

2021 Hazardous Waste Scanning Project

File Form Naming Convention.

(File_Type).(Program).(Site_Number).(YYYY-MM-DD).(File_Name).pdf

Note 1: Each category is separated by a period "."

Note 2: Each word within category is separated by an underscore "_"

Specific File Naming Convention Label:

Report.HW.932020.1985-03-01.Rem-Alt-Eval-Vol-2.pdf

**LOVE CANAL SEWER AND CREEK
REMEDIAL ALTERNATIVES
EVALUATION AND RISK ASSESSMENT
EPA 138.2L05.0
VOLUME II**

March 1, 1985

WDR102/004

Appendix A
TECHNOLOGY SUMMARIES

DRAFT

Appendix A TECHNOLOGY SUMMARIES

A wealth of background information has been developed and an extensive amount of literature has been published in the past 25 years regarding the extremely toxic chemical 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD or dioxin). This is the result of the response by the scientific community and sponsoring agencies to the concern over dioxin. An excellent summary of much of the available literature can be found in the EPA report "Dioxins" (November 1980). The reader's attention is directed to this document and the other literature sources listed in the bibliography.

Unfortunately, only a small amount of laboratory research work to date is directly applicable to TCDD levels found in contaminated soils and sediment, i.e., little research has focused specifically on removal or destruction of TCDD bound to soil or sediment.

The following summaries briefly describe the state of development for several dioxin disposal/destruction technologies. All of these methods have been researched or considered for use on dioxin-contaminated materials. The technologies described in this appendix are listed below:

<u>Technology</u>	<u>Page</u>
Incineration	A-2
Ultraviolet Degradation	A-20
Biological Treatment	A-22
Solvent Extraction (Leaching)	A-32
Fixation of Soil	A-34
High-Temperature Fluid-Wall (HTFW) Reactors	A-40
Supercritical Water Oxidation	A-41
Microwave Plasma Destruction	A-42
Plasma Arc Pyrolysis	A-43
Gamma Ray Radiolysis	A-44
Molten Salt Combustion	A-45
Carbon Adsorption	A-47
Chemical Degradation--Chlorination	A-48
Chemical Degradation--Catalytic Oxidation	A-49
Chemical Degradation--Chloriodides	A-50
Chemical Degradation--Dechlorination	A-51
Wet Air Oxidation	A-53
Ultraviolet Ozonation	A-55

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Incineration

DESCRIPTION OF TECHNOLOGY: Soil-bound dioxin can be incinerated in two different forms: Either the raw dioxin-

contaminated soil is incinerated directly or it is first treated in a solvent extraction process and the extraction residue is incinerated. In both cases, incineration destroys the dioxin by high temperature degradation.

Incineration takes place in an environment of excess oxygen or a starved-oxygen environment (pyrolysis) at temperatures and material retention times sufficient to destroy the chlorinated hydrocarbon molecules. Currently, the minimum conditions for dioxin incineration are unknown. Since dioxin is similar to other chlorinated hydrocarbons (PCB's, for example), it will likely require incineration criteria similar to that used for PCB's, Federal regulations (40 CFR 761) call for PCB's to be incinerated at 2200°F and at 2 seconds retention time in an environment having 3 percent excess oxygen in the exhaust gases.

When dioxin-contaminated soil is to be incinerated, the process consists of two basic steps: 1) the dioxin is vaporized from the soil in a primary combustion chamber, and 2) the vapor is destroyed in a secondary combustion chamber, or afterburner. Temperatures in the primary combustion chamber must be kept below 1800°F to prevent the soil and any metals it may contain from melting and causing problems with slagging and ash handling. While temperatures above 1300°F may be sufficient to vaporize the dioxin from the soil, the vapor should be destroyed at temperatures above 2200°F. Thus, once the dioxin is vaporized, the soil is removed from the primary chamber for disposal, and the off-gases and dioxin are incinerated at higher temperatures in an afterburner.

Heterogeneous soil and rock mixtures will not be suitable for incineration in bulk form. Since the incineration process relies on heat transfer into the soil and rock particles to vaporize the dioxin, the particles must be small enough to allow adequate heat transfer into the center of the particles. Also, large rocks must be segregated or crushed to prevent physical damage to the incinerator. For these reasons, soil incineration will require a size reduction facility for proper preparation of the soil before feeding the primary combustion chamber.

Also, equipment to control air and water emissions from an incineration facility will be required. Without such equipment, particulates and some pollutant gases would be discharged from the exhaust stack of an incinerator. Gas scrubbing equipment generally requires scrubbing water, which will require clarification or other treatment before discharge to a waterway or sewage system.

Incinerating a solid-free solvent containing traces of dioxin would be a simpler process than incinerating soil. A much simpler incinerator could be used, a secondary combustion chamber may not be necessary, and scrubbing may not be required. Particle size reduction would not be required in preparation for solvent incineration, but filtering to remove soil particulate from the solvent would likely be required prior to injecting the solvent into a combustion chamber. Because the residuals from the solvent extraction process will include a large amount of inert solids in a solvent, contaminated soil residue will still have to be dealt with. Therefore, a pure solvent incineration process is not applicable to the dioxin-contaminated soil problem.

The equipment needed for these processes is further described in the following sections.

Primary Combustion Devices: Because of the difficulties with soil fusion and metal slagging, the primary combustion chamber should operate at below fusion temperatures, or at approximately 1600°F. Dioxin is believed to vaporize at 1200°F to 1300°F, so the primary combustion chamber should be capable of removing the dioxin from the soil. The amount of time that the soil would have to be held at this temperature to ensure dioxin removal to the one-ppb level is unknown. Operating variables such as this must be determined during extensive testing programs before the design process can begin.

Many types of incinerators are available for use as primary combustion devices, including both oxidizing incinerators and starved air combustion processes. However, only a limited number of these incinerators in present commercial use are capable of handling solid wastes. These include:

- o Rotary kiln
- o Fluidized bed
- o Rotary hearth
- o Multiple hearth
- o Other fixed or shaker-type hearth arrangements

A recent study summarized the present status of the incinerator manufacturing industry. The study indicated that by far the incinerators used most often for hazardous waste destruction in the U.S. are liquid injection, fixed hearth, and the rotary kiln. Other equipment types, with more development, may see increased use for commercial destruction of hazardous wastes in the future.

Of the incinerator types available, the rotary kiln is the most applicable to incinerating soil (see Figure A-1). The rotary kiln offers a controllable solid retention time which

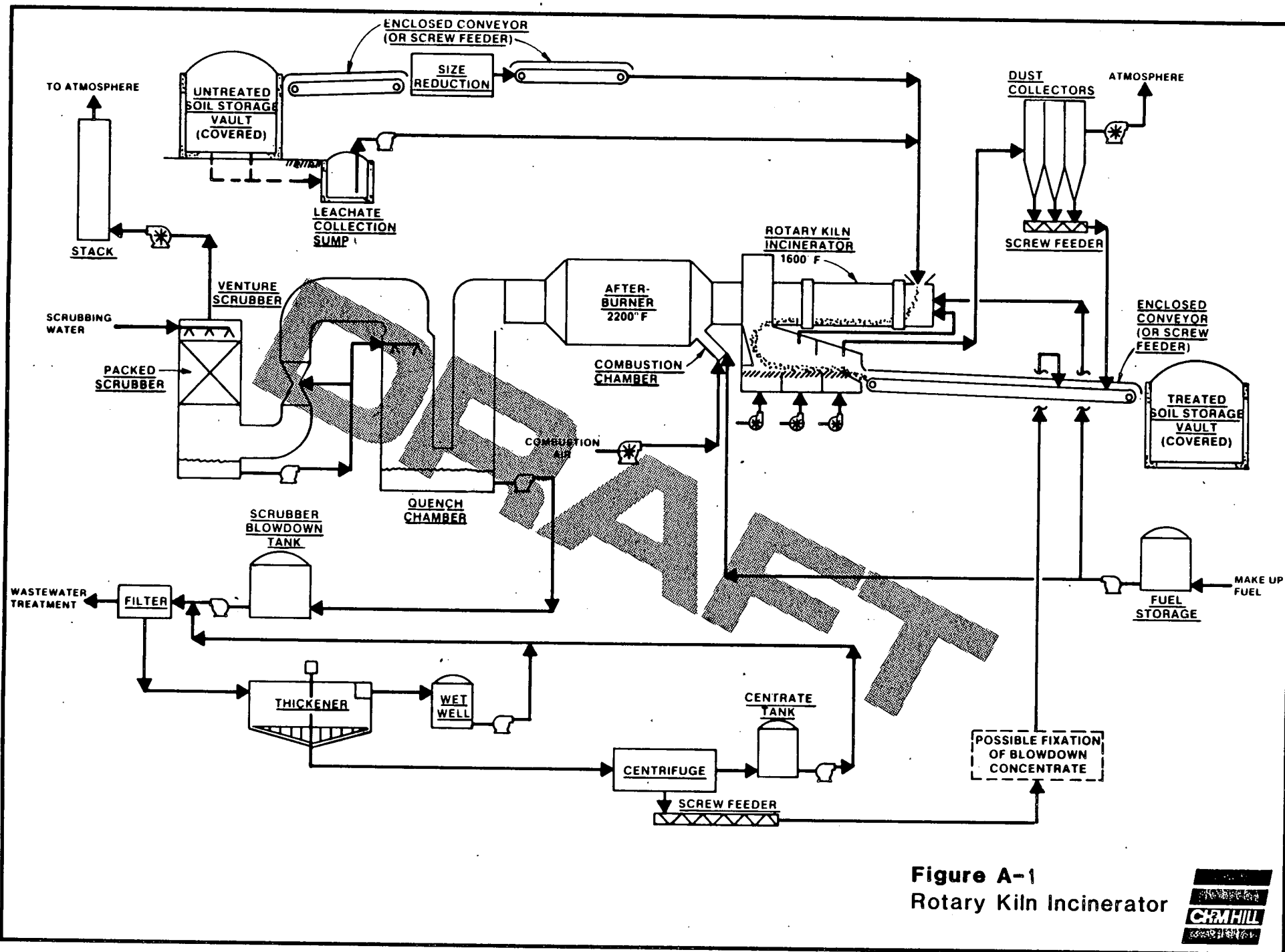


Figure A-1
Rotary Kiln Incinerator



can be as long as one to four hours, depending on kiln size, speed, and slope. Also, it is capable of maintaining temperatures as high as 2900°F. The rotary action of the kiln provides excellent air turbulence and solids mixing and tumbling which will enhance even heat transfer to soil particles.

The fluidized bed process has been used considerably less than the rotary kiln. Combustion temperatures approaching 1800°F are possible, but solid retention time in the bed is difficult to control. The system is not ideal for handling inert material, as it is difficult to remove residual ash from the fluidized bed. Literature states that the process is not well-suited for irregular or bulky wastes or wastes with a fusible ash content.

The rotary hearth, multiple hearth, and other moving or stationary hearth furnaces have seen extensive use in hazardous waste incineration, particularly fixed hearth incinerators. They are capable of maintaining temperatures of 1800°F with solids retention times greater than one hour. The lack of tumbling and mixing action (provided by the rotary kiln) may cause hearth incinerators to have less even heat transfer to soil particles.

Any of these primary combustion devices will leave a large portion of the raw soil and sediment feed as residue, or ash. The ash may contain traces of dioxin. Unless this ash is delisted from RCRA regulations, it will require disposal in a secure landfill.

All of the devices mentioned above for soil and sediment incineration may also incinerate solvent. However, a liquid injection incinerator may also be used. Liquid injection incineration is a technology with substantial operating experience. These systems are capable of incinerating virtually any liquid waste, but have no capability for solid incineration. Combustion temperatures on the order of 3000°F are possible with residence times ranging from 1/10 second to in excess of 2 seconds.

Secondary Combustion Devices: Once the dioxin is vaporized from the soil in the incinerator, it will be destroyed in a secondary combustion chamber or afterburner. This device will be either a refractory lined cylinder, or a large, baffled chamber or series of chambers; burners will be provided to heat the exhaust gas from the primary combustion device to a desired temperature and to retain the gas for a specified period of time. As previously mentioned, the conditions usually discussed for dioxin destruction are: more than 2 seconds residence time at 2200 to 2500°F with at least 3 percent oxygen in the final gas mixture.

Because of prior testing with dioxin-bearing liquids, the afterburner will require less testing than the rotary kiln operations. There are, however, some physical problems that may be caused by dust and ash carried into the afterburner with the gas from the kiln. Depending upon the quantity of entrained material, slagging on the walls of the afterburner can become a problem.

A secondary combustion chamber for a solvent incinerator would be similar to that for soil incineration. However, if the primary combustion chamber is designed to operate at the conditions necessary for dioxin destruction, it is possible that dioxin in a soil-free solvent could be destroyed without the use of a secondary combustion chamber. However, the likelihood of a high solid content in the residue from a solvent extraction process makes it probable that a secondary combustion chamber will be required even for solvent incineration.

Air Pollution Control Devices: Flue gases from the secondary combustion chamber of either a soil incinerator or from a liquid injection incinerator would require emission control devices, primarily for particulate removal.

In the sequence shown, the gas will first be quenched by spraying water into the gas stream, which will cool the gas to below 200°F. The next step will be a venturi scrubber, which will provide improved contact with water to assist in particulate removal. The final step will be a packed bed which will be sprayed with water.

Alkaline chemicals might be added to the packed tower sprays if required for removal of SO₂ or HCl. Formation of SO₂ and HCl are not expected from combustion of soil, but might occur if a large amount of some extraneous material such as salt (NaCl) or fertilizer (e.g., ammonium sulfate) is hauled from the site.

An induced draft fan will pull the gases through the emission control equipment and the entire incineration process. The induced draft is important because it will maintain a partial vacuum in the processing equipment, minimizing the potential for fugitive emissions of uncombusted dioxin.

Treatment of Scrubber Blowdown: The wastewater (blowdown) from the quenching and scrubbing operations will contain the particulates removed from the flue gas. Much of this particulate matter will be relatively insoluble and can be removed from the water by settling and centrifuging. Depending upon the analysis of the clarified water, further treatment may be required. For example, the wastewater may contain dissolved metals, which can make the water unsuitable

for direct discharge. Moreover, the discharge may have to meet strict standards.

Other Equipment: Size reduction equipment is needed for the incineration of soil for particle size control, to assure vaporization of the dioxin, and to minimize damage to a kiln.

A liquid injection solvent incineration process does not require particle size reduction, but does require filtering of the solvent to remove suspended solids before injecting the solvent into the incinerator. This filtering is necessary to keep the injection nozzles clean and operable. The solids residue and filter media will have to be disposed of, possibly in a hazardous waste landfill, if they contain traces of dioxin.

STATE OF DEVELOPMENT: Table A-1 shows some of the results of the search of current information regarding incineration of dioxin. To our knowledge, the only significant published test information regarding TCDD incineration is from the work by the Air Force to destroy Agent Orange and from published research by Dow Chemical. In both of these cases liquid waste was incinerated.

Cases involving the incineration of soils contaminated with waste chlorinated hydrocarbons other than TCDD are shown in Table A-1. The two cases shown are the only attempts to incinerate soil contaminated with hazardous chlorinated hydrocarbons similar to TCDD.

Table A-1 shows two examples of incineration of PCB's in electrical components. The difference in the final PCB concentrations between incinerating whole electrical components as opposed to hammermilled components indicates the importance of proper preparation of feed materials. The destruction efficiency during soil incineration will similarly be effected by feed preparation and size reduction procedures.

Hazardous Waste Incineration Facilities: Dioxin-contaminated soil or a dioxin-laden solvent can be destroyed in a dedicated hazardous waste incineration facility. Such a facility could be either an existing hazardous waste incinerator or it could be a newly constructed facility specifically intended for the purpose of destroying this dioxin.

Some commercial stationary waste incinerators are licensed to burn PCB's at several locations around the country. Also, the EPA and others are working to develop relocatable incinerators.

Table A-1
INCINERATION TECHNICAL STATUS

Category	Material Burned	Incinerator Type	Organization	Initial Concentration	Destruction Efficiency or Final Concentration	Comments
TCDD	o Agent Orange ^a	Liquid injection	USAF (on-board M/T <u>Vulcanus</u>)	1.9 ppm TCDD average	≥99.9% TCDD	Avg. flame temp., 1500°C Retention time, 1.0 sec. Between 7% and 12% excess O ₂ .
	o Agent Orange ^a	Liquid injection	Marquardt Co.	1.9 ppm TCDD average	99.96% TCDD	Flame temp., 1450°C to 1850°C. Retention time, 0.14-0.18 sec. Between 6.6 and 9.7% excess O ₂ .
	o 2,3,7,8-TCDD test standard ^a	Bench-scale quartz-tube heater	Dow Chemical	Unknown	99.5% TCDD	Reactor temp., 1470°F Retention time, 21 sec. Oxygen concentration unknown.
Soil	o PCB-contaminated ^b soil	Rotary kiln w/afterburner	ENSCO	Unknown	Unknown	
	o 3,3-dichlorobenzene ^b contaminated dredgings	Rotary hearth w/afterburner	Midland Ross Surface Combustion Division	1300 ppm	<4 ppb	Starved air combustion in rotary hearth.
PCB's	o Electrical components (whole) ^c	Rotary kiln w/afterburner	Rollins Env. Services	.74 $\frac{\text{kg PCB}}{\text{kg ash}}$	470 $\frac{\text{mg PCB}}{\text{kg ash}}$	
	o Electrical components (hammermilled) ^c	Rotary kiln w/afterburner	Rollins Env. Services	.74 $\frac{\text{kg PCB}}{\text{kg ash}}$	<0.1 $\frac{\text{mg PCB}}{\text{kg ash}}$	

^a Liquid state

^b Solid state

^c Oil in non-combustible solid

A recent study identified 57 manufacturers of hazardous waste incinerators in the U.S. A large number of the existing hazardous waste incinerators produced by these manufacturers are in private use onsite by their owners.

