot 1 \text{ L} \cdot (1/29 \text{ kg}) \cdot 1$$

$$= 3.11\text{E-}01 \text{ mg/kg/day}$$

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TABLE D-2  
STANDARD PARAMETERS FOR CALCULATION OF DOSAGE AND INTAKE  
NEW CASTLE SPILL SITE

	Symbol	Adult	Child Age 6-12	Child Age 2-6
<b>PHYSICAL CHARACTERISTICS</b>				
Average Body Weight	(a) BW	70 kg	29 kg	16 kg
Average Skin Surface Area	(a,e) SS	18,150 cm <sup>2</sup>	10,470 cm <sup>2</sup>	6980 cm <sup>2</sup>
<b>ACTIVITY CHARACTERISTICS</b>				
Amount of Water Ingested Daily	(a) W	2 liters	1 liter	1 liter
Amount of Air Breathed Daily	(d) A	20 m <sup>3</sup>	11 m <sup>3</sup>	6 m <sup>3</sup>
Duration of Soil Contact				
Subchronic	(d) DSC	3 hr	3 hr	3 hr
Chronic		1 hr	1 hr	1 hr
Frequency of Soil Contact	(d) FS	5 days/year	150 days/year	150 days/year
Percentage of Skin Surface Area Contacted by Soils	(d) %AS	20%	20%	20%
Skin Absorption Rate of Compounds in Soil	(c) SAR	6%	12%	12%
Incidental Soil Ingestion (per event)	(c) I I	50 mg	50 mg	100 mg
Frequency of Surface Water Contact Casual				
Duration of Surface Water Contact (Casual)	(d) DSW	1 event	1 event	1 event
Percentage of Skin Surface Area Immersed	(a) %AW	20%	20%	20%
Percentage of Surface Area Immersed While Bathing	(a) %SB	80%	80%	80%
Length of Time While Bathing	(b) TB	20 min.	20 min.	20 min.
Amount of Air Breathed While Bathing	(a) I R	0.83 cu m/hr	0.46 cu m/hr	0.25 cu m/hr
Length of Additional Exposure After Bathing	(b) AE	10 min.	10 min.	10 min.
Volume of Showerstall	(b) VS	3 cu m	3 cu m	3 cu m
Volume of Bathroom	(b) VB	10 cu m	10 cu m	10 cu m
Volume of Water Used While Showering	(b) VW	200 L	200 L	200 L
Absorption via Inhalation (%)	%INH	50	50	50
Absorption via Ingestion (%)	%ING	100	100	100
<b>MATERIAL CHARACTERISTICS</b>				
Dust Adherence (Potting Soil)	(a) DA	0.51 mg/cm <sup>2</sup>	0.51 mg/cm <sup>2</sup>	0.51 mg/cm <sup>2</sup>
Soil Matrix Effect	(c) ME	15%	15%	15%
Mass Flux Rate (water-based)	(a) MF	0.5 mg/cm <sup>2</sup> /hr	0.5 mg/cm <sup>2</sup> /hr	0.5 mg/cm <sup>2</sup> /hr

a - Superfund Exposure Assessment Manual, 1988

b - K.G. Symms, "An Approximation of the Inhalation Exposure to Volatile Synthetic Organic Chemicals from Showering with Contaminated Household Water," Paper to be presented at the Symposium of American College of Toxicologists, 11/15/86

c - J.K. Hawley, "Assessment of Health Risk from Exposure to Contaminated Soil," Risk Analysis, Vol. 5, No. 4, 1985