EXISTING HAZARDOUS WASTE INCINERATOR FACILITIES

Two commercial incineration firms have expressed preliminary interest in destroying the dioxin contaminants in their existing stationary incinerators. These firms are Rollins Environmental Services Co. of Deer Park, Texas, and SCA Chemical Services Co. of Chicago, Illinois. Both firms have incinerators licensed to burn PCB's and believe it may be feasible to bring the dioxin into their respective states. Both firms indicate that they may have the capability to effectively incinerate the contaminated soil. ENSCO, another commercial incineration firm, has not expressed interest in incinerating the dioxin soil at their El Dorado, Arkansas, facility.

Either of these existing hazardous waste incineration facilities could destroy dioxin in a soil-free solvent form with little modification to the facility. However, if soil were to be incinerated at one of these facilities, extensive equipment retrofits would be necessary.

Because there are no solid hazardous waste incinerators in New York, controlled transport of the soil out of the state will be necessary. Enclosed storage of the soil at the incinerator site will be required, including leachate collection and fugitive emission control. A size reduction facility will have to be constructed to prepare the raw soil for incineration. Also, improved ash handling equipment will probably be required to take care of fugitive emissions and controlled ash storage.

CONSTRUCTION OF A NEW HAZARDOUS WASTE FACILITY

A new facility for the destruction of the dioxin can be constructed to deal with the dioxin soil. Such a facility will need all of the equipment for the incineration process, either for soil or solvent, as described previously. For soil, this includes enclosed soil storage, leachate control, size reduction, primary and secondary combustion equipment, air pollution control devices, wastewater treatment, ash handling and storage, and equipment for fugitive emission control. An ample supply of fresh water will be required, as well as facilities for treated water discharge. The solvent incineration facility may be somewhat simplified, but will include most of the above equipment.

A location for a new incineration facility will probably have to be remote, yet have facilities for maintenance shops, testing laboratories, office space, housing accommodations, parking, fuel storage, and material loading and unloading for transport. The siting and permitting for such a facility will be an extensive process, and will take into account geologic, hydrogeologic, topographic, and political factors.

USE OF RELOCATABLE INCINERATORS

Relocatable incinerators, mounted on tractor trailers or flatcars, could be used as the basic incinerator portion of a somewhat temporary hazardous waste incineration facility. Trailer-mounted liquid injection units and rotary kiln/afterburner systems are available.

The Tracor-MBA Co. of San Ramon, California, has constructed such a relocatable incinerator under contract with the EPA. The trailers include rotary kiln, afterburner, air pollution equipment, and fuel storage. To date, it has not incinerated dioxin, but a test is planned for February, 1985.

Other units may soon be available from Pyrotech Systems, Inc., of Tullahoma, Tennessee, and Pyro-Magnetics Corp., of Whitman, Massachusetts.

OCEANGOING INCINERATOR SHIPS

One of the few published tests monitoring dioxin destruction efficiencies in an incinerator was conducted during at-sea destruction of Agent Orange on board the M/T Vulcanus in 1974-1977. The successful destruction of dioxin in the liquid herbicide showed that dioxin could be destroyed under certain conditions and that at-sea incineration of waste solvents may be a practical technology.

Presently, however, there appears to be no commercial development of an incinerator ship to incinerate solids in a rotary kiln. Though studies have addressed this topic, at the present time at-sea incineration applies only to liquid wastes. Three firms are currently building new liquid incinerator ships, though there is some disagreement as to the cost-effectiveness of at-sea incineration.

There is some difficulty involved with permitting and licensing a port facility for storing and loading hazardous wastes for these incinerator ships. Assuming one or more such ports are licensed in the country, there would be some transportation necessary to get the dioxin-contaminated solvent to the port.

MODIFIED CEMENT KILNS AS HAZARDOUS WASTE INCINERATORS

A modified cement kiln could be used as a type of rotary kiln incinerator to incinerate liquid or solid wastes. Current research is in the use of cement kilns, both for co-incineration of wastes while producing cement, and in the use of a modified kiln to incinerate solids without the cement product.

There are two methods of modifying a cement kiln to incinerate solids without manufacturing cement:

- o Pass solids through the existing kiln and add an afterburner for the exhaust gases.
- o Divide the kiln into sections and provide supplementary burners to make a primary combustion chamber for solids and an afterburner for gases.

There are other methods to coincincinerate waste while manufacturing cement. These include:

- o Liquid injection of waste into the hot end of a kiln
- o Comingling solid waste (soil) with the clinker in the kiln
- o Incinerating solids (soil) in a separate prekiln and using the cement kiln as an afterburner

These last three methods will be discussed in the Coincincineration section of this appendix.

Either a wet process cement kiln or a dry process kiln could be used to incinerate wastes. However, the wet process is the more widely used. Wet process kilns vary in size, ranging up to 25 feet in diameter and up to 760 feet long. Gas temperatures may reach 3000°F at the hot end of a typical wet process kiln and drop to 800°F at the cold exhaust end. Solids pass countercurrent to gas flow and typically are subjected to temperatures ranging from ambient at the cold end to 2700°F at the hot end of the kiln. Retention times in a kiln may be from one to four hours for solids and up to 10 seconds for gases.

There is some incentive to investigate the use of existing cement kilns as hazardous waste incinerators because any idle cement kiln could potentially be used. However, any use of a cement kiln for hazardous soil incineration will require new facilities for enclosed soil storage, soil size reduction, improved ash handling and storage, improved

fugitive emission control, and possibly air pollution control modification. Other equipment will be required as dictated by the approach taken to the kiln modifications.

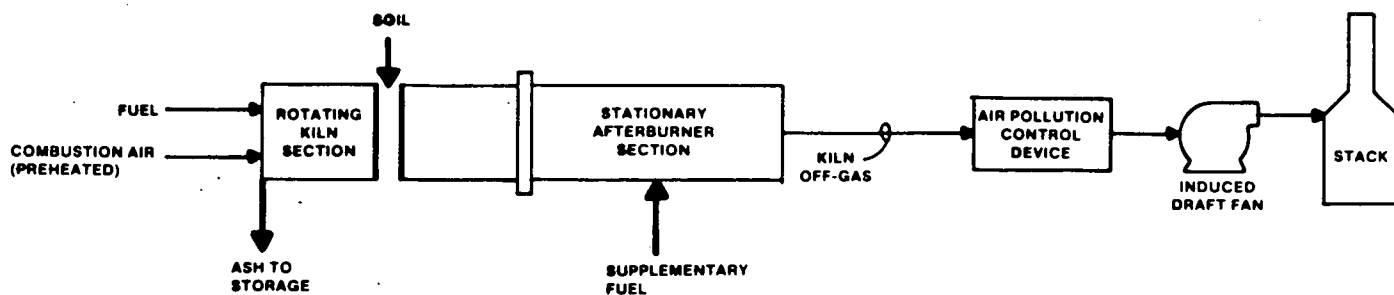
Any cement kiln designed to be fired on coal, if used as a soil incinerator without cement product, will require major modifications for exhaust gas scrubbing equipment.

Also, because of the possibility of fusing and slagging of the soil and any metals contained in soil and sediment, the temperature the soil is exposed to should be limited to 1800°F. As previously discussed, dioxin probably will be destroyed at 2200°F. For this reason, a single kiln cannot act as both primary and secondary combustion chamber without extensive modifications. Therefore, any cement kiln will have to undergo major renovations if it is to be used as a hazardous waste incinerator.

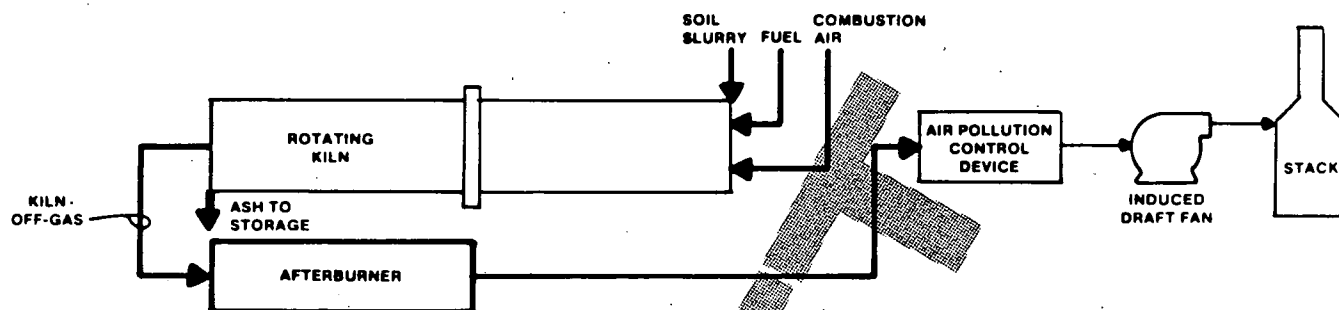
One method of modifying a kiln has been developed by Marblehead Lime Co. of Chicago, Illinois. They have segmented an existing kiln to create a rotating primary combustion section in series with a stationary, supplementary fired, secondary combustion chamber. This concept is shown schematically in Figure A-2A, and the company will be conducting pilot tests in the near future. Soil could be fed into the mid-section of such a kiln and pass through the primary chamber at less than 1800°F. Combustion air would flow countercurrent to the soil, pick up the vaporized dioxin, and then enter the secondary combustion chamber for high-temperature dioxin destruction. Extensive modifications are required including new rotary seals at mid-sections of the kiln, a rotary feed mechanism for soil feed, supplementary burners and controls for the secondary combustion chamber, and possibly new air pollution control equipment.

Another method of modifying a kiln is to pass the soil through the existing kiln and add an external afterburner in the exhaust gas stream. This method is shown in Figure A-2B. The soil could enter the kiln and flow cocurrently or countercurrently to the air. Temperatures in the kiln would be kept below 1800°F, and the exhaust gases would enter the external afterburner. The modifications would include the new afterburner, and possibly reversing the air flow in the kiln by relocating the burners and combustion air ducting to the opposite end of the kiln. New air pollution equipment may also be needed. This approach is similar to the approach assumed by the EPA Office of Solid Waste in their preliminary study of Decontamination of Dioxin-Containing Soil in an Upgraded Cement Kiln for Region VII EPA.

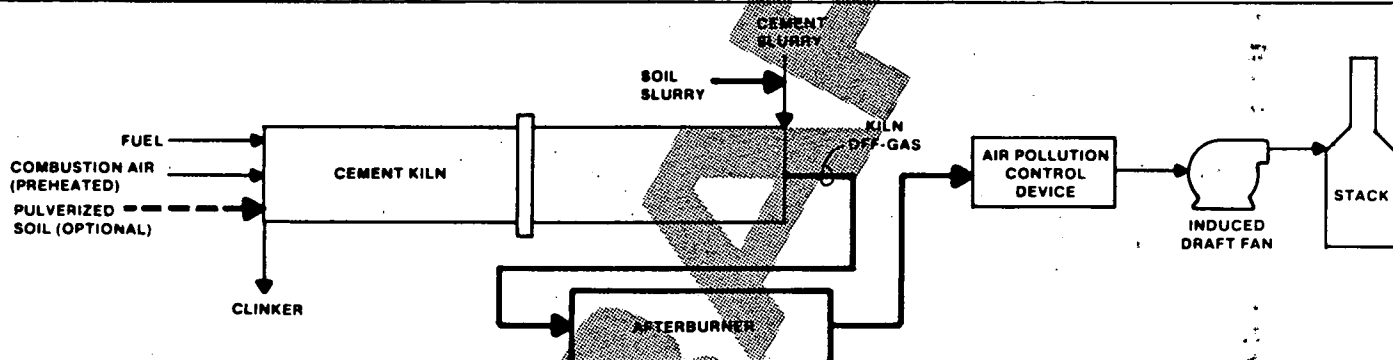
Coincineration: Coincineration is the incineration of a waste material as a secondary purpose in an incineration/



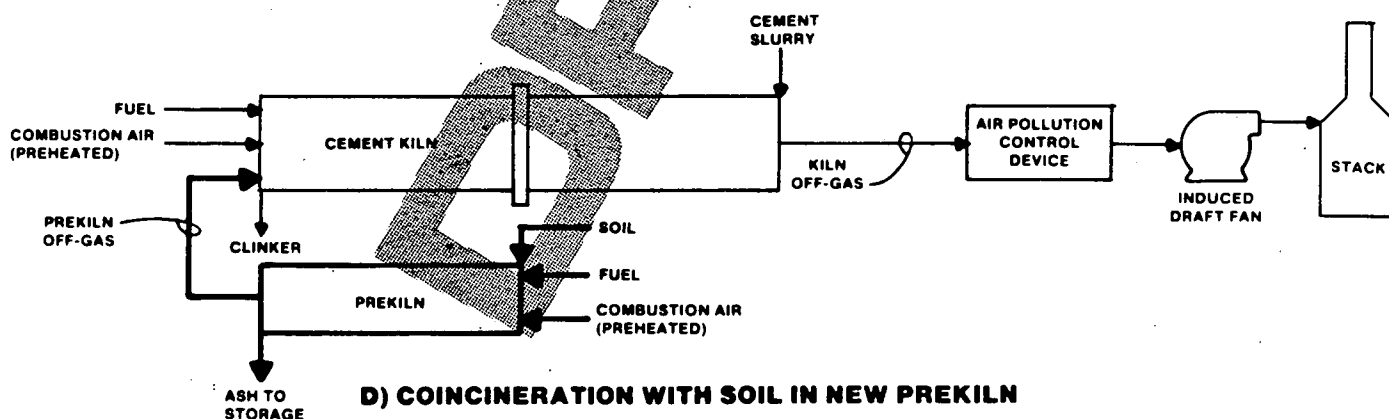
A) WASTE INCINERATION IN SEGMENTED KILN



B) WASTE INCINERATION WITH NEW AFTERBURNER



C) COINCINERATION WITH SOIL IN CEMENT PRODUCT



D) COINCINERATION WITH SOIL IN NEW PREKILN

LEGEND:

— NEW

— EXISTING

Figure A-2
Cement Kiln
Conceptual Flow Diagrams



combustion process. Coincineration of hazardous wastes can be and has been performed in the following types of equipment:

- o Cement kilns
- o Municipal solid waste (MSW) incinerators
- o Utility boilers

Dioxin-contaminated soil sediment or solvent could be coincinerated in any of this equipment.

CEMENT KILNS

The use of cement kilns to coincinerate liquid hazardous wastes while producing cement has seen considerable research and success. The work at St. Lawrence Cement Co., Peerless Cement Co., Stora Vika Cement Plant, San Juan Cement, and others show the possibilities of liquid waste destruction by the cement industry. However, the work at San Juan Cement showed some difficulty in maintaining kiln operating stability and also in achieving even 99.9-percent destruction of the chlorinated wastes used in the tests.

Another method coincinerating a waste while producing cement is to comingle a solid waste, in this case soil and sediment, with the cement product. There has been no substantial research on this approach. It is possible to carry out this process by two methods. Either soil could be added directly to the cement feed to the kiln, or the soil could be pulverized and sprayed into the hot end of the kiln. However, in either case, the soil residue may act as an impurity in the cement product. The quantity of soil and sediment incinerated may have to be small in comparison to the amount of cement produced to assure cement quality. In addition, this method requires that soil temperatures reach the same temperature as the cement clinker, possibly 2700°F, which may cause vaporization of any metals in the soil and sediment. These methods are shown in Figure A-2C.

In either of these two methods, an exterior afterburner may have to be added in the exhaust gas stream to ensure destruction of any vaporized dioxins. Especially where soil and sediment are added to the cement slurry feed in the cold end of the kiln there is a potential for dioxins to vaporize from the sediment in the cold end of the kiln and exit the kiln before being destroyed.

Where soil and sediment are pulverized, slurried, and sprayed into the hot end of the kiln, there is less chance of dioxin vapor passing out of the kiln. However, the retention time in the kiln may be limited. The dioxin may not have an opportunity to vaporize from the particles before the particle exits the kiln in the clinker.

Equipment needed for these options would include enclosed storage and leachate collection, pulverizing and slurry feed equipment, as well as possibly an exterior afterburner and new air pollution equipment.

A final method to co-incinerate soil and sediment while manufacturing cement is to install an auxiliary kiln or a pre-kiln at the cement plant. This kiln would serve as the primary combustion chamber for soil and sediment, while ductwork would carry its dioxin-bearing exhaust gases to the hot end of the existing cement kiln. The cement kiln would then act as the secondary combustion chamber. This option, shown in Figure A-2D, is currently under consideration by Environmental Specialists, Inc., of Kansas City, Missouri, in co-operation with River Cement of Festus, Missouri.

Equipment needed to accomplish this method will include the prekiln, ductwork and fans, air pollution control equipment, enclosed soil storage and leachate collection, soil size reduction equipment, ash handling and enclosed storage, and improved fugitive emission control. The ash from the pre-kiln will have to be transported from the cement plant to a landfill for disposal.

MUNICIPAL SOLID WASTE (MSW) INCINERATORS

MSW incinerators have been studied for co-incineration of pesticides and herbicides during the destruction of municipal sludge. This has proved somewhat effective for these liquid wastes, but it would not be as effective for soil and sediment incineration. MSW facilities usually use hearth incinerators (either fixed- or multiple-hearth, or shaker-grate). Existing plants may have limitations on ash handling capabilities, fuel firing rates, grate designs, combustion temperatures, and solid and gas retention times that may affect their applicability to destruction of dioxin and soil incineration.

In general, MSW incinerators, as other incinerators, can be either excess oxygen type or starved air types. The excess oxygen units typically do not have the temperature capabilities to vaporize and destroy dioxin from soil. These excess air systems may require major modifications to handle dioxin destruction, including a retrofit high-temperature secondary combustion chamber for the plant's exhaust gases.

The starved air MSW incinerators must have secondary combustion chambers included in their design. Therefore, they may be capable of vaporizing the dioxin from the soil and sediment at temperatures below the fusion point, and destroying the dioxin in the secondary combustion chamber. Typical temperatures in these MSW secondary combustion

chambers may be as high as 2000°F, so it may be possible to operate a secondary combustion chamber at the conditions required for dioxin vapor destruction.

However, for both the starved air and the excess oxygen systems, the heat transfer and retention time of the soil may not be ideal. Satisfactory vaporization and destruction of dioxin from the soil and sediment may not be accomplished. Feed preparation (size reduction) would be required before soil and sediment entered the incinerator, and enclosed storage and possibly ash storage would have to be built on-site.

UTILITY BOILERS

High-efficiency utility boilers have been tested for PCB destruction by coincineration of liquids. This approach may be satisfactory for solvents containing dioxin only if gas temperatures and residence times within the boiler can meet the 2200°F and 2-second criteria. The excess oxygen requirement of 3 percent is probably above the efficient operating oxygen level for a large boiler, and may therefore require excess fuel for boiler operation. The coincineration of sediment and soils in a boiler has additional limitations. A solid fuel boiler would have to be used so that ash handling equipment would have sufficient capacity to carry the residue from the firebox. It is likely that the limitations of this ash equipment would severely restrict the throughput of soil and sediment. Again, temperatures and residence times are important. But with soil in the firebox, dioxin would have to be first vaporized, then destroyed. Therefore, long solids retention times with temperatures less than 1800°F would be required in the boiler firebox. The boiler would have to be retrofitted with a secondary combustion chamber to bring its exhaust gases up to 2200°F for 2 seconds. Overall, this would result in severe penalties to boiler efficiency and in major retrofitting expenses.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: The primary research need is to test the ability of an incineration process to effectively destroy dioxin in soil to below the 1-ppb limit. Variables such as temperature, retention time of both solids and gases, and oxygen requirements for this waste must be determined before a full-scale incinerator could be built. Other information must also be collected before full-scale design could begin. This includes soil, sediment and particulate fusion temperatures, and determination of waste materials other than dioxin that may be in the soil and sediment.

The effect of particle size and moisture content on the effective incineration of the dioxin must also be investigated. One incineration firm, Rollins Environmental Services, indicated that they could not guarantee that incineration of the soil and sediment in their incinerator would leave ash with less than 1 ppb dioxin without costly testing and taking major steps to control particle size and agglomeration in the incineration process.

Also, Dow Chemical, Inc., of Midland, MI, has indicated that dioxins bound to particulate matter have passed through incinerators at 2100°F without any change in dioxin concentration. There are no data available to investigate this possibility. The impact of this statement could significantly effect incinerator design criteria, and it should be investigated further.

References (see Section 11): 58, 60, 73, 74, 77, 79, 81, 87, 91-93, 96, 100, 101, 106, 107, 109, 121, 123, 129, 137, 139, 140, 142, 144, 152, 156, 165, 167, 169, 179, 180, 182, 195, 198, 203-205, 210, 223-226, 228, 229, 231, 232, 234, 239, 242, 248, 250, 251, 258, 261, 264, 271, 275, 279

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Ultraviolet Degradation

DESCRIPTION OF TECHNOLOGY: Ultraviolet degradation is the process of breaking chemical bonds with ultraviolet (UV) light. Ultraviolet degradation is achieved by exposing a compound in a suitable medium to a sufficient intensity of UV light from a specific wavelength range.

STATE OF DEVELOPMENT: A substantial amount of laboratory and small-scale field test research has been performed on UV degradation of TCDD. A general conclusion is that, for UV degradation of 2,3,7,8-TCDD to take place, the following conditions must occur: 1) TCDD must be dissolved in a suitable light-transmitting solvent phase; 2) a suitable organic hydrogen donor must be present (e.g., methanol, diesel oil, olive oil, liquid phenoxy ester, hexane, xylene, etc.); 3) UV light in an appropriate range of wavelengths for dioxin in solution must be supplied and absorbed by TCDD in the solvent phase. The TCDD then is photodegraded to dichloro and trichloro homologs, which in turn are photodegraded to "non-toxic" dibenzo-p-dioxin (no chlorines). There is evidence that dibenzo-p-dioxin undergoes additional UV degradation, thereby destroying the dioxin structure.

The TCDD photodegradation rate is solvent-dependent; solutions of certain cationic surfactants (like 1-hexadecylpyridinium chloride, CPC) appear to enhance photochemical degradation of TCDD. One study showed "total" TCDD degradation in 4 hours in a 0.05 M CPC solution compared to 18 hours for "total" TCDD degradation in methanol. Numerous studies concur that the photodegradation of dioxin in suitable organic solvents is relatively quick and complete.

A solvent extraction/UV photolysis process, conceptualized by IT Enviroscience, was piloted and used in a full-scale application in Verona, Missouri. In that application, the UV process substantially destroyed the dioxin in 4300 gallons of dioxin-laden sludge at the Syntex Agribusiness plants. In the first process step, dioxin was batch extracted with hexane (which reduced the dioxin concentration in the sludge from 343 ppm to less than 0.5 ppm). In the second step, the extract solution was exposed to UV light from 10-kW, high-intensity mercury lamps for an average of 20 to 40 hours to break down the dioxin. The solvent was then distilled for recycling within the process (the waste solvent contained 0.1 ppm dioxin). The equipment and installation cost was \$500,000. This process was chosen for high efficiency, low pressure, low temperature, self-contained system, safety for personnel and surroundings, and economic feasibility.

TCDD photodegrades only very slowly, if at all, from inert surfaces, dry soil, wet soil, or in aqueous suspensions. The addition of a hydrogen-donating organic solvent can result in substantial photodegradation in surface layers. Ninety percent TCDD destruction was observed over a 9-day period in Seveso after the application of olive oil (as a 40-percent aqueous emulsion or as an 80-percent solution in cyclohexanone) to TCDD-contaminated grass. Lower photodecomposition rates for TCDD in Herbicide Orange were observed from soil as compared to glass surfaces or leaves, presumably because lower layers were shaded by soil particles. Studies have shown that partial TCDD destruction can occur in soil at least up to 3 cm deep upon application of a hydrogen-donating solvent and after exposure to intense sunlight or artificial UV radiation. In this same study, virtually total TCDD destruction was observed in the top one cm of soil with 9 days exposure to summer sun in Rome.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: UV degradation of dioxin in solvent in a full-scale process has been demonstrated. Further development is needed to determine what level of dioxin concentration reduction is feasible. Much research and development is needed to determine if and when UV destruction of dioxin in soil is feasible.

Reference: 62, 66, 110, 126, 141, 180, 187, 188, 189, 191, 238, 275, 276, 278

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Biological Treatment

DESCRIPTION OF TECHNOLOGY: Biodegradation is the molecular breakdown of an organic substance resulting from the biochemical reactions of living organisms. For TCDD, microbial enzymes, or combination of enzymes, are regarded as the principal catalyst of these desired reactions.

Using radioactive carbon labeling to trace microbial breakdown with subsequent production of metabolites, a slow breakdown of TCDD has been monitored under laboratory conditions. This rate of reaction has been estimated to be between 2 to 5 percent of the TCDD present per year.

Biomanipulation and bioengineering research is currently directed at producing a microbe capable of detoxifying dioxin in a natural system. The principal limitations in developing a microbe capable of degrading TCDD in a soil system include:

- o TCDD's toxic effects on microbial metabolism
- o Energy-starved soil systems, which reduce overall microbial activity
- o Low TCDD content in soil, reducing the opportunity for microbial adaptation as an energy source
- o The problems of transferring an engineered microbe from a laboratory into a natural environment
- o Low water solubility of TCDD

STATE OF DEVELOPMENT: Biodegradation research can be categorized into two groupings differentiated by the technique used for analyzing the TCDD content of soil. Before 1980, the technique consisted of first extracting TCDD from the sample with methanol and then with dichloromethane. The TCDD was concentrated into dichloromethane by diluting the combined organic phases with water. Analysis utilized gas chromatography with electron capture detector.

In 1980, Dr. Ralph Hutter, a biologist at the University of Zurich, identified a binding phenomena between TCDD and soil, which formed an undefined TCDD/soil complex. TCDD became increasingly bound to the soil with time, decreasing the amount that could be extracted by the methanol and dichloromethane solvents. Therefore, TCDD was not completely recovered from the soil by the solvent and was not observed in the analysis. This loss was accredited to either

biological degradation, UV light degradation, evaporation, or some unknown factor. Since the measured TCDD content was in error, the interpretation of data, likewise, was incorrect.

Following the discovery of this binding phenomena, a more rigorous extraction technique has been used for extracting TCDD from soil. A Soxhlet extractor is now used to reflux toluene or hexane through the soil sample for 24 hours.

Research before 1980 monitored different aspects of TCDD degradation in soil environments. Kearney, et.al., (1973) monitored the effect of various soil types on TCDD degradation. Soils with higher organic contents had lower levels of recovered TCDD with the passage of time.

Kearney and co-workers studied two types of soil inoculated with TCDD at concentrations of 1, 10, and 100 ppm with ^{14}C -labeled TCDD. The two soils were also inoculated with unlabeled TCDD. Over a 9- to 10-month period, the soil samples were monitored weekly for evolution of carbon dioxide as an indication of microbial degradation of the labeled dioxins. Very little CO_2 was liberated from soils containing either labeled or unlabeled TCDD. In most cases, 75 to 85 percent of the dioxin was recovered from both soil types up to 160 days after addition. No ^{14}C metabolites were found in TCDD-treated soil after one year.

Matsumura and Benezet (1973) monitored the biodegradation of persistent pesticides using 100 microbial strains. Five strains were suspected of having a limited capability for metabolizing TCDD, though no specific metabolites were identified.

Camoni, et.al. investigated the microbial degradation of TCDD in soil by adding organic compost. Compost, with its indigenous high microbial activity, was not documented as affecting TCDD degradation.

Young, et.al. investigated the biodegradation of TCDD administered to soils in Florida, Kansas, and Utah via application of Herbicide Orange. The parameters monitored in this test included TCDD soil penetration, TCDD biodegradation in soil, TCDD uptake by animal life, and the effect of charcoal on TCDD degradation. TCDD was detected principally on the soil surface, with less than 25 ppt below 30 cm. TCDD loss was assumed to be due to biodegradation, with an estimated half-life of 225 to 275 days. The measured TCDD content in rodent, bird, reptile, insect, and fish tissue was not higher than environmental levels. Finally, TCDD adsorbed on the surface of charcoal was not desorbed or degraded by environmental conditions.

In light of the TCDD/soil binding phenomena, the early research which predicted a TCDD half-life of 200 to 300 days was actually documentation of TCDD becoming bound to soil. This binding made it increasingly difficult for the extraction solvents to solubilize the TCDD for analysis. In support of this phenomena, Dr. Al Young performed an exhaustive extraction process on TCDD/soil samples, and was able to account for virtually all of the TCDD originally detected in the sample. The original technique, using the methanol/dichloromethane extraction, indicated a time-related binding phenomena in the TCDD/soil complex.

The TCDD/soil binding mechanism is not well understood. The mechanism is suspected to be either a surface adsorption, preferential covalent bonding between the TCDD and soil humus, or an encrustation into the soil structure.

The mechanism of TCDD/soil binding and the subsequent effect on absorption was demonstrated by Poiger and Schlatter in a 1979 study. For any material to be toxic to living organisms, it must be adsorbed into the system before it can interfere with normal metabolism. This study investigated the influence of solvents and adsorbents on the rate of TCDD absorption through the skin and intestine of rabbits. After oral administration of 14.7 nanograms TCDD using 50 percent ethanol as the vehicle, 36.7 percent of the total dose was found in the rabbit liver after 24 hours. When TCDD was mixed with soil particles and administered, only about half of the original absorbed dose was found in the liver. The absorbed dose and monitored liver level also decreased with increased time of contact between the soil and TCDD. Adsorption onto activated carbon almost completely prevented uptake of TCDD.

Similar effects were observed after dermal application of TCDD on rabbits in various formulations. For TCDD absorption through the skin, the highest liver content, 14.8 percent of the dose, was found after contact of the pure compound with the ear surface. The inhibiting effects of soil and activated carbon were even more pronounced with ear absorption. After incorporation of the dioxin into Vaseline (a lipophilic ointment), 1.4 percent of the dose was found in the liver whereas after incorporation into polyethyleneglycol 1,500 (a hydrophilic ointment) containing 15 percent water, 14.1 percent was found in the liver. The capability of microbial absorption is essential for innercellular degradation. This binding complexing phenomena is important because the TCDD bioavailability is a fundamental limiter to accommodating biological degradation.

Hutter, et.al. completed a 2-year study on the biodegradation of TCDD in contaminated soil at Seveso, Italy. The

results showed a TCDD metabolite production rate of 2 to 5 percent of the input TCDD after one year of incubation. The ^{14}C was used to trace TCDD and its metabolites. The metabolite detected was $^{14}\text{CO}_2$. Due to ^{14}C -labeled impurities in the TCDD sample, most of the $^{14}\text{CO}_2$ was theorized to be metabolites of these impurities.

The microbial degradation in the laboratory occurred so slowly that it was not useful to purify the Seveso soil. The microbial populations that were tried included natural unsterilized Seveso soils and Pseudomonas strains capable of degrading chlorinated phenols. No bioengineered strains were used.

Matsumura, et.al. have identified polar metabolites from ^{14}C -labeled TCDD in work with aquatic and soil systems. Two microbes were specifically identified which are capable of co-metabolizing TCDD in the presence of other specific aromatic compounds. The metabolites containing the ^{14}C traces were Nocardiopsis and B. megaterium.

Dr. D. T. Gibson and G.M. Klecka at the University of Texas in 1982 documented that dioxin toxicity to microbes was increased by higher degrees of chlorination. Two specific genera of microbes, Pseudomonas and Beijerinckia, were grown in the presence of a reducible carbon energy source and either nonchlorinated dibenzo-p-dioxin, monochlorinated dibenzo-p-dioxin, and two dichlorinated dibenzo-p-dioxins. In summary, the unsubstituted dibenzo-p-dioxin can be readily metabolized by a mutant strain of Pseudomonas with an alternative source of reducible carbon for energy. With greater chlorine substitution on the dioxin molecule, microbial degradation is reduced. No organisms capable of utilizing dibenzo-p-dioxin were found as a sole carbon source. This work supports the observed increase in microbial toxicity of dioxins with increased chlorine substitution.

The viability of bioengineering a microbial genetic code to effect TCDD detoxification has been questioned. If microbial degradation is observed in the environment, it could probably be bioengineered. The basic steps from identification of the desired reaction to successful bioengineered microbe application include:

1. The microbe must be isolated from its environment and propagated in an incubator.
2. DNA must be restructured, combining a desired growth rate gene with a desired gene capable of creating the desired enzyme.

3. The microbe with the recombined DNA must be capable of thriving when reintroduced into the natural system.

If the desired microbial degradation is not observed in the environment, a soil system can be created to maximize microbe diversity. Microbial activity increases cellular reproduction, which increases the opportunity for microbe adaptation to a new reducible carbon food source.

In a dilute soil matrix, TCDD is particularly difficult to biodegrade for the following reasons:

- o The low TCDD concentration in the soil documented in parts per billion provides insufficient exposure to allow microbes to adapt.
- o TCDD is virtually insoluble in water, i.e., .2 ppb, and most microbial systems require metabolizable nutrient sources to have a greater water solubility.
- o Soil is an energy-starved system lacking a sufficient reducible carbon source, which supplies the cellular energy for normal microbe metabolism. Less than 1 percent of the microbes are normally active in soil systems due to this energy-starved state. This state is contrasted with a reducible-carbon-rich, energy-rich system, such as an organic compost pile. The opportunity for microbial manipulation decreases proportionately with the rate of microbial activity.
- o TCDD bioavailability to microbe metabolism appears to be severely limited. Thus, TCDD is essentially a permanent component of humus such that its release in any significant quantity as either the parent material or similar product is limited.