d - ERM Staff Professional Judgement

e - Anderson et al, 1984

APPENDIX E  
CALCULATION OF AN ACCEPTABLE DAILY INTAKE  
FOR tris(2-CHLOROPROPYL)PHOSPHATE

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## APPENDIX E

The Acceptable Daily intake for Chronic exposure (AIC) or reference dose (RfD) of 0.125 mg/kg/day for tris(2-chloropropyl)phosphate was calculated from a rat subchronic study described in the Product Safety Information Sheet supplied by Stauffer Chemical Company. The RfD for humans was calculated to be equal to the No-Observable-Adverse-Effect Level (NOAEL) for an experimental animal divided by appropriate safety factors. In a toxicology study, the NOAEL is the dose at which the animal does not receive any toxic effects from the chemical in question. The safety factors usually consist of multiples of ten which are applied to account for the uncertainty in extrapolation of animal data to the human and another factor of ten may be applied to account for the differences in sensitivity between individuals in the human population. The safety factor of 100 that results is judged by US EPA to be appropriate for many chemicals. Additional safety factors, usually in multiples of ten, may be applied in situations where the database for a chemical is incomplete. One example of this is when the NOAEL value which is used to calculate the RfD is based on a study shorter than a chronic or lifetime study. This procedure for calculating an RfD is summarized in the background documents which supports the Integrated Risk Information System (IRIS). The results of the rat subchronic study for tris(2-chloropropyl)phosphate are summarized as follows.

A three month toxicology study was performed on male and female rats receiving daily dietary concentrations of tris at 800, 2,500, 7,500 and 20,000 ppm. An increase in relative and absolute liver weight is observed in male rats at all dose levels and in female rats receiving 7,500 or 20,000 ppm tris in the diet. This type of change is not judged to be an adverse or toxic effect of tris exposure. Toxic effects were observed in the female rats receiving the highest dose and in male rats at both the 7,500 and 20,000 ppm dose levels. These effects included mild cortical tubular degenerative changes in the kidney and mild histopathological changes in the liver (only in animals receiving 20,000 ppm). Very mild hypoplasia of the sternal bone marrow and very mild thyroid follicular hyperplasia was observed in the female rats receiving the highest dose of tris.

In this study the NOAEL is the dietary concentration of 2,500 ppm because the toxic effects observed in this study only occur at higher doses. To use this NOAEL in calculating the RfD for humans, it must first be converted from a dietary concentration in ppm to one expressed in milligram per kilogram of body weight. The dietary concentration is multiplied by a factor of 0.05, which is the fraction of body weight that a rat eats daily. The actual dose of 125 mg/kg/day is calculated in this manner. This actual daily dose is then divided by the safety factors to determine an acceptable long-term daily intake for humans. A safety factor of 1,000 incorporates a factor of 10 for extrapolation of animal data to humans; a factor of 10 to protect sensitive human subpopulations; and a factor of 10 for substituting a subchronic study for a chronic one. The resultant RfD (for oral exposure) is 0.125 mg/kg/day.

C

In this case, an Acceptable Intake for a Subchronic (or short term) exposure (AIS) can be easily calculated from the RFD by removing the safety factor of 10 which accounts for the uncertainty in substituting a subchronic study for a chronic one. The calculation of the AIS will be equal to the dose of 125 mg/kg/day divided by a safety factor of 100, resulting in an AIS of 1.25 mg/kg/day.

The RfD and AIS calculations are as follows:

NOAEL for rats = 2500 ppm in the diet (Product Safety Data Sheet  
- Stauffer Chemical)

DOSE = NOAEL \* Daily Intake for Rat (expressed as % of body  
= 2,500 ppm \* 0.05 weight)  
= 125 mg/kg/day

RfD (chronic oral) = Dose/Safety Factors  
= (125 mg/kg/day)/1000  
= 0.125 mg/kg/day

AIS (subchronic oral) = Dose/Safety Factors  
= (125 mg/kg/day)/100  
= 1.25 mg/kg/day

APPENDIX F  
TOXICOLOGICAL PROFILES OF THE  
INDICATOR COMPOUNDS

AR301121



## APPENDIX F

### TRICHLOROETHENE

#### Synonyms

1,1,2-trichloroethene, acetylene trichloride, ethinyl trichloride, ethylene trichloride, TCE, TRI, trichloroethylene

#### References

International Programme on Chemical Safety, Environmental Health Criteria No. 50, Trichloroethene, WHO, Geneva 1985.

U.S. Environmental Protection Agency, Health Assessment Document for Trichloroethene, July 1985, EPA/6008-82/006F.

World Health Organization, International Agency for Research on Cancer, IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Suppl.4, Lyon, October 1982.

#### Summary of Health Effects Data

The acute toxicity of trichloroethene is relatively low, mainly central nervous system depression at high concentration levels. In experimental animals, kidney and liver toxicity may be induced by chronic exposure at elevated doses. There is evidence that trichloroethene is carcinogenic in rodents at high concentrations, but the significance of these findings with respect to low-level human exposure is controversial. Extensive

epidemiological investigations have failed to substantiate an increased carcinogenic risk for humans.

### Toxicokinetics

Trichloroethene can be absorbed by dermal or oral contact, or by inhalation. Absorption by the dermal route is normally not high enough to elicit toxic effects. Pulmonary uptake of the substance is rapid, and distribution occurs to all body tissues with a considerable fraction in adipose (fatty) tissue. It readily crosses the placental barrier. In humans part of the absorbed trichloroethene (about 10%) is expired unchanged in exhaled air. Metabolic conversion in the liver results in urinary excretion of 30-50% as trichloroethanol (partly as a glucuronide) and 10-30% as trichloroacetic acid. Estimation of these metabolites in urine may be utilized for the biological monitoring of exposure. After a single exposure, the level of trichloroacetic acid in blood and urine increases for up to 20-40 hrs, whereupon the concentration decreases with a half-life of 70-100 hrs. Although elimination from the tissues occurs at a slow rate, virtually all the trichloroethene from a single high dose is excreted within 48 hours of administration.

### Toxicology Studies

Non-neoplastic effects Trichloroethene has a low acute oral toxicity in mammals with LC<sub>50</sub> values in the range 5000-15,000 ppm. In humans, higher concentrations of this volatile solvent have anesthetic as well as analgesic properties and may occasionally elicit cardiac arrhythmias. Chronic exposure to high levels has been reported to induce neurotoxic symptoms like ataxia, sleep disturbances and psychotic episodes as well as neuropathy of the

cranial nerves. Humans exposed to extremely high concentrations of trichloroethene have experienced liver and kidney damage similar to the effects noted in animal studies. The induction of irreversible neuropathies may involve decomposition products of trichloroethene, like highly toxic dichloroacetylene. This idea is supported by the finding that such effects have not been found consistently in epidemiological studies involving high exposure levels.

Carcinogenicity Studies There is evidence that trichloroethene, with and without epoxide stabilizers induces liver tumors in mice upon inhalation or oral administration of high doses. There is limited evidence that this solvent also induces renal tumors associated with toxic nephrosis in male rats, but this assay (NTP) has been considered inadequate to evaluate the carcinogenic response.

The hepatocarcinogenic action of trichloroethene in mice has been associated with peroxisome induction (caused by the metabolite trichloroacetic acid). Opinions differ as to the significance of these findings with respect to its relevance to man. Further, the suitability of the linearized multistage model used by the U.S. EPA for low-dose extrapolation with respect to this type of rodent carcinogen has been questioned.

A number of epidemiological investigations including occupationally exposed population groups have been carried out to examine the possible carcinogenic action of trichloroethene, but so far no adequate support for a carcinogenic action in humans has been obtained. These studies tend to support the view that the carcinogenic potency factor derived by EPA, which is of the same order of magnitude as for the well established human

carcinogens benzene and vinyl chloride, represents a significant overestimation of risk. It appears extremely unlikely that if trichloroethene is a potent human carcinogen it would have escaped detection in the epidemiological surveys already conducted.

Mutagenic Effects and Adverse Effects on Reproduction Due to the presence of mutagenic impurities and other factors present in trichloroethene, the results from short-term mutagenicity testing have been ambiguous. The mutagenic activity of trichloroethene must be regarded as low or non-existent.

Trichloroethene does not seem to induce any biologically significant embryotoxic or teratogenic effects in experimental animals.