A 2-year research program, funded by the EPA, into microbe bioengineering for TCDD degradation is being conducted by Dr. Chakrabarty, University of Illinois Medical Center. Dr. Chakrabarty has been successful in biogenetic manipulation of microbes, producing microbial system capable of detoxifying 2,4,5-T and dibenzothiophene. Neither of these biochemical reactions had been observed in nature before the biogenetic manipulation began. Of particular interest to TCDD detoxification is the microbial detoxification of dibenzothiophene (DBT), which is water-insoluble. A mixed microbial culture was developed capable of utilizing carbon and sulphur in DBT as an energy source.

Dr. Chakrabarty is "hopeful" of developing a TCDD-reducing microbial system. He identifies the principal issues for microbial manipulation as:

- o TCDD has been demonstrated to be toxic to microbial systems
- o The low TCDD content in the soil limits microbes in "locating" the material, limiting their adaptation to the TCDD as a potential carbon source.
- o The manipulated microbe system must thrive in nature.

Dr. Chakrabarty also is "hopeful" that a microbial system for TCDD degradation can be developed. If a microbe is identified, the degradation trait can be passed on to gram negative microbes which are prolific in nature.

However, it is the general consensus of microbiologists familiar with TCDD degradation that biodegradation of TCDD in a natural soil system will not be possible for at least 5 years. Several significant limitations to biodegradation will need to be overcome after a microbial strain with the desired characteristics is isolated. Some of these limitations are:

- o Once a microbe is isolated, a population will need to be generated of sufficient size to handle the extent of the contamination at sites such as Love Canal.
- o Once the microbes are applied to the contaminated soil, providing nutrients to the microbial population will be difficult.
- o Likely, a microbe that degrades TCDD will survive and function only within specific environmental limits, and controlling environmental factors in a natural soil system will be difficult.
- o To avoid competition and interference from other microbes, the natural soil system may have to be sterilized before application of the TCDD specific microbe.
- o Achieving the desired one-ppb concentration of dioxin in soil will be difficult since that is a very low concentration for any significant microbiological activity.

FUTURE RESEARCH NEEDS: The EPA, in coordination with the Air Force Engineering and Services Center, is supporting a research program to establish organisms that will ultimately biodegrade dioxins, especially TCDD, and other recalcitrant chlorinated compounds in the following media:

- o Contaminated soils
- o Leachate from disposal sites
- o Contaminated aqueous effluent from chemical plants

The funded work includes the following studies:

University of Illinois Bacteria Study: Researchers at the University of Illinois have developed a mixed culture of bacteria that can break down 2,4,5-T. This technique involved combining the plasmids of naturally occurring organisms (isolated from areas previously contaminated with chlorinated compounds) with the plasmids of other organisms whose specific degradative functions or capabilities had been previously studied and defined. The organisms were exposed to gradually increasing concentrations of 2,4,5-T over a 6- to 9-month period. A mixed culture was eventually obtained that would not only survive in the 2,4,5-T solution but could use it as its sole food source. Through further experimentation, a pure culture responsible for the 2,4,5-T degradation has been isolated from the original mixture of bacteria. By using the gradual acclimation method used with 2,4,5-T, it may be possible to develop a form or variant of the 2,4,5-T organism that will degrade compounds in the dioxin family. This is the goal of the second phase of this program, which began in February 1983.

University of Helsinki Bacteria Study: Research at the University of Helsinki in Finland developed organisms that could degrade pentachlorophenol in either anaerobic or aerobic conditions. Since these organisms appeared to be able to degrade a specific chlorinated compound, researchers felt they might also degrade dioxins. In initial tests, the organisms would not degrade the most toxic dioxins (2,3,7,8-TCDD). Consequently, further efforts have been redirected toward understanding the degradative pathway for pentachlorophenol. It is hoped that further studies will reveal the mechanisms for biodegradation of recalcitrant chlorinated compounds.

Battelle Columbus Laboratories Plant Study: A project entitled "Development of Photosynthetic Plants Genetically Adapted to Degrade Organo-Chlorine Compounds" is being conducted at Battelle Columbus Laboratories. The goal is to modify plant cultures so that original and subsequent generations of the plants can actually tolerate and degrade toxic chlorinated compounds. In studies at Battelle, the

researchers are exposing plant cultures (milkweed initially) to different chemicals such as (1) hexachlorobenzene, (2) 2,2',3,3',5,5'-hexachlorobiphenyl (a PCB isomer), (3) pentachlorophenol, and (4) "Lindane"-gamma-hexachlorocyclohexane in an attempt to determine if they will degrade the compounds in question or determine if the plants can be modified to perform the desired degradation.

The results to date indicate that milkweed is unable to bioaccumulate and/or degrade hexachlorobenzene. Studies on the other compounds continue.

University of Cincinnati Yeast Study: The project being conducted at the University of Cincinnati is entitled, "Genetic Engineering of Yeasts for the Degradation of Hazardous Waste." This work entails the utilization of cytochrome P-450 enzymes by exploiting metabolic pathways common to both mammals and yeast. Yeasts and other fungi, because of their characteristics, may survive and perform their desired function in a real-world environment better than bacteria. The goal of this research is to determine if genes for chemical degradative characteristics of certain mammals (e.g., rats) can be transferred to yeast. If successful, the modified yeast could be grown in large quantities at low cost with fairly simple technology. Yeast with the adopted mammalian trait of degrading chlorinated compounds could then be used to detoxify contaminated soil sites. (Applicability to severely contaminated sites with multiple components is unknown.) This decontamination process would probably be a slow one, but inexpensive, and would not require that the soil be excavated.

Recent publications support the premise that positive results from this research can be obtained. Investigations by H. Poiger indicate the presence of several polar biotransformation products in the bile of dogs that were given 2,3,7,8-TCDD. The structure of five phenolic metabolites was determined and the metabolic breakdown scheme for 2,3,7,8-TCDD in the dogs was proposed. Another paper by J.D. Brooker indicates that it is possible to isolate m-RNA, which is acetamide in this case, from chicks. In view of these two papers and the genetic similarity between dogs and yeast (both are eucaryotes), it can be reasoned that there is a good possibility of inserting the dog's genetic ability to degrade dioxins, etc., into yeasts.

IT Enviroscience Treatise on Biological Degradation: IT Enviroscience is conducting a technical evaluation of available data concerning the biological degradation of chlorinated aromatic compounds. When completed, this study should be useful in defining further studies that may utilize genetic engineering techniques.

Other Work: One additional study identified by microbiologists is the definition of bioavailable TCDD in a soil system. It is apparent from the Poiger and Schlatter work, Hutter work, and analysis of historic samples, that a time-related complexing phenomena exists between TCDD and soil. Hashimoto has identified TCDD in this complex as being non-degradable in soil and sediment incubation trials. For TCDD degradation in the biosphere, definition of bioavailability is necessary to assess both the potential of biological degradation and risk of TCDD toxicity to living organisms.

References: 22, 25, 37, 86, 117, 124, 125, 131, 134, 135, 146, 147, 175, 176, 180, 184, 200, 201, 270, 281, 282, 283

DRAFT

DRAFT

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Solvent Extraction (Leaching)

DESCRIPTION OF TECHNOLOGY: Solvent extraction of dioxin from soil and sediment is achieved by intimately contacting adequately processed soil and sediment with a solvent that will preferentially remove dioxin from soil to a desired level in a specified contacting time. A multiple batch contacting process or a continuous countercurrent process are needed when a single contacting stage does not accomplish the desired level of removal.

STATE OF DEVELOPMENT: Solvent extraction of materials from soil is a well-developed industrial process. As examples, solvent extraction of materials from soil is commonly used in the mining industry and has been demonstrated for extraction of bitumen from tar sands. Experience with tar sand has shown that some soil types allow for easier extraction than do others. Sandy soil allows easier extraction and solvent recovery than does diatomaceous earth.

To date, no pilot or large-scale processes using solvents to extract TCDD from soil and sediment have been used. However, TCDD was extracted from contaminated sludge in distillation bottoms with hexane in a full-scale solvent extraction process at the Syntex Agribusiness facility in Verona, Missouri. The dioxin concentration in the sludge was reduced from 343 ppm to 0.1-0.5 ppm.

Solvent extraction is used in analytical procedures for measuring TCDD in soil and sediment. Extraction of TCDD from soil samples has had mixed results in laboratory analytical work. Laboratory extractions are generally done on pulverized soil samples. For some samples, quantitative extractions have been accomplished by shaking the samples with a relatively common solvent (petroleum ether, hexane, methanol) at a 3:1 ratio of sample volume to solvent. TCDD was extracted from 90-gram samples of soil from Seveso at 97.5 percent efficiency by treating the samples twice with 300 ml of methanol. Acetone-hexane mixtures were used to extract TCDD from soil and to remove it from contaminated surfaces at Seveso. However, some laboratory samples have required much more rigorous extraction, procedures to accomplish quantitative extraction. One such procedure that has been required is a Soxhlet extraction for up to 3 days with pure methylene chloride.

Research has shown that TCDD binds to soil and becomes increasingly difficult to extract with time. Therefore, using a solvent to extract dioxin from soil that has been contaminated for several years may be difficult. Since each soil

matrix is unique, TCDD-contaminated soil samples must be tested to determine the required solvent and level of processing necessary to achieve desired residual TCDD levels.

Acurex Corporation and Chemical Waste Management, Inc., are two companies that are known to be independently developing solvent extraction processes on a laboratory scale. Both companies have obtained TCDD-contaminated soil samples from Missouri sites for testing.

FUTURE RESEARCH NEEDS: The theoretical basis for solvent extraction of dioxin from soil and sediment has been established by research on analytical procedures for dioxin analysis. Further laboratory work is necessary to establish the applicability of a specific solvent process to specific contaminated soil samples.

Besides laboratory development, the chosen solvent extraction process must be tested for effectiveness on a pilot-plant scale. Detailed studies must be performed to assess process risks and benefits and to determine environmental and economic impacts of implementing a full-scale process. Finally, detailed design and development of a full-scale solvent extraction process that would be effective in treating all desired contaminated materials would need to take place.

There is considerable evidence that dioxin in a solvent phase poses a greater potential hazard than dioxin in soil or sediment. This would require further analysis to properly assess the risks associated with solvent extraction.

References: 47, 111, 153, 154, 193, 237

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Fixation of Soil

DESCRIPTION OF TECHNOLOGY: The fixation of organic wastes in soils has been attempted in a multitude of ways in the past. The immobilization of dioxin-contaminated soil and sediment may be achieved by one or a combination of these processes. The methods may be grouped into three categories: inorganic, organic treatment, and encapsulation.

Inorganic Fixation: The common inorganic fixation techniques use portland cement, pozzolanic (flyash) materials with or without lime or cement, and sorbent clays. In the process involving cement and pozzolanic materials, the soil and sediment is mixed with the cementing agent and allowed to set up. The advantages of these processes are plentiful raw materials, low cost, the fact that the organic wastes are adsorbed or mechanically trapped (although both may allow leaching of some wastes), and proven technology. Disadvantages include porosity of cemented soil and the increased volume of the original waste by factors of 2 to 3-1/2 times the original waste volume. This expansion results in increased mixing, packaging, transportation, and disposal site expense.

The process using sorbent clays relies on mixing the finely-ground powdered clay with the contaminated materials and then isolating them mechanically. The clays, with the addition of water, become very cohesive and relatively impermeable, which reduces the possibility of particle loss or leaching. The advantages are low cost, availability of materials, and ease of mixing. On the other hand, the process does not completely eliminate leaching, the mixture has to be contained to prevent exposure to contaminated materials that may be on the surface, and the volume of material expands substantially.

Another method of immobilizing the sediment and soils is to mix 10 to 15 percent portland cement with the soils. After proper mixing, compacting, and curing, a hard durable solid forms. The process of making soil-cement involves soil conditioning operations; therefore dust control measures are required. The soil-cement is essentially insoluble, which immobilizes the contaminated soil. However, environmental factors such as wetting, drying, freezing, and thawing may in the long-term cause deterioration of the soil-cement treatment.

Organic Fixation: Stabilization chemicals are available that, in general, react with moisture in the soil and sediment or an aqueous catalyst, to form a hydrophobic cross-

linked polymer-based gel. The semi-solid gel coats and binds the soil particles together. The chemical and water (or catalyst) mixture is sprayed on cultivated or loosened soil to react with the upper 3 to 4 inches of soil. The resulting gel-soil mixture then becomes a barrier to water infiltration. Such chemical grouts are not hazardous when basic safety and handling precautions are observed.

Chemical grouts cost more than other stabilization methods. Some questions remain regarding the depth of penetration, application rates, and overall effectiveness that can be achieved for Love Canal creeks. A laboratory or field testing program is needed before this treatment can be used. The gels are subject to UV degradation and, thus, the soil needs to be covered by an opaque paint or soil cover.

Some of the common chemical grouts worthy of further study, testing, and evaluation are described below by trade or chemical name:

- o American Cyanamid Acrylonide is a mixture of two monomers that, when catalyzed in an aqueous solution, form a stiff gel. This polymerization-cross linking action binds water molecules to the gel to form a substantially impermeable material.
- o Deneef Co. Polyurethane. This low viscosity, one-component liquid reacts with water to form a polyurethane foam. The closed cell foam has a free expansion volume 15 times the original volume. It may be used with an additive to delay reaction time up to 10 minutes to allow the material to soak into the soil.
- o Pene Grout of North America has a urea-based polymer, which reacts with water to form a hydrophobic non-hydrated gel material that binds the soil particles together.
- o Terra Firma by Intrusion Prepakt, Inc., uses a chrome-lignin compound to form a dark brown non-reversible gel, which is insoluble in water and organic solvents and is substantially impermeable to water.

A polyphenol grout is also available in which a phenolic polymer combines with a catalyst and inhibitors to form a gel.

Other special grouts are used for particular applications. Examples are sodium silicate-calcium chloride or sodium silicate-sodium aluminate reactions, which have transformed

soft sands into a material that has strength and permeability properties similar to weak sandstone. Epoxy resins, silicone rubbers, lime, and specially formulated bituminous compounds have also been mentioned as soil stabilization materials.

The advantages some of the chemical grouts offer are that they are easy to mix, they penetrate soil much like water (since they have a viscosity similar to water), they can be applied by spraying, and they are generally non-toxic when handled properly. Also, most of these grouts seek and react with water in the soil or groundwater, they form irreversible compounds of indefinite life (under proper conditions), they do not substantially increase the volume of the treated soil, and their use is proven. On the negative side, grouts are more expensive than other stabilization methods, they are sensitive to freeze-thaw and wet-dry conditions, and some grouts deteriorate under ultraviolet light.

The chemical soil stabilization methods are not without merit and deserve further investigation. They may be best used in conjunction with other containment methods. Some other containment methods are discussed below.

Asphalt Membrane: An asphalt industrial membrane also provides an impervious and totally confining cap. The two-component cover consists of fabric reinforcement sheets laid on the soil and then sprayed with a special asphalt emulsion to form a 50-mil-thick membrane. This system can be formed in place, which only requires smoothing the soil surface. The asphalt material is not mixed with the soil to immobilize the soil particles; it only bonds to the fabric to confine the soil beneath the cover.

Polymeric Membrane: Thin polymeric membranes of generally inert materials can also be used to cover contaminated sites. The membrane is installed in large panels and joined in the field to provide a leak-proof cover. Materials such as reinforced chlorosulfonated polyethylene and high-density polyethylene have very good resistance to ultraviolet light. The membranes have to be covered with at least 1 foot of fine-grained soil for protection from mechanical damage and extreme environmental conditions.

Encapsulation: The technology for encapsulation of toxic wastes such as dioxin is borrowed from other hazardous waste containment processes that completely coat or seal the waste material. Before the coating or sealing process, the wastes are commonly prepared by some kind of fixation or adsorption scheme as described before. In some methods the resulting solid material is coated with a 1/4-inch-thick polyethylene jacket. Steel and plastic drums are also used and sealed to

secure the materials. Heavy, securely sealed polymeric or rubber bags containing solidified waste are another possibility.

The encapsulation technique offers the advantages that no material is lost or leached out as long as the encapsulation remains intact. The materials can be handled, transported, inventoried, and incorporated into a disposal site easily. The disadvantages are that these processes are very expensive both from a capital and operations standpoint, skilled labor is required to operate the equipment, and the effective bulk volume of the waste is greatly increased due to the pretreatment fixation and usually irregularly-shaped encapsulation.

FUTURE RESEARCH OR DEVELOPMENT: Research into fixation methods should be continued, specifically on untried methods and the feasibility of using fixation on dioxin-contaminated sediment. For untried methods, laboratory and/or field testing may be required to establish data regarding application, permanency, side effects, etc. Research into the feasibility of using fixation for dioxin-contaminated soil and sediment is needed, particularly in the area of insitu treatment or neutralization of the waste.

One new product on the marketplace by RADECCA. Inc., of Austin, Texas, claims to absorb and stabilize organics. The material acts to sequester organics and could be useful in stopping the organics from interfering with pozzolanic, cementitious reactions involved in the traditional solidification methods. The more insoluble the organic compound is, the better the product is said to work. This product may be incorporated in clay cutoff walls to "filter out" any organics carried into the cutoff wall by groundwater, for example. Research on this and other similar products needs to be conducted and their use evaluated. They may be used alone or in conjunction with the soil fixation methods discussed in this section.