#### Regulatory Standards

On the basis of the long-term studies in rodents, EPA has classified trichloroethene as a Group B2 carcinogen (probable human carcinogen) with a carcinogenic potency factor (oral) of  $0.011 \text{ (mg/kg/day)}^{-1}$ . IARC considers that only limited evidence is available that trichloroethene is carcinogenic in mice and has classified the substance in Group 3 (non-classifiable as to its carcinogenicity for humans). The position of the IPCS International Task Group concerning the induction of tumors in rodents was that "the significance of these findings needs to be evaluated in the context of further studies on the mechanism of action of trichloroethene." In the European Common Market this solvent is classified as "Harmful" (X).

According to EPA, a mutagenic potential cannot be ruled out, but EPA takes the position that if the compound is mutagenic, the available data suggest that the substance would be a very weak, indirect mutagen. IARC has judged available evidence to be inadequate to assess the mutagenicity of trichloroethene.

The current ACGIH 8 hrs TWA (TLV) for trichloroethene is 50 ppm (270 mg/m<sup>3</sup>). The ambient water quality criterion for the protection of aquatic life in fresh water has been set at 45 mg/L. The Maximum Contaminant Level in drinking water has been adjusted to 0.005 mg/L.

A summary of critical toxicity values and regulatory standards for trichloroethene are summarized in the attached table.

#### Synonyms

Trans-1,2-dichloroethylene, trans-acetylene dichloride, Dioform.

#### Sources

The major use of trans-1,2-dichloroethene is as a captive intermediate in the production of other chlorinated solvents. trans-1,2-Dichloroform is also formed by the degradation of trichloroethylene and tetrachloroethylene in ground water.

#### References

U.S. EPA, 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites, Final. Prepared by Clement Associates.

U.S. EPA, 1985. Health Advisories for 52 Chemicals Which Have Been Detected in Drinking Water. PB86-1118338.

U.S. EPA, 1984. Health Effects Assessment for trans-1,2-Dichloroethene.

#### Summary of Health Effects Data

There have not been many studies on the toxic effects of trans-1,2-dichloroethene. Acute exposure can product a narcotic effect and possibly liver and kidney damage. Studies indicate that trans-1,2-dichloroethene is not a mutagen. There is not enough evidence to evaluate its carcinogenicity or effects on reproduction and development.

#### Toxicokinetics

No studies have been conducted to determine the absorption of trans-1,2-dichloroethene into the body. Based upon its chemical properties (low molecular weight, high lipid solubility, and neutral electrical charge), it is expected to be absorbed through inhalation, ingestion and skin contact. Using studies of the absorption of trichloroethene, the U.S. EPA has estimated that nearly 100% of ingested trans-1,2-dichloroethene would be absorbed systemically and that 35-50% of inhaled trans-1,2-dichloroethene would be absorbed systemically. Once absorbed, the compound is distributed to all body tissues with the highest concentrations expected to be found in the liver and kidney. In vitro studies indicate that trans-1,2-dichloroethene is metabolized to 2,2-dichloroethanol and 2,2-dichloroacetic acid. Elimination of trans-1,2-dichloroethene from the body occurs primarily through exhaled air, but the metabolites are eliminated

through the urine. If the excretion of this compound is similar to that of 1,1-dichloroethene, then the major portion of a single dose would be eliminated within 24 to 72 hours of exposure.

## Toxicology Studies

### Acute

Acute exposure to trans-1,2-dichloroethene is associated with central nervous system (CNS) depression and narcosis. The trans-isomer is two times as potent as the cis-isomer in depressing the CNS. The oral LD<sub>50</sub> for trans-1,2-dichloroethene in rats is 1300 mg/kg. Rats exposed for four hours to concentrations of trans-1,2-dichloroethene ranging from 8,000 to 16,000 ppm in the air experienced narcosis and death. Acute exposure to lower concentrations of trans-1,2-dichloroethene is associated with liver and kidney damage.

### Subchronic

Several studies have been conducted on the long-term effects of trans-1,2-dichloroethene. Rats, rabbits, guinea pigs, and dogs exposed to either 500 or 1,000 ppm in the air for 7 hours per day, 5 days per week for 6 months showed no changes in growth, mortality, body and/or organ weight, hematology, clinical chemistry, or gross or microscopic pathology. In another study rats were exposed to air with 0, 200, 1,000, or 2,000 ppm (0 to 7940 mg/m<sup>3</sup>) for 8 hours per day, 5 days per week for 1, 2, 8, or 16 weeks. Exposure to 200 ppm caused degeneration of the liver lobule and fat accumulation in the Kupffer cells (hepatic macrophages). Severe histopathological changes in the lungs were observed in the rats exposed to 200 ppm for 8 and 16 weeks.

### Carcinogenicity and Mutagenicity Studies

No data are available on the carcinogenic potential of trans-1,2-dichloroethene. The EPA has placed the chemical in Group D, which means that it is not classifiable due to inadequate animal evidence of carcinogenicity.

Tests of the mutagenicity of trans-1,2-dichloroethene have been negative. It was not mutagenic in an Escherichia coli assay or a host-mediated assay using Salmonella tester strains in mice. It was also reported to have no genetic effects in an in vivo mutagenicity study.

No data are available on the reproductive or teratogenic effects of trans-1,2-dichloroethene.

### Regulatory Standards:

The ambient water quality criterion for the protection of fresh water life is 135 mg/L of trans-1,2-dichloroethene. A Maximum Contaminant Level Goal (MCLG) in drinking water has been proposed at 0.07 mg/L. A reference dose (RfD) of 0.01 mg/kg/day has been established based upon the US EPA's Health Advisory for lifetime exposure. A tentative acceptable intake for subchronic exposure (AIS) has been calculated by ERM at 0.272 mg/kg/day. This calculation is based on a one-day EPA Health Advisory of 2.72 mg/L for a 10 kg child. Regulations for workplace exposures have been set by OSHA and ACGIH at 200 ppm or 790 mg/m<sup>3</sup>.

A summary of critical toxicity values and regulatory standards for trans-1,2-dichloroethene are summarized in the attached table.

Synonyms

Fyrol PCF flame retardant, tri(beta-chloropropyl)phosphate, tri(1-chloromethyl ethyl)phosphate, tris(2-chloroisopropyl)-phosphate.

References

Fyrol PCF Product Safety Information Sheet, issued April 1986, Stauffer Chemical Co., Specialty Chemicals Division, Westport, CN.

Flame Retardants in Kirk-Othmer Concise Encyclopedia of Chemical Technology edited by M. Grayson, 1985, John Wiley & Sons, New York, NY.

Ulsamer, A.G., R.E. Osterberg, and J. McLaughlin, Flame Retardant Chemicals in Textiles, 1980, Clinical Toxicology, 17 (1), pp. 101-131.

Vaughan-Dellarco, V., Mutagenicity Assessment of Fyrol PCF tris(b-chloropropyl)phosphate 1983. OHEA-R-114, Office of Health and Environmental Assessment, EPA.

Sprague, G.L., L.L Sandvik, M.J. Brookins-Hendricks, and A.A. Bickford, Neurotoxicity of Two Organophosphorus Ester Flame Redardants in Hens, 1981, Journal of Toxicology and Environmental Health, 8, pp. 507-518.

#### Summary of Health Effects Data

The primary routes of human exposure to tris(2-chloropropyl) phosphate are skin contact and inhalation. This chemical has a very low acute and subchronic toxicity. It is non-mutagenic. To date no chronic toxicology studies have been performed.

#### Toxicokinetics

Tris(2-Chloropropyl)phosphate can enter the body through skin contact, inhalation and ingestion. No information is available on the pharmacokinetics of this compound.

#### Toxicology Studies

##### Acute

The acute oral LD<sub>50</sub> for female and male rats is 2800 mg/kg and 4200 mg/kg, respectively. No observable abnormalities were noted at necropsy. Female rats receiving single oral doses higher than 794 mg/kg experienced convulsions, hyperactivity, decreased physical activity, salivation, stained fur, tremors and bloated stomachs. Male rats receiving single oral doses higher than 2,000 mg/kg experienced symptoms of acute toxicity similar to those of the female rats.