Other potential fixation methods may use bonding agents such as latex and asphalt emulsions and byproduct materials like lignosulfonates. These are generally untried, but worthy of consideration.

References: 75, 130, 143, 157, 164, 174, 216, 240, 254, 262

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: High-Temperature Fluid-Wall (HTFW) Reactors

DESCRIPTION OF TECHNOLOGY: In this process, waste in a central porous cylinder is heated by radiation from surrounding electrodes to 3,000°F to 4,000°F. The central cylinder is made of porous carbon or ceramic material that is transparent to the infrared radiation from the electrodes and is protected from thermal or chemical destruction through contact with the heated waste by a fluid film of inert gas that is drawn through the inside of the cylinder. This process results in a rapid and complete waste heating allowing for a high degree of combustion completeness. A high degree of process control is possible since the radiation source is electricity.

STATE OF DEVELOPMENT: The Thagard Research Corporation has achieved 99.999 percent destruction of PCB's in soil (one percent by weight) in a 1/4-lb/min laboratory-scale reactor. Thagard has claimed the same destruction efficiency for burning hexachlorobenzene in tests of a 10-ton-per-day reactor unit. EPA certification test results on PCB-contaminated soil destruction in a 15-lb/min HTFW reactor owned by the J. M. Huber Corporation have shown destruction efficiencies of greater than 99.9999 percent. Huber has also processed 200 pounds of soil at Times Beach, Missouri. The soil started with a dioxin concentrations of 80 ppb. After treatment the soil had a dioxin concentration of less than 0.1 ppb. Southern California Edison, Inc., has reportedly been considering the Thagard process for destruction of PCB-contaminated soil. A mobile Thagard reactor project has reportedly been considered with possible support indicated from a Miami investment firm.

OTHER EQUIPMENT: Size reduction equipment is needed for particle size control to assure evenly distributed heat transfer.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Specific areas that need to be developed are durable scaled-up central cores and the determination of necessary air pollution control equipment.

References: 65, 114, 115, 247

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Supercritical Water Oxidation

DESCRIPTION OF TECHNOLOGY: Supercritical water oxidation uses air or oxygen in water above its critical temperature and pressure (374°C and 218 atm, respectively) to destroy organics. Oxygen and hydrocarbons are almost completely miscible with water and salts precipitate out. The waste is slurried, pressurized, and then educted into the supercritical water reactor. A base is added to the system so that anions present can be reacted to salts. Salt, water, carbon dioxide, and traces of organic feed exit the reactor.

STATE OF DEVELOPMENT: Modar Incorporated has laboratory tested a supercritical water process on PCB's and other chlorinated hydrocarbon compounds. Destruction efficiencies of greater than 99.99 percent were recorded.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Supercritical water oxidation needs to be laboratory tested on TCDD-contaminated materials and the feasibility for further development should then be evaluated.

References: 19, 57, 65, 247

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Microwave Plasma Destruction

DESCRIPTION OF TECHNOLOGY: Organic compounds are broken down into smaller molecules when combined with partially ionized gas produced by microwave-induced electron reactions.

STATE OF DEVELOPMENT: It appears that this technology has recently been tested specifically for breakdown of dioxin with some success, although these results have apparently not been published. Microwave plasma destruction has been tested on a bench scale using other chlorinated organics, including PCB's. Organic hazardous wastes have been treated as pure liquids, as slurries, or solutions in methanol or water, or as compressed solids. Test units so far have processed up to 7 pounds per hour of hazardous organic material.

Lockheed Palo Alto Research Laboratory achieved over 99 percent destruction of a variety of toxic organics, including two commercial PCB's, Malathion and Kepone. The feed rates for these tests ranged from 0.18 to 3.0 kilograms per hour. Treatment of PCB's produced carbon monoxide, carbon dioxide, water, phosgene, and chlorine oxides. Some of these products are highly toxic and require subsequent treatment.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: This technology needs to be laboratory tested and documented for dioxin-contaminated materials of interest. This is a bench-scale technology and needs development through pilot and large-scale tests to determine economic feasibility and technical success in treating large volumes of hazardous organic materials.

Westinghouse Electric Corporation is now operating a 20,000-kW test facility featuring a plasma torch system. Toxic chemical waste destruction at the facility is currently under development.

References: 14, 17, 87, 180, 247, 272, 275

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Plasma Arc Pyrolysis

DESCRIPTION OF TECHNOLOGY: The Plasma Arc process uses energy from ionized gas molecules, created by an electrical current discharge through a vortex of low-pressure gas, to destroy organic molecules. Temperatures equivalent to 50,000°K are achieved in the plasma, and rapid decomposition follows exposure to waste materials. The primary products from TCDD destruction would likely be CO, CO₂, HCl, H₂, and H₂O. Gas volumes supplied to the reactor are on the order of 5 percent of the gas volumes required by conventional incineration. Scrubbers are needed for exit gases from processing halogenated wastes.

STATE OF DEVELOPMENT: Preliminary laboratory scale tests have shown PCB destruction from liquid wastes in excess of 99 percent. Canadian government laboratory scale testing has demonstrated destruction efficiencies in excess of 99.9999 percent for pure transformer oil fluids (Aroclor and Askarel) containing up to 58 percent chlorine by weight. No tests are reported on TCDD.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Laboratory testing on TCDD's is needed. Determination of commercial feasibility and scale of operation for this process is needed.

References: 65, 247

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Gamma Ray Radiolysis

DESCRIPTION OF TECHNOLOGY: Toxic organics are exposed to radiation from a gamma ray source after dissolving in a suitable organic solvent.

STATE OF DEVELOPMENT: Gamma ray radiolysis has been laboratory tested on several toxic organics, including TCDD. No specific results have been reported for treatment of TCDD in soil by this technique. A 97-percent TCDD reduction was observed after 30 hours of gamma ray irradiation at a million rads per hour for a 100-ppb solution of TCDD in ethanol. All samples tested showed the presence of the less chlorinated dioxins tri-CDD (three chlorines) and DCDD (two chlorines).

Other research using ionizing gamma irradiation on other toxic chlorinated organics (including pentachlorophenol 2,4,5-T, and PCB's) has led to the conclusion that this method of destruction is inefficient and prohibitively costly for large-scale use.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Gamma ray radiolysis has yet to be effectively demonstrated on a laboratory scale in a manner that warrants development on a larger scale.

References: 83, 180, 247

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Molten Salt Combustion

DESCRIPTION OF TECHNOLOGY: Chlorinated hydrocarbon wastes are injected in a continuous feed below the surface of a 800°C to 1000°C molten salt bath, which contains a mixture of sodium or potassium carbonate and 10 percent sodium sulfate by weight. Rapid heating and thorough mixing of the waste can be achieved in this fluid heat-conducting reaction medium. The chlorinated hydrocarbons oxidize in the molten salt to CO₂, water, and sodium chloride. Supplemental heat from combusted combustible materials is required when low-Btu value wastes are destroyed. Materials generated during the combustion process can be retained and the spent molten salt can be either regenerated or landfilled. Solids fed to the reactor must undergo size reduction to at least 1/4- to 1/8-inch pieces. A particulate baghouse is necessary for the off-gas. Ash and any metal, phosphorous, halogen, or arsenic salts build up in the melt and must be removed.

STATE OF DEVELOPMENT: Molten salt combustion has been tested by Rockwell International on a 200-lb/hr bench scale unit for decomposition of chlorinated hydrocarbons (including 2,4-D, chlordane, chloroform, PCB's, trichloroethane). More than 99.9999 percent decomposition has been achieved. It appears that this technology has not been tested specifically on dioxin.

Two firms are actively marketing molten salt equipment for hazardous waste destruction. However, no units are under construction or are in commercial use at the present time.

A full-scale molten salt combustion process might be difficult to develop due to the requirements for materials of construction. The spent molten salt also presents disposal and materials handling problems. The molten salt process has been identified as "totally suitable" for potential destruction of "contaminated soils from spill and old dump site cleanup," having the characteristics of "water and mineral matter mixed with various hazardous organic compounds."

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Needs to be laboratory tested for various dioxin-contaminated materials.

References: 87, 180, 247, 275

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Carbon Adsorption

DESCRIPTION OF TECHNOLOGY: Solutions containing materials to be absorbed are passed through beds of activated carbon that provide sufficient solution residence time to achieve the desired removal.

STATE OF DEVELOPMENT: Activated carbon has been shown to selectively adsorb TCDD from solvents. Coconut charcoal has been used in full-scale operations to adsorb dioxin from the herbicide 2,4,5-T.

Tests have shown that, after adsorption of TCDD, the carbon cannot be regenerated. Several sites around the world are currently storing dioxin-contaminated carbon. Incineration of the TCDD-contaminated carbon has been proposed for the final disposal of TCDD. However, experience with other organics has shown that, to ensure complete combustion, activated carbon requires processing before incineration. This additional processing introduces the risk of handling highly contaminated materials.

Other technologies that have been proposed for regeneration of TCDD-contaminated carbon includes supercritical fluids and microwave radiation.

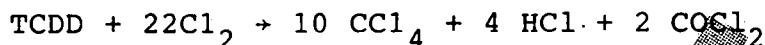
FUTURE RESEARCH OR DEVELOPMENT NEEDS: A data base needs to be developed by laboratory research to provide quantitative information on carbon adsorption loadings and removal efficiencies for various dioxin-contaminated solvents.

References: 180, 275

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Chemical Degradation--Chlorination

DESCRIPTION OF TECHNOLOGY: Chlorolysis (extensive chlorination) is a process where gaseous feed materials are reacted with chlorine usually at pressures of 200 to 700 atmospheres and temperatures up to 800°C, producing carbon tetrachloride and associated products. The reaction with TCDD is:



STATE OF DEVELOPMENT: Under EPA contracts, the Diamond Shamrock Corporation and the Hoechst-Uhde Corporation conducted laboratory studies of chlorolysis of Herbicide Orange to form carbon tetrachloride, phosgene, and hydrogen chloride gas. The concentration of TCDD in the Herbicide Orange used by Diamond Shamrock was 14 ppm. The Herbicide Orange was diluted 1:1 with CCl_4 and chlorinated at 600-800°C and 150 psig. The TCDD in the product was less than the detectable limit of 10 ppt. The Herbicide Orange used in the Hoechst-Uhde study contained 14-18 ppm TCDD. TCDD was not measurable to a detection limit of 1 ppb in the carbon tetrachloride product.

To our knowledge, no further studies on dioxin treatment by this method have been done and no experience with soil-bound dioxin exists.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Since so little is known about this process, extensive laboratory development is required to determine its applicability to soil-bound dioxin.

References: 61, 138, 180

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Chemical Degradation--Catalytic Oxidation

DESCRIPTION OF TECHNOLOGY: TCDD is dissolved in a non-nucleophilic solvent and reacted with ruthenium tetroxide (RuO_4). Examples of solvents include chloroform, nitromethane, and carbon tetrachloride. In the reaction, ruthenium tetroxide oxidizes the TCDD and ruthenium dioxide is formed. Ruthenium dioxide can be converted back to ruthenium tetroxide by combining it with hypochlorite. Therefore, only catalytic quantities of RuO_4 are required for the oxidation of TCDD. In lab experiments, a ratio of about 30:1 RuO_4 to TCDD was used.

STATE OF DEVELOPMENT: Catalytic oxidation of TCDD has been accomplished in the laboratory. The TCDD half-life was measured at 560 minutes at 30°C and 15 minutes at 70°C. RuO_4 itself is a toxic compound and is not commonly available in commercial quantities. Only laboratory experiments have been conducted, and no pilot scale or soil-bound dioxin experiments have been done. The high cost and high toxicity of ruthenium tetroxide make this process infeasible for large-scale applications.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Future laboratory development required to determine the feasibility of this process.

References: 12, 13, 61

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Chemical Degradation--Chloriodides

DESCRIPTION OF TECHNOLOGY: Two types of chloriodides have shown promising results for degrading TCDD. They are: 1) alkyldimethyl benzyl ammonium (benzalkonium) chloriodide, and 2) 1-hexadecylpyridinium (cetylpyridinium) chloriodide. The chloriodides are dissolved in a micellar solution due to low water solubility of these substances. TCDD contaminated materials are then contacted with the chloriodides in micellar solution at ambient temperatures. TCDD degradation occurs by the cleavage of ether bonds. Chlorinated phenolics are expected as reaction byproducts.

STATE OF DEVELOPMENT: Moderate levels of TCDD destruction from contaminated residues have been experienced in the laboratory. TCDD in benzene was vacuum-evaporated and the residue treated with benzalkonium chloriodide; 71 percent of the TCDD was decomposed. Using cetylpyridinium chloriodide, a 92-percent decomposition level was achieved. Lab experiments were also conducted on a soil sample from Seveso, Italy, using a chloriodide micellar solution. The TCDD concentration dropped from 23 $\mu\text{g}/100\text{g}$ of soil to 11.0 $\mu\text{g}/100\text{g}$ in 24 hours. Cationic surfactants worked best on the soil sample.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Further laboratory development is required to determine feasibility of this process.

References: 29, 61, 180

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Chemical Degradation--Dechlorination

DESCRIPTION OF TECHNOLOGY: Dechlorination of TCDD involves the use of chemical reagents or catalysts to remove chlorine atoms from the TCDD structure, thereby reducing its toxicity and making it amenable to other forms of treatment or disposal. Dechlorination processes have been tested and considered for use in detoxifying TCDD by Vertac, Acurex, and Wright State University. These processes have used alkali metal reagents at both ambient and elevated temperatures to remove the chlorine atoms from TCDD. The Vertac process achieves dechlorination of TCDD by the addition of anhydrous alkali metal salts of polyhydroxy alcohols at atmospheric pressure. In an alternate Vertac process, dechlorination is accomplished by reacting TCDD with an alcohol and a water solution of an alkali metal hydroxide. The Acurex process uses a proprietary sodium reagent in a mobile batch treatment process to dechlorinate TCDD. Research studies at Wright State University have used sodium polyethylene glycol reagents (NaPEG) in batch processes at ambient temperatures to dechlorinate TCDD.

Several companies have also developed chemical dechlorination processes for PCB's that are viable candidates for treating TCDD-contaminated materials, based on similarities in process and chemical reactions between PCB's and TCDD. These processes have not been tested on TCDD.

Research work on other polychlorinated compounds, including DDT's and several other commercial pesticides, has shown that a catalyst can be used at room temperature to cause a rapid reduction reaction where chlorines on the organic compound are replaced with hydrogen atoms. Research has shown that this process does not completely dechlorinate most organic chemicals. The catalyst most frequently used in research work is nickel borohydride, prepared by mixing sodium borohydride with nickel chloride in alcohol. No catalytic dechlorination testing has reportedly been done on TCDD.

STATE OF DEVELOPMENT: Degradation of TCDD by dechlorination has been tested to a limited extent in the laboratory. Greater than 99.95 percent TCDD degradation was observed from a 2,4,5-TCP still bottoms sample contaminated with 250 ppm TCDD in laboratory testing of chemical dechlorination at Vertac. The Acurex mobile batch sodium reagent dechlorination process has been developed commercially for PCB's. Test data from the Acurex PCB dechlorination process showed a reduced dioxin concentration in transformer oil from 310 parts per trillion (ppt) to 40 ±20 ppt.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Currently undergoing further laboratory development at Wright State University and Acurex Corporation. Extensive pilot studies needed to determine: 1) technical feasibility, 2) economics, 3) process safety, 4) environmental impact, and 5) specific process requirements for soil treatment.

References: 60, 61, 65, 112, 180, 247, 275

DRAFT

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Wet Air Oxidation

DESCRIPTION OF TECHNOLOGY: Wet air oxidation is a physical/chemical treatment process for the destruction of organic compounds in water under high temperatures and pressures. Under these conditions, organics are oxidized to alcohols, aldehydes, acids, and ultimately to carbon dioxide and water by injecting oxygen into the process. Typical operating temperatures and pressures are 150-350°C and 500-2500 psig.

The process is best suited to aqueous solutions or slurries where organic matter constitutes between 2 percent and 20 percent. One manufacturer states that, at 5 percent organic content, the process is autothermal. At lower organic concentrations, large amounts of energy may be needed to maintain the process. In the process, oxygen is injected into a high-temperature, high-pressure reactor. Mixing within the reactor improves the oxidation of the organics. Waste materials in which oxygen transfer to the organics is difficult and may require higher than normal operating pressures to achieve adequate destruction efficiencies.

When destroying certain wastes in this type of system, the environment within the reactor and other parts of the system is highly corrosive. Required construction materials for treating chlorinated hydrocarbons may include nickel, titanium, hastelloy, and zirconium.

STATE OF DEVELOPMENT: Wet air oxidation technology has been commercially applied to several waste types, including municipal wastewater, organic sludges, pulps, and various other chemical wastes. However, its application to TCDD has been only on a test basis. Although an extensive literature search did not indicate that treating TCDD in soil with a wet air oxidation system has ever been attempted, several manufacturers have indicated such a system can be developed. WetCom Engineering Limited is currently studying an application for wet air oxidation of large quantities of lake sediment.

The only data available regarding the destruction of TCDD is from tests conducted on a pilot model of a variation on the wet air oxidation process by IT Enviroscience. A 99-percent reduction in TCDD was observed in lab tests of catalyzed wet oxidation conducted at 200°C and with a 4-hour reaction time. The oxidation reaction was catalyzed by a bromide-nitrate solution. While a substantial amount of lab experience has been published, no industrial or soil decontamination work is mentioned. At one time, IT Enviroscience

planned to construct a pilot plant for this process, but the pilot plant was never built.

Before TCDD in soil can be treated by wet air oxidation, the soil must be pulverized and mixed into a slurry. The slurry then enters the system through a high-pressure pump. Careful control of soil particle size is, therefore, important.

Because of the low organic content of soil, thermal energy requirements of up to 1000 Btu/gallon are required, as indicated by Zimpro Inc. and WetCom Engineering Limited. Zimpro has indicated that a maximum deficit of 500 Btu/gallon of wastewater is required when the organic content of wastewater is very low (1 G/l COD). Therefore, such a soil treatment process will not be autogenous.

WetCom Laboratory tested their wet air oxidation process for PCB destruction. Long batch times and temperatures exceeding 250°C were used, but destruction efficiencies were only in the 50- to 70-percent range. PCB destruction efficiencies of >99 percent were achieved in pilot plant testing of the IT Enviroscience catalyzed wet air oxidation process.

FUTURE RESEARCH OR DEVELOPMENT NEEDS: Research in the application of wet air oxidation to soils and dioxins is needed. The methods of reactor agitation to facilitate oxidation of organics which are bound to soil need to be investigated.

References: 65, 179, 247, 275

DIOXIN TECHNOLOGY SUMMARY

SUBJECT: Ultraviolet Ozonation

DESCRIPTION OF TECHNOLOGY: Ultraviolet ozonation is a combination of breaking chemical bonds with ultraviolet light and oxidation of the activated organic compounds with ozone. It is achieved by bringing ozone into contact with the liquid organic waste in the presence of ultraviolet radiation of a specified wavelength range and intensity.

STATE OF DEVELOPMENT: One ppb of TCDD was completely degraded with an ultraviolet/ozone system by researchers at California Analytical Laboratories and the Carborundum Company. Other test results indicate that products of ultraviolet ozonation are more biodegradable than initial contaminants such as 2,4,5-T, PCB, and TCDD. Ultraviolet ozonation may, therefore, be considered as a pretreatment alternative for soil disposal.

In fresh field soils, low concentrations (one ppm in H₂O) of 2,4,5-T and PCP were rapidly degraded when first irradiated for one hour in the presence of O₂. TCDD and PCB were degraded more slowly under the same conditions.

Soil degradation of TCDD in Aroclor 1242 was also monitored for nonirradiated samples and samples irradiated in the presence of N₂. Total degradation was less than 2,4,5-T and PCP for the same conditions. After 28 days, the conversion to CO₂ was 1.5 percent nonirradiated, 3.1 percent N₂, 12.7 percent O₂.

Large-scale ultraviolet ozonation has been conducted by Pure Water Systems, Inc., in an Ultra-Violet Purifier. Results have not been published.

FURTHER RESEARCH OR DEVELOPMENT NEEDS: Ultraviolet ozonation of dioxin in solvent has been demonstrated, but further scale-up is needed. Further development is needed to determine what level of dioxin concentration reduction is feasible. Research and development is also needed to determine if ultraviolet ozonation of dioxin in soil is feasible.

References: 76, 127

WDR102/003

Section 11

BIBLIOGRAPHY

1. Ackerman, D.G. et al. 1981. Guidelines for the disposal of PCBs (polychlorinated biphenyls) and PCB items by thermal destruction. Final report Oct. 1979-Apr. 1980. Prepared by TRW, Inc., Redondo Beach, CA. EPA report no. EPA-600/2-81-022. NTIS report no. PB81-182339. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
2. Acurex Corp. 1980. Test incineration of electrical capacitors containing PCBs. Disposal of polychlorinated biphenyls (PCBs) and PCB-contaminated materials, Vol. 4. Prepared for Electric Power Research Institute, Palo Alto, CA. Report no. EPRI FP-1207. Mountain View, CA: Acurex Corp., Energy and Environmental Division.
3. Administrative order requires cleanup of dioxin wastes at Vertac, Inc., site. 1979. Environment Reporter (June 22):298. New York: Springer-Verlag, publication no. 0013-9211/79.
4. Ahling, Bengt, et al. 1977. Formation of polychlorinated dibenzo-p-dioxins and dibenzofurans during combustion of a 2,4,5-T formulation. Chemosphere 8:461-468. London: Pergamon Press.
5. Ahling, Bengt, and Leif Johansson. 1977. Combustion experiments using pentachlorophenol on a pilot scale and full-scale. Chemosphere, 7:425-436. Great Britain: Pergamon Press Ltd.
6. Ahling, Bengt, and Anne/Lindskog. 1978. Thermal destruction of PCB and hexachlorobenzene. The Science of the Total Environment 10(1978):51-59.
7. Ahling, Bengt. 1979. Destruction of chlorinated hydrocarbons in a cement kiln. Environmental Science & Technology 13(11):1377-1379.
8. Akermarck, Bjorn. n.d. Photodechlorination of haloaromatic compounds. In Dioxin: Toxicological and chemical aspects, ed. P. Cattabeni, A. Cavallaro, and G. Galli, 201. New York: SP Medical & Scientific Books. Spectrum Publications, Inc.
9. Alert on dioxin: Special dioxin section. n.d. Alert Newsletter. University City, Missouri: Coalition for the Environment.
10. Arnold, E.L., A.L. Young, and A.M. Wachinski. n.d. Three years of field studies on the soil persistence and movement of 2,4-D, 2,4,5-T, and TCDD. Colorado: Department of Chemistry and Physiology, U.S. Air Force Academy.
11. Arthur D. Little, Inc. 1981. Study on state-of-the-art of dioxin from combustion sources. ASME publication no. HQ180. New York: Research Committee on Industrial and Municipal Wastes, The American Society of Mechanical Engineers.
12. Ayres, D.C. 1981. Destruction of polychlorodibenzo-p-dioxins. Nature 290(March):323-324.
13. Ayres, D.C. 1981. Ruthenium tetroxide destroys dioxin: The oxidative control of aromatic pollutants. Platinum Met. Rev. 25(4):160.

14. Bailin, L.J., and B.I. Hertzler. 1977. Development of microwave plasma detoxification process for hazardous wastes. Phase I. EPA report no. 600/2-77-030. Cincinnati, Ohio: Municipal Environmental Research Laboratory, Office of Research and Development, U.S. EPA.
15. Bailin, L.J. 1978. Detoxification of navy red dye by microwave plasma. Summary report. EPA report no. 600/2-78-081. Cincinnati, Ohio: Municipal Environmental Research Laboratory, Office of Research and Development, U.S. EPA.
16. Bailin, L.J., and B.I. Hertzler. 1978. Detoxification of pesticides and hazardous wastes by the microwave plasma process. In Disposal and decontamination of pesticides, ed. M.V. Kerred, 48-72. American Chemical Society Symposium Series 73. Washington, D.C.: ACS.
17. Bailin, L.J. 1978. Microwave plasma detoxification process for hazardous wastes. Phase II: Systems application evaluation. EPA report no. 600/2-78-050. Cincinnati, Ohio: Municipal Environmental Research Laboratory, Office of Research and Development, U.S. EPA.
18. Bartleson, F.D., Jr., D.D. Harrison, and J.D. Morgan. 1975. Field studies of wildlife exposed to TCDD contaminated soils. Prepared for the Air Force Armament Lab, Armament Development and Test Center, Eglin AFB, Florida. Air Force report no. AFATL-TR-75-49. NTIS no. ADB 009 710. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
19. Basta, Nicholas. 1982. Firms avidly seek new hazardous waste treatment routes. Hazardous wastes, Part 2. Chemical Engineering (September 6):53-57.
20. Bastian, R.E. and W.R. Seeman. 1978. The design and operation of a chemical waste incinerator for the Eastman Kodak Company. Proceedings, National Waste Processing Conference. Vol. 8.
21. Beale, Mary G., et al. 1977. Long-term effects of dioxin exposure. Letter to the editor. The Lancet (April 2):748.
22. Beck, S.P., W.H. Macklin II, and E.L. Arnold. n.d. Degradation of chlorophenoxy-alkanoic acid herbicides by purified cultures of soil microorganisms. Colorado: Department of Chemistry and Physiology, U.S. Air Force Academy.
23. Bertoni, G., et al. 1978. Gas chromatographic determination of 2,3,7,8-tetrachlorodibenzodioxin in the experimental decontamination of Seveso soil by ultraviolet radiation. Analytical Chemistry 50(6):732-735.
24. Blowing smoke in Midland. 1983. Chemical Week (April 13):3.
25. Bollen, W.B. and L.A. Norris. 1979. Influence of 2,3,7,8-tetrachlorodibenzo-p-dioxin on respiration in a forest floor and soil. Bulletin of Environmental Contamination and Toxicology 22:648-652.
26. Bolton, Lois. 1978. Seveso dioxin: No solution in sight. Chemical Engineering 85(22):78.

27. Bonner, T., et al. 1981. Hazardous waste incineration engineering. Pollution Technology Review no. 88. Park Ridge, New Jersey: Noyes Data Corporation.
28. Botre, Claudio, Adriana Memoli, and Franco Alhaïque. 1978. TCDD solubilization and photo-decomposition in aqueous solutions. Environmental Science & Technology 12(3):335-6.
29. Botre, Claudio, Adriana Memoli, and Franco Alhaïque. 1979. On the degradation of 2,3,7,8-tetrachlorodibenzopara-dioxin (TCDD) by means of a new class of chloriodides. Environmental Science & Technology 13(February):228-231.
30. Bovey, Rodney W., and Alvin L. Young. 1980. The science of 2,4,5-T and associated phenoxy herbicides. A volume in Environmental Science and Technology, a Wiley-Interscience publication, ed. R.L. Metcalf and W. Stumm. New York: John Wiley & Sons.
31. British plant shut because of health risk. 1978. Chemical Engineering (September 11):107.
32. Brown, Michael H. 1979. Love Canal, U.S.A. The New York Times Magazine (Jan. 21).
33. Brown, Michael. 1979, 1980. Laying waste: The poisoning of America by toxic chemicals. New York: Pantheon Books.
34. Bumb, R.R., et al. 1980. Trace chemistries of fire: a source of chlorinated dioxins. Science 210(24 October):385-390.
35. Burger, Edward J., Jr. 1973. Summary: Conference on dibenzodioxins and dibenzofurans, National Institute of Environmental Health Services, April 2-3, 1973. Environmental Health Perspectives (September 1973):279-282.
36. Burning hazardous wastes on the high seas. 1983. Chemical Week (February 23):10-11.
37. Camoni, I., et al. 1982. Laboratory investigation for the microbiological degradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin in soil by addition of organic compost. Environmental Science 5:95-103. London: Pergamon Press, Ltd.
38. Carlson, R.E. 1981. Letter to the editor concerning Bumb, R.R., et al., 1980. Science 213(4 September):1159.
39. Carreri, V. n.d. Review of the events which occurred in Seveso. In Dioxin: toxicological and chemical aspects, eds. F. Cattabeni, A. Cavallaro, and G. Galli, 1-5. New York: SP Medical & Scientific Books, Spectrum Publications, Inc.
40. Carter, Coleman D., et al. 1975. Tetrachlorodibenzodioxin: An accidental poisoning episode in horse arenas. Science 188(May 16):738-40.
41. Chapman, Gerald O., et al. 1982. Chemical waste incinerator ships: The interagency program to develop a capability in the United States. Marine Technology 19(4, Oct. 1982):325-340.
42. Checking dioxin. 1978. Chemical Week (May 17):17.

43. Choudhry, Chulam Ghaus, and Otto Hutzinger. 1982. Mechanistic aspects of the thermal formation of halogenated organic compounds including polychlorinated dibenzo-p-dioxins: Part II, Thermochemical generation and destruction of dibenzofurans and dibenzo-p-dioxins. Toxicological and Environmental Chemistry 5:67-93.
44. Ciaccio, Edward I. 1979. Dioxin contamination. Letter to the editor. Chemical & Engineering News (April 16):3.
45. Clark, R. Douglas. 1976. Defoliant 2,4,5-T: Ecological Russian roulette. The Nation (November 20):530-532.
46. Commoner, B., and R.E. Scott. 1976. Accidental contamination of soil with dioxin in Missouri: Effects of countermeasures. St. Louis, Missouri: Center for the Biology of Natural Systems, Washington University.
47. Cormack, Donald E., et al. 1977. Parameters and mechanisms in the solvent extraction of mined athabasca oil sand. The Canadian Journal of Chemical Engineering 55(October):572-580.
48. Cotter, J.L., et al. 1981. Facilities evaluation of high efficiency boiler destruction PCB waste. Research brief, Jan.-Apr. 1980. Prepared by TRW, Inc., Redondo Beach, CA. EPA report no. EPA-600/7-81-031. NTIS report no. PB81-178287. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
49. The cracks in Dow's theory of dioxins. 1983. Chemical Week (April 13):26-27.
50. Criteria and Standards Division, Office of Water Planning and Standards, U.S. EPA 2,3,7,8-tetrachlorodibenzo-p-dioxin: Ambient water quality criteria. Washington, D.C.: U.S. EPA. Xerox.
51. Crosby, D.G. Experimental approaches to pesticide photodecomposition.
52. Crosby, D.G., et al. 1971. Photodecomposition of chlorinated dibenzo-p-dioxins. Science 173(August 20): 748-749.
53. Crosby, D.G. and A.S. Wong. 1977. Environmental degradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Science 195(March):1337-1338.
54. Crosby, D.G. 1978. Conquering the monster--the photochemical destruction of chlorodioxins. In Disposal and Decontamination of Pesticides, ed. M.V. Kerred, 1-12. American Chemical Society Symposium 73. Washington, D.C.: ACS.
55. Crow, K.D. 1977. Effects of dioxin exposure. Letter to the editor and response from Joan-Ramon LaPorte. The Lancet (July 9):82.
56. Crummett, W.B. 1981. Letter to the editor concerning Bumb, R.R., et al., 1980. Science 213(4 September):1160.
57. Currents: Process chemistry. 1983. Chemical Engineering 90(1):23-28.
58. Dallaire, Gene. 1981. Hazardous waste management in California: Lessons for the U.S. Civil Engineering (April):53-56.

59. Dennis, William H. Jr. October 1972.. Methods of chemical degradation of pesticides and herbicides--A review. USAMEERU report no. 73-04. NTIS AD-752 123. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
60. Dennis, W.H. Jr., and W.J. Cooper. 1975. Catalytic dechlorination of organochlorine compounds. I. DDT. Bulletin of Environmental Contamination & Toxicology 14(6):738-744.
61. des Rosiers, Paul E. 1983. Remedial measures and disposal practices for wastes containing dioxins and furans. Chemosphere 12:727-744. London: Pergamon Press.
62. Destroying dioxin: A unique approach. 1980. Waste Age (October):60-63.
63. Destruction of pesticides and pesticide containers by molten salt combustion. In Disposal and decontamination of pesticides, ed. M.V. Kerred, 119-130. American Chemical Society Symposium Series 73. Washington, D.C.: ACS.
64. Detoxifying dioxin. 1977. Chemical Week (November 23):17.
65. diDomenico, Alessandro, et al. 1978. Accidental release of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at Seveso: Assessment of environmental contamination and of effectiveness of decontamination treatments. CODATA Bulletin 29:53-59.
66. diDomenico, Alessandro, et al. March 1980. Accidental release of 2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD) at Seveso, Italy. Parts I-VI: Sensitivity and specificity of analytical procedures adopted for TCDD assay (Part I), TCDD distribution in the soil surface layer (Part II), Monitoring of residual TCDD levels in reclaimed buildings Part III), Vertical distribution of TCDD in soil (Part IV), Environmental persistence of TCDD in the Seveso area (Part V), TCDD levels in atmospheric particles (Part VI). Ecotoxicological Environmental Safety 4(3):283-297, 298-320, 321-326, 327-338, 339-345, 346-356.
67. Dillon Ltd. n.d. Destruction Technologies for Polychlorinated Biphenyls (PCB's). Waste Management Branch, Environmental Impact Control Directorate, Environmental Protection Service, Environment Canada.
68. The dioxin curse. 1977. Atlas World Press Review (Nov.):13-15. Also appeared in the 1977 Yearbook of the Stockholm International Peace Research Institute.
69. Dioxin down under. 1978. Chemical Week (May 10):21.
70. Dioxin is found in cleanup workers' blood. 1979. Chemical Week (May 23):22.
71. Dioxin lingers on. 1978. Chemical Week (April 12):22.
72. Drain system is installed to collect leachate from Love Canal 'bathtub.' 1980. Solid Wastes Management/Refuse Removal Journal 23:2:20.
73. Duckett, E. Joseph. 1981. Dioxins in perspective: Knowns, unknowns, resolving the issues. Solid Wastes Management/Refuse Removal Journal (May):56.
74. Dunn, K.S. 1979. Problems and practicalities of incinerating chemical wastes. The Chemical Engineer 350(November):79-782.