Rats inhaling 4.6 mg/m<sup>3</sup> of tris(2-chloropropyl)phosphate (greater than 90% respirable) for a single 4-hour period experienced mild lethargy, matted fur, and less than 10% decrease in body weight. A single dermal application of 5,000 mg/kg produced only mild skin irritation and mild diarrhea in rabbits.

### Subchronic

A three months study was performed on male and female rats receiving daily dietary concentrations of tris at 800, 2,500, 7,500, and 20,000 ppm. An increase in relative and absolute liver weight is observed in male rat at all dose levels and in female rats receiving 7,500 or 20,000 ppm doses. This type of change is not judged to be an adverse or toxic effect of tris exposure. Toxic effects were observed in the female rats receiving the highest dose and in male rats at both the 7,500 and 20,000 ppm dose levels. These effects included mild cortical tubular degenerative changes in the kidney, and mild histopathological changes in the liver (only in animals receiving 20,000 ppm). Very mild hypoplasia of the sternal bone marrow and very mild thyroid follicular hyperplasia was observed in the female rats receiving the highest dose of tris. Though the LD<sub>50</sub> is higher for the male rats, they seem to be more sensitive than the females to the toxic end points examined in this study.

In this study, the NOAEL is the dietary concentration of 2,500 ppm because the toxic effects observed in this study occur only at higher doses. To use this NOAEL in calculating the RfD for humans, it must first be converted from a dietary concentration in ppm to one expressed in milligram per kilogram of body weight. The dietary concentration is multiplied by the factor 0.05, which

is the fraction of body weight that a rat eats daily. The actual dose of 125 mg/kg/day is calculated in this manner.

A neurotoxicity study performed by Sprague et al. in 1981 on 18 hens exposed to two 10 mg/kg oral doses 21 days apart determined that tris(2-chloropropyl)phosphate did not cause the acute delayed neurotoxicity associated with structurally similar compounds. Toxic effects included one mortality, severe feather loss, reduction in body weight and decreased egg production.

### Chronic

No chronic studies have been performed on tris (2-chloropropyl) phosphate.

### Mutagenicity and Carcinogenicity Studies

Extensive mutagenicity testing, sponsored by both US EPA and Stauffer Chemical Company, has been performed on tris(2-chloropropyl)phosphate. The majority of these tests were negative and indicated that tris was not behaving as a mutagenic compound. The few positive results were judged by Dr. Vaughan-Dellarco, an EPA geneticist, to be nonrepeatable and not dose responsive and may have been due to variation within test systems or chemical toxicity. The conclusion of the mutagenicity assessment conducted in 1983 by Dr. Vaughan-Dellarco was that tris(2-chloropropyl)phosphate is not mutagenic. The mutagenicity assessment included many tests, such as several short-term tests using bacteria, yeast, Drosophila, mammalian cell systems and whole rodents (rats and mice). The biological endpoints measured during the mutagenicity assessment were gene mutations, chromosomal aberrations, sister chromatid exchanges, mitotic

es, making those resources available to address releases  
rdous substances at another site or sites. This is in  
ance with EPA's Superfund enforcement strategy.

#### Precedential Value

There are no issues of precedential value in this case.

#### Value of Obtaining a Present Sum Certain

As discussed above, the proposed settlement would  
the United States with 100% of total costs through  
22, 1989 associated with the CertainTeed Pile. It is  
that the United States can obtain a substantially better  
via litigation. In addition, the settlement provides  
for cleanup to be undertaken very soon and allows funds  
could have been expended for this action to be preserved for  
atters.

#### Inequities and Aggravating Factors

None.

#### Nature of the Case Remaining

If the United States enters into the proposed  
nt, no further claims will remain (no further operable  
s planned). However, the ROD calls for a verification -  
determine the source of metal contamination in the  
uart Farm Creek. Should the study indicate that the  
the source of contamination, another ROD or an amendment  
ertainTeed Pile ROD may be required. The Consent Decree  
a waiver of the defenses of res judicata, collateral  
and claim splitting.

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