75. Ebon Research Systems. May 1978. Liners for sanitary landfills and chemical and hazardous waste disposal sites. Prepared for Municipal Environmental Research Laboratory, EPA. NTIS no. PB-293 335. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
76. Edwards, Barbara H., John N. Paulin, and Kathleen Coghlan-Jordan. 1981. Emerging technologies for the destruction of hazardous waste: ultraviolet ozone destruction. Land Disposal: Hazardous Waste, Proceedings of the Seventh Annual Research Symposium, March 16-18, 1981, Philadelphia, Pennsylvania. EPA-680/9-81-0026 (March 1981)265-271.
77. Edwards, Barbara H., et al. 1982. Emerging technologies for the control of hazardous wastes. Final report. Prepared by EBON Research Systems, Washington, D.C. EPA report no. EPA-600/2-32-011. NTIS report no. PB82-236993. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
78. Epstein, Samuel A., Lester O. Brown, and Carl Pope. 1982. Hazardous waste in America. San Francisco, California: Sierra Club Books.
79. Envirotech Corp. 1975. A study of pesticide disposal in a sewage sludge incinerator. EPA report no. SW-116c. Available from National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia.
80. Essentials of soil-cement: Construction, inspection, field control. 1958. Chicago, IL: Portland Cement Association.
81. Fabian, H.W., P. Reher, and M. Schoen. 1979. How Bayer incinerates wastes. Hydrocarbon Processing (April):183-192.
82. Fadiman, Anne. 1979. A poisoned town. Life 110(10):43-49.
83. Fanelli, R., et al. 1978. Degradation of 2,3,7,8-tetrachlordibenzo-p-dioxin in organic solvents by gamma ray irradiation. Experientia 34(9):1126-1127.
84. Fanelli, R., et al. 1980. 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic effects and tissue levels in animals from the contaminated area of Seveso, Italy. Arch. Environmental Contam. Toxicol. 9:569-577.
85. Ferm, Ronny and Lars Renberg. 1978. An industrial cover-up: The case of BT Kemi. Ambio 7:211-17.
86. Forrester, Ray. 1981. The Denney farm site remedial project: A model for the safe excavation, storage, and elimination of dioxin. In Proceedings, U.S. EPA National Conference on Management of Uncontrolled Hazardous Waste Sites, 326-328. Silver Spring, Maryland: Hazardous Material Control Research Institute.
87. Frankel, Irwin, Neil Sanders, and Greg Vogel. 1983. Reducing plant pollution exposure: Survey of the incinerator manufacturing industry. Chemical Engineering Progress (March):44-55. Also, American Institute of Chemical Engineering publication 0360-7275/83/7007-0044.

88. Gebefugi, I., R. Baumann und F. Korte. 1977. Photochemischer Abbau von 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) unter simulierten Umweltbedingungen. Naturwissenschaften 64:486-87.
89. Geiser, Edward M. and Russell W. Johnson. September 1981. Purification of chlorophenolic derived compounds. U.S. Patent 4,287,038.
90. Ghezzi, Italo, et al. 1982. Potential 2,3,7,8-tetrachlorodibenzo-p-dioxin exposure of Seveso decontamination workers: A controlled prospective study. Scandinavian Journal of Work & Environmental Health 8(suppl 1):176-179.
91. Gizzi, F., et al. 1982. Polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) in emissions from an urban incinerator. 1. Average and peak values. Chemosphere 11(6):577-582. Report no. 0045-6535/82/060577-07. Great Britain: Pergamon Press.
92. Global Marine will burn chemical wastes at sea. 1979. Chemical Week (July 11):20.
93. Gregory, R.C. 1981. Design of hazardous waste incinerators. Chemical Engineering Progress 77(4):43-47.
94. Gribble, Gordon W. 1974. TCDD: A deadly molecule. Chemistry 47(2, February):15-18.
95. Hall, Richard S., Jay Matley, and Kenneth J. McNaughton. 1982. Current costs of process equipment. Chemical Engineering (April 5, 1982):79+.
96. Hardison, L.C. and E.J. Dowd. 1977. Emission control via fluidized bed oxidation. Chemical Engineering Progress 73(8):31-35.
97. Harrison, Don D., Charles I. Miller, and Richard C. Crews. 1979. Residual levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) near herbicide storage and loading areas at Eglin AFB, Florida. Prepared for Air Force Armament Laboratory, Armament Development and Test Center, Eglin AFB, Florida. Air Force report no. AFTL-TR-79-20. NTIS no. ADA-078 819. National Technical Information Service, Springfield, Virginia: U.S. Department of Commerce. Copies also available from the DDC.
98. Harrison, John Michael, and Rodney Gordon Wilkinson. April 1981. A process for the electrochemical degradation of persistent organic compounds, with harmful or potentially harmful properties. European patent application, publication number 0027745. London, England: Creconsult Limited.
99. Hay, Alastair. 1981. Chlorinated dioxins and the environment. Nature 289 (Jan. 29):351-52.
100. Hay, Alastair. 1981. The chemical scythe: lessons of 2,4,5-T and dioxin. Disaster Research in Practice Series, ed. Frances D'Souza. New York and London: Plenum Press.
101. Hazardous Waste Management Division of the Office of Solid Waste Management Programs, U.S. EPA. 1975. Incineration in hazardous waste management: a current report on solid waste management. EPA report no. 530/SW-141. U.S. EPA.
102. Hazardous waste management system: Proposed rule by the Environmental Protection Agency. Part III. April 4, 1983. Federal Register 48(65):14514-14529.

103. Hazelwood, Douglas L., et al. 1982. Assessment of waste fuel use in cement kilns. Prepared by A.T. Kearney, Inc., Alexandria, VA. EPA report no. EPA-600/2-82-013. NTIS report no. PB82-236043. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
104. Helling, C.S., et al. 1973. Chlorodioxins in pesticides, soils, and plants. Journal of Environmental Quality 2(2):171-178.
105. Heritage, J. 1978. Major American toxic disasters. EPA Journal (September):8-10.
106. Hescheles, C.A. and R.F. Bouner, Jr. 1974. Ultimate disposal of wastes by pyrolysis and incineration. In Proceedings, National Incinerator Conference, Miami, Florida, May 12-15, 1974. ASME.
107. Hollis, James R. 1983. Plasma temperature incineration. Environmental Progress 2(1):7-9.
108. Hooker dumpsites may pose dioxin threat. 1979. Chemical Week (January 2):16.
109. Hornig, A.W. 1982. Decomposition of chlorinated hydrocarbons using a novel high-temperature fluid wall reactor. In Proceedings, 36th Industrial Waste Conference, Purdue University, 203-211. Ann Arbor, MI: Ann Arbor Science.
110. Houston Research, Inc. 1976. Oxidation of pesticides by ozone and ultraviolet light. Army Mobility Equipment Research and Development Command report no. 7206. NTIS no. AD-A028 306. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
111. How to commercialize a tar sand deposit. 1975. Mining Engineering (24 January):24-29.
112. Howard, Kenneth J., and Albert E. Sidwell. April 1982. Chemical detoxification of toxic chlorinated aromatic compounds. U.S. Patent 4,327,027.
113. Hryhorczuk, Daniel O., et al. 1981. A wire reclamation incinerator as a source of environmental contamination with tetrachlorodibenzo-p-dioxins and tetrachlorodibenzofurans. Archives of Environmental Health 36(5):228.
114. Huber Corp. n.d. Huber Technology Fluid-Wall (HTFW) Reactor: Technical Bulletin. Borges, TX.
115. Huber Corp. 1983. J.M. Huber PCB Destruction Process Trial Burn Report. Prepared by Radian Corp., Austin, TX.
116. Huff, James E., and John S. Wasson. 1973. Chlorinated dibenzodioxins and dibenzofurans. Environmental Health Perspectives (September 1973):283-312.
117. Hutter, R. and M. Philippi. Studies on microbial metabolism of TCDD under laboratory conditions. In Chlorinated dioxins and related compounds by O. Hutzinger, et al., 87-93. New York: Pergamon Press.
118. Hutzinger, O., et al. 1973. Photochemical degradation of di- and octachlorodibenzofuran. Environmental Health Perspectives (Sept.):267-71.

119. Jansson, Erik. 1978. 2,4,5-T: The horror goes on. Not man apart--FOE 8(12, August/September):8.
120. Josephson, Julian. 1983. Chlorinated dioxins and furans in the environment. Environmental Science Technology 17(3):124A-128A. American Chemical Society publication no. 0013-936X/83/0916-0124A.
121. Junk, G.A. and C.S. Ford. 1980. A review of organic emissions from selected combustion processes. Department of Energy report no. IS-4727. Ames, IA: Ames Laboratory, U.S. Department of Energy.
122. Jungclaus, Gregory A., Viorica Lopez-Avila, and Ronald A. Hites. 1978. Organic compounds in an industrial wastewater: A case study of their environmental impact. Environmental Science & Technology:88-96.
123. Kamlet, Kenneth S. 1981. Ocean disposal of organochlorine wastes by at-sea incineration. In Ocean dumping of industrial wastes. Vol. 12 of Marine Science. New York: Plenum Press.
124. Kearney, Philip C., Edwin A. Woolson, and Charles P. Ellington, Jr. 1972. Persistence and metabolism of chlorodioxins in soils. Environmental Science & Technology 6(12):1017-1019.
125. Kearney, P.C. et al. 1973. Environmental significance of chlorodioxins. In Chlorodioxins: Origin and fate, ed. E.H. Blair, 105-111. Advances in Chemistry, Series 120. Washington, D.C.: American Chemical Society.
126. Kearney, P.C. 1973. Tetrachlorodibenzodioxin in the environment: Sources, fate, and decontamination. Environmental Health Perspectives (Sept.):273-77.
127. Kearney, P.C., jr., J.R. Plimmer, and Zheng-Ming Li. 1983. UV-ozonation and land disposal of aqueous pesticide wastes. Volume 4 in Pesticide chemistry: Human welfare and the environment. Proceedings of the 5th International Congress on Pesticide Chemistry.
128. Kenchington, John M. and Colin R. Phillips. 1981. Operating cost parameters in solvent extraction of bitumen from oil sand mineral deposits. Energy Sources 4:317-338. Publication no. 0090-8312/81/010317-00, Crane, Russak & Co., Inc.
129. Kennedy, M.V., B.J. Stojanovic, and F.L. Shuman, Jr. n.d. Chemical and thermal methods for disposal of pesticides.
130. Kezdi, A. 1979. Stabilized earth roads, 1st ed. New York: El Sevien Publication Co.
131. Kilbane, J.J., et al. 1983. Biodegradation of 2,4,5-trichlorophenoxyacetic acid by a pure culture of Pseudomonas cepacia. Applied and Environmental Microbiology 44(1):72-78.
132. Kimble, B.J., and M.L. Gross. 1980. Tetrachlorodibenzo-p-dioxin quantitation in stack-collected coal fly ash. Science 207(4 January):59-61.
133. Kimbrough, Renate D. 1974. The toxicity of polychlorinated polycyclic compounds and related chemicals. CRC Critical Reviews in Toxicology (January):445-498. Reprinted by the U.S. Department of Health, Education, and Welfare, Public Health Service.

134. Klecka, G.M. and D.T. Gibson. 1980. Metabolism of dibenzo-p-dioxin and chlorinated dibenzo-p-dioxins by a *Beijerinckia* species. Applied and Environmental Microbiology 39(2):288-296.
135. Klecka, G.M. and D.T. Gibson. 1981. Bacterial degradation of dibenzo-p-dioxin and chlorinated dibenzo-p-dioxins. EPA report no. 600/4-81-016. NTIS no. PB81-171 639. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
136. Kriebel, D. 1981. Letter to the editor concerning Bumb, R.R., et al., 1980. Science 213 (4 September):1159.
137. Langer, H.G., et al. 1973. Thermal chemistry of chlorinated phenols. In Chlorodioxins: Origin and fate, ed. E.H. Blair. Advances in Chemistry Series 120. Washington, D.C.: American Chemical Society.
138. Lavergne, Edgar A. July 1974. Study of feasibility of Herbicide Orange chlorinolysis. Prepared for the Office of Research and Development, U.S. Environmental Protection Agency. Painesville, Ohio: Diamond Shamrock Corporation, T.R. Evans Research Center.
139. Lauber, Jack D. 1982. Burning chemical wastes as fuels in cement kilns. Journal of the Air Pollution Control Association 32(7):771-777.
140. Lewis, C. Randall, Richard E. Edwards, and Michael A. Santoro. 1976. Incineration of industrial wastes. Chemical Engineering (Deskbook issue) (October 18):115-121.
141. Liberti, A., et al. 1978. Solar and UV photodecomposition of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the environment. The science of the total environment 10:97-104. Amsterdam: Elsevier Scientific Publishing Co.
142. Lombardi, E.F. 1981. Incineration of polychlorinated biphenyl using a fluidized-bed incinerator. Rockwell International report no. RFP-3271*. Golden, CO: Rockwell International Corp.
143. Machan, G., et al. 1977. Laboratory study of the effectiveness of cement and of lime stabilization for erosion control. Trans. Res. Rec. 641:24-28.
144. Manson, L., et al. 1979. Hazardous material incinerator design criteria. Prepared by TRW, Inc., Redondo Beach, CA. EPA report no. EPA-600/2-79-198. NTIS report no. PB80-131964. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
145. Marshall valuation service. n.d. Los Angeles: Marshall and Swift Publication Company.
146. Matsumura, Fumio, and Herman H. Benezet. Studies on the bioaccumulation and microbial degradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin. Environmental Health Perspectives (Sept.):352-58.
147. Matsumura, Fumio, and Claudia T. Ward. 1976. Studies on the degradation of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) in lake water and sediment. M.S. thesis, Department of Entomology, University of Wisconsin, report no. W7705602; OWRTA-058-WIS(2). Prepared for the Office of Water Research and Technology, U.S. Department of the Interior. NTIS no. PB-264 881. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.

148. Matsumura, F., John Quensen, and G. Tsushimoto. 1983. Microbial degradation of TCDD in a model ecosystem. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 191-219. Proceedings of an international symposium on Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.
149. Matthiaschk, G. n.d. Survey about toxicological data of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). In Dioxin: Toxicological and chemical aspects. ed. Flaminio Cattabeni, Aldo Cavallaro, and Giovanni Galli, 185. New York: Spectrum Publications, Inc.
150. McBride, James L. and James A. Heimbuch. 1982. Skid mounted system gives California haz wastes hot times. Pollution Engineering (July):24-26.
151. McConnell, E.E. and J.A. Moore. n.d. The toxicopathology of TCDD. In Dioxin: Toxicological and chemical aspects. ed. Flaminio Cattabeni, Aldo Cavallaro, and Giovanni Galli, 137. New York: Spectrum Publications, Inc.
152. McGill, Douglas L. and Elbridge M. Smith. 1970. Fluidized bed disposal of secondary sludge high in organic salts. In Proceedings, National Incinerator Conference, Cincinnati, Ohio, May 17-20, 1970. ASME.
153. Meadus, F.W., P.J. Chevrier, and B.D. Sparks. 1982. Solvent extraction of Athabasca oil-sand in a rotating mill. Part 1. Dissolution of bitumen. Fuel Processing Technology 6(1982):277-287. Also, publication no. 0373-3820/82/0000-0000, Elsevier Scientific Publishing Company, Amsterdam.
154. Meadus, F.W., B.P. Bassaw, and B.D. Sparks. 1982. Solvent extraction of Athabasca oil-sand in a rotating mill. Part 2. Solids-liquid separation and bitumen quality. Fuel Processing Technology 6(1982):289-300. Also, publication no. 0378-3820/82/0000-0000, Elsevier Scientific Publishing Company, Amsterdam.
155. Means building construction cost data 1982. 1981. Kingston, MA: Robert Snow Means Company, Inc.
156. Merle, Robert L., Melvin C. Young, Glen R. Love. 1976. Design and operation of a suspension fired industrial solid waste disposal system for Kodak Park. In Proceedings, Vol. 7, National Waste Processing Conference.
157. Metcalf, J.B. 1977. Principles and application of cement and lime stabilization. Austral. Rd. Res. Board, research report no. 49.
158. Miller, R.A., L.A. Norris, and C.L. Hawkes. 1973. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic organisms. Environmental Health Perspectives (September):177-186.
159. Miller, R.A., L.A. Norris, and B.R. Loper. 1979. The response of coho salmon and guppies to 2,3,7,8-tetra-chlorodibenzo-p-dioxin (TCDD) in water. Transactions of the American Fisheries Society 108:401-407.
160. Miller, R.A., et al. 1981. Destruction of toxic chemicals by catalyzed wet oxidation. Proceedings, Industrial Waste Conference 35:425-429.
161. Minker/Stout/Romaine Creek (Zone II) study (draft). 1982. Prepared by CH2M HILL, Corvallis, OR, for U.S. EPA, Kansas City, MO.

162. Minker/Stout/Romaine Creek (Zone II) work plan (draft). April 1983. Prepared by CH2M HILL, Corvallis, OR, for U.S. EPA, Kansas City, MO.
163. Missouri Department of Natural Resources. March 29, 1983. Report on potential and confirmed uncontrolled hazardous waste sites in Missouri.
164. Morrison, W.R. and L.R. Simmons. 1977. Chemical and vegetative stabilization of soil: laboratory and field investigations of new materials and methods for soil stabilization and erosion control. In Bureau Reclamation, Engineering Research Center report no. 7613.
165. Mountjoy, D.S. 1977. Incineration of industrial wastes and subsequent effluent control. Progress in Water Technology 9:937-940. London: Pergamon Press.
166. National Research Council of Canada, Associate Committee on Scientific Criteria for Environmental Quality. 1978. Phenoxy herbicides: Their effects on environmental quality. With accompanying scientific criteria for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). NRCC publication no. 16075. Ottawa, Canada: NRCC.
167. NATO Committee on the Challenges of Modern Society. 1981. NATO-CCMS pilot study on disposal of hazardous wastes. Project: Thermal Treatment. Prepared for the Federal Republic of Germany. NATO/CCMS report no. 118. Brussels, Belgium: NATO Committee on the Challenges of Modern Society. NTIS report no. PB82-114521. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
168. Newton, M., and L.A. Norris. 1980. Potential exposure of humans to 2,4,5-T and TCDD in the Oregon Coast Range. Paper presented at the Weed Science Society of America, February 7, 1980, Toronto, Ontario, Canada.
169. Noe, Luigi. 198_. Reclamation of the TCDD-contaminated Seveso area. (Typescript of Chapter 3 of unidentified document provided by Dr. Phil Kearney, USDA.)
170. Norris, L.A. and R.A. Miller. 1974. The toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in guppies (Poecilia reticulatus Peters). Bulletin of Environmental Contamination & Toxicology 12(1):76-80.
171. Norris, Logan A. 1981. The movement, persistence, and fate of the phenoxy herbicides and TCDD in the forest. Residue Reviews 80:65-135. New York: Springer-Verlag New York, Inc. Also published separately as document 799-363 1981 by the Government Printing Office, for the USDA Forest Service.
172. Norris, Logan A. 1981. Persistence of TCDD in the forest environment. A progress report on NAPIAP research. Corvallis, Oregon: Pacific Northwest Forest and Range Experiment Station, USDA Forest Service. Xerox (February 17).
173. Old landfill site poses health problems. 1978. Chemical and Engineering News (August 7):6.
174. Oldham, J.C. et al. 1977. Materials Evaluated as Potential Soil Stabilizers. U.S. Army Waterw. Exper. Str. Vicksburg misc. paper no. S-77-15.
175. Panel discussion on Environmental Chemistry, Philip C. Kearney, Chairman. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 777-781. Proceedings of an international symposium on

Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.

176. Panel discussion on Environmental Toxicology, Eugene E. Kenaga, Chairman. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 783-785. Proceedings of an international symposium on Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.
177. Panel discussion on Human Observations, Kenneth D. Crow, M.D. and Giuseppe Reggiani, Co-Chairmen. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 795-797. Proceedings of an international symposium on Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.
178. Parks, Marion. 1978. An environmental calamity: The Seveso case. EPA Journal 4(8, September):11-15.
179. Paulson, E.G. 1977. How to get rid of toxic organics. Chemical Engineering (Deskbook Issue) (October 17):21-27.
180. PEDCo Environmental, Inc. 1980. Dioxins. EPA report no. 600/2-80-197. NTIS no. PB82-136 847. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
181. Pemberton, John M. 1979. Pesticide degrading plasmids: A biological answer to environmental pollution by phenoxyherbicides. Ambio 8:202-5.
182. Peters, James A., Thomas W. Hughes, and Robert E. Mournighan. 1983. Evaluation of hazardous waste incineration in a cement kiln at San Juan Cement. Xerox copy being reviewed for publication by the U.S. EPA.
183. Peters, Max S. and Klaus D. Timmerhaus. 1980. Plant design and economics for chemical engineers. Third ed., McGraw-Hill Chemical Engineering Series, New York: McGraw-Hill Book Company.
184. Philippi, Martin, et al. 1981. Fate of TCDD in microbial cultures and in soil under laboratory conditions. In Microbial degradation of xenobiotics and recalcitrant compounds by Th. Leisinger, A.M. Cook, R. Hutter, and J. Nuesch, New York: Academic Press, 221-233.
185. Philippi, Martin, et al. 1982. A microbial metabolite of TCDD. Experientia 38:659-661.
186. Piper, Walter N., James Q. Rose, and Perry J. Gehring. 1973. Excretion and tissue distribution of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the rat. Environmental Health Perspectives (September 1973):241-244.
187. Plimmer, J.R. and U.I. Klingebiel. 1973. Photochemistry of dibenzo-p-dioxins. In Chlorodioxins: Origin and fate, ed. E.H. Blair, 44-54. Advances in Chemistry Series 120. Washington, D.C.: American Chemical Society.

188. Plimmer, J.R. 1978. Approaches to decontamination or disposal of pesticides: Photodecomposition. In Disposal and decontamination of pesticides, ed. M.V. Kerred, 13-23. American Chemical Society Symposium Series 73. Washington, D.C.: ACS.
189. Plimmer, J.R. 1978. Photolysis of TCDD and trifluralin on silica and soil. Bulletin of Environmental Contamination and Toxicology 20(1):87.
190. Pocchiari, Francesco 1978. 2,3,7,8-tetrachlorodibenzo-para-dioxin decontamination. Ecological Bulletin 27 (Chlorinated Phenoxy Acids and Their Dioxins):67-70.
191. Pocchiara, Francesco. 1980. Foreword to papers I-VI: Accidental release of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy. Ecotoxicology and Environmental Safety 4:282.
192. Poiger, H. and Ch. Schlatter. 1979. Biological degradation of TCDD in rats. Nature 281(October 25):706-707.
193. Poiger, H. and Ch. Schlatter. 1980. Influence of solvents and adsorbents on dermal and intestinal absorption of TCDD. Food & Cosmetic Toxicology 18:477-481. London: Pergamon Press.
194. Pojasek, Robert B., ed. 1979. Toxic and hazardous waste disposal. Vol. 1 of Processes for stabilization/solidification. Ann Arbor, Michigan: Ann Arbor Science Publishers, Inc.
195. Pojasek, Robert B., ed. 1980. Toxic and hazardous waste disposal. Vol. 4 of New and promising ultimate disposal options. Ann Arbor, Michigan: Ann Arbor Science Publishers, Inc.
196. Poland, Alan and Edward Glover. Studies on the mechanism of toxicity of the chlorinated dibenzo-p-dioxins. Environmental Health Perspectives (September 1973):245-251.
197. Portland Cement Association. 1968. Design and control of concrete mixtures. 11th ed. Engineering Bulletin. Skokie, Illinois: Portland Cement Association.
198. Princeton, Irma. 1982. Three new ships to burn toxic wastes. Solid Wastes Management (July):20.
199. Quensen, J.F. and F. Matsumura. n.d. Oxidative degradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin by microorganisms. Presented at the Second Annual Meeting of Environmental Toxicology and Chemistry, November 22-25, 1981, Arlington, VA. Xerox.
200. Quensen, J.F., III, and F. Matsumura. 1983. Oxidative degradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin by microorganisms. Environmental Toxicology and Chemistry 2:261-268.
201. Rappe, Christoffer. n.d. Decontamination of products formed during the industrial preparation of 2,4,5-trichlorophenol. In Dioxin: Toxicological and chemical aspects, ed. F. Cattabeni, A. Cavallaro, and G. Galli, 201. New York: SP Medical & Scientific Books, Spectrum Publications, Inc.

202. Rappe, Christoffer. n.d. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD): Introduction. In Dioxin: Toxicological and chemical aspects. Ed. F. Cattabeni, A. Cavallaro, and G. Galli, 9. New York: SP Medical and Scientific Books, Spectrum Publications, Inc.
203. Rappe, et al. 1982. Polychlorinated dioxins (PCDDs), dibenzofurans (PCDFs), and other polynuclear aromatics (PC PNAs) formed during PCB fires. Chemica Scripta 20:56-61.
204. Rawls, Rebecca L. 1979. Dow finds support, doubt for dioxin ideas. Chemical & Engineering News (February 12):23-29.
205. Reason, John. 1982. Does European technology work here? Power, 126 (December):80-81.
206. Reggiani, G. n.d. TCDD contamination in Italy: The risk assessment of low level exposure, cumulative effect and long-term consequences. Basle, CH: Medical Research Board, F. Hoffman-La Roche & Co., Ltd.
207. Reggiani, G. 1978. Medical Problems raised by the TCDD contamination in Seveso, Italy. Archives of Toxicology 40:161-188. New York: Springer-Verlag, publication no. 0340-5761/78/0040/0161.
208. Renzoni, Aristeo. 1977. The increasing number of environment-degrading accidents in Italy. Environmental Conservation 4(1, Spring):21-26.
209. The Richardson rapid construction cost estimating system. 1982. Process plant construction estimating standards. 4 vols. San Marcos, CA: Richardson Engineering Services, Inc.
210. Riley, Boyd T. Jr. October 1975. Summation of conditions and investigations for the complete combustion of organic pesticides. Prepared for U.S. EPA. EPA report no. 600/2-75-044. Cincinnati, Ohio: Municipal Environmental Research Laboratory; Office of Air, Land, and Water Use; U.S. EPA. Also available from the National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia.
211. Roadbed Stabilization Lime Injection Conference. 1976. In Proceedings of Roadbed Stabilization Lime Injection Conference, Little Rock, Arkansas, November 1975, ed. J. Blacklock. NTIS report no. PB-251-681. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
212. Robbins, Anthony. n.d. Dioxin studies. Letter to the editor.
213. Roderick, Kevin. 1976. Toxic war defoliant may be buried in West Covina. Los Angeles Times (Sept. 1).
214. Rogers, Charles J. and Robert Allen. 1978. Developing technology for detoxification of pesticides and other hazardous materials. In Disposal and decontamination of pesticides, ed. M.V. Kerred. American Chemical Society Symposium Series 73, 100-11. Washington, D.C.:ACS.
215. Ross, R.D. 1972. Incineration of solvent-air mixtures. Chemical Engineering Progress 68(8):59-64.

216. Sabry, M.M.A. and J.V. Parcher. 1979. Engineering properties of soil-lime mixes. Note 1. In Transportation Engineering Journal, Proceedings ASCE 105.
217. Schaum, John. December 10, 1982. Exposure assessment of Missouri horse arenas and related areas. Xerox of an unpublished report from the Office of Solid Waste and Emergency Response, U.S. EPA, Washington, D.C.
218. Schwetz, B.S., et al. 1973. Toxicology of chlorinated dibenzo-p-dioxins. Environmental Health Perspectives (September 1973):283-312.
219. Scrubdown for dioxin. 1978. Chemical Week (June 21):13.
220. SCS Engineers. n.d. Cost comparisons of treatment and disposal alternatives for hazardous wastes. Volume II, Appendices. Prepared for U.S. EPA, Contract No. 68-03-2754. Cincinnati, Ohio: Municipal Environmental Research Laboratory.
221. SCS Engineers. n.d. Costs of remedial response actions at uncontrolled hazardous waste sites. Prepared for U.S. EPA, Contract No. 68-01-4885. Cincinnati, Ohio: Municipal Environmental Research Laboratory.
222. SCS Engineers. 1982. Hazardous waste site scenario 1 for cost estimation of remedial action unit operations. Covington, Kentucky: SCS Engineers.
223. Seagoing furnace destroys toxics. 197_. EPA Journal (September):16-17.
224. Sebastian, F.P., et al. 1974. Latest developments on polychlorinated biphenyls decomposition in BSP multiple-hearth furnaces. In Proceedings, American Institute of Chemical Engineering Workshop, November 1-3, 1972, Midland, Michigan. Vol. 5, Industrial Process Design for Pollution Control. New York: American Institute of Chemical Engineering.
225. Sekulic, Tim S., et al. 1983. Air quality effects of a hazardous waste incineration system. Environmental Progress 2(1):47-50. Also, American Institute of Chemical Engineers publication 0278-4491-83-6802-0047.
226. Serper, Allen. 1980. Incineration may be best disposal method for most hazardous wastes. Solid Wastes Management/Refuse Removal Journal 23(4, April):122.
227. Seveso cleanup still not solved. 1979. Chemical Week (July 18):23.
228. Shaub, Walter M. 1982. Estimated thermodynamic functions for some chlorinated benzenes, phenols, and dioxins. Thermochemica Acta 38:11-44. Amsterdam: Elsevier Scientific Publishing Company.
229. Shaub, W.M. and W. Tsang. 1983. Physical and chemical properties of dioxins in relation to their disposal. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 731-748. Proceedings, international symposium on Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.
230. Shea, Kevin P. and Bert Lindler. 1975. Pandora and the storage tank. Environment 17(Sept.):12-15.

231. Shen, T.T., M. Chen, and J. Lauber. 1978. Incineration of toxic chemical wastes. Pollution Engineering (October):45-50.
232. Shih, C.C., et al. 1978. Comparative cost analysis and environmental assessment for disposal of organo-chlorine wastes. Prepared by TRW, Inc., Redondo Beach, CA. EPA report no. EPA/600/2-78/190. NTIS report no. PB-286 095. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
233. Singal, Mitchell, et al. (National Institute for Occupational Safety and Health, Hazard Evaluations and Technical Assistance Branch). 1981. Health hazard evaluation report 79-022-789. Washington, D.C.: U.S. Government Printing Office.
234. Sittig, Marshall. 1979. Incineration of industrial hazardous wastes and sludges. Park Ridge, New Jersey: Noyes Data Corporation.
235. Smith, Robert M., et al. 1983. 2,3,7,8-tetrachlorodibenzo-p-dioxin in sediment samples from Love Canal storm sewers and creeks. Environmental Science & Technology 17(1):6-10.
236. Soil-cement laboratory handbook. 1959. Chicago, IL: Portland Cement Association.
237. Sparks, B.D. and F.W. Meadus. 1981. A study of some factors affecting solvent losses in the solvent extraction--spherical agglomeration of oil sands. Fuel Processing Technology 4(1981):251-264. Also issued as publication no. 18821, National Research Council of Canada, Chemistry Division, Ottawa, Ontario, Canada.
238. Stalling, David L., et al. May 1977. Method of removing polynuclear compounds by adsorption with coconut charcoal. U.S. Patent 4,026,917.
239. Stehl, R.H., et al. 1973. The stability of penta-chlorophenol and chlorinated dioxins to sunlight, heat, and combustion. In Chlorodioxins: Origin and fate, ed. E.H. Blair, 119-125. Advances in Chemistry Series 120. Washington, D.C.: American Chemical Society.
240. Stewart, Wilford S. Exxon Research and Engineering Co. December 1978. State-of-the-art study of land impoundment techniques. EPA report no. 600/2-78-196. Cincinnati, Ohio: Municipal Environmental Research Laboratory, Office of Research and Development, U.S. EPA.
241. Still more hassles for Hooker. 1979. Chemical Week (April 25):23.
242. Stretz, L.A., and J.S. Vavruska. 1982. Hazardous waste incineration research at the Los Alamos National Laboratory. Report no. LA-UR-82-126. Los Alamos, NM: Los Alamos National Laboratory. Department of Energy report no. DE82 010367. Technical Information Center, U.S. Department of Energy.
243. Stolzenburg, Thomas and John Sullivan. 1983. Dioxin: A Cause for Concern? Prepared by the University of Wisconsin Sea Grant Institute, Madison, WI. NTIS report No. PB84-104199. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
244. Suits boost claims for dioxin-spill damage. 1979. Chemical Week (July 25):21.
245. Suskind, R.R., M.D., and V.S. Hertzberg, Ph.D. 1984. Human health effects of 2,4,5-T and its toxic contaminants. Journal of the American Medical Association 251(May 11):2372-2380.

246. TCDD in the environment. 1979. Transactions of the American Fisheries Society. 108:103-109.
247. Technologies and management strategies for hazardous waste control. March 1983. Office of Technology Assessment report no. OTA-M-196. Washington, D.C.: U.S. Congress Office of Technology Assessment.
248. Tenzer, Raymond, et al. 1980. Mobile system for the detoxification/incineration of cleanup residuals from hazardous material spills. In Disposal of oil and debris resulting from a spill cleanup operation, ASTM STP 703. Ed. J.S. Farlow and Clare Swanson, 118-136. American Society for Testing and Materials.
249. Troxell, George Earl, Harmer E. Davis, and Joe W. Kelly. 1968. Composition and properties of concrete. 2nd ed., McGraw-Hill Civil Engineering Series. New York: McGraw-Hill Book Company.
250. TRW, Inc. April 1978. At-sea incineration of Herbicide Orange onboard the M/T Vulcanus. EPA report no. 530/SW-155c. NTIS no. PB-278 816. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
251. Tucker, Richard E., Young, Alvin L., and Gray, Alan P., eds. 1982. Human and environmental risks of chlorinated dioxins and related compounds. Environmental Science Research, vol. 26. Proceedings of an international symposium on chlorinated dioxins and related compounds, Oct. 25-29, 1981. Arlington, VA. New York: Plenum Press.
252. U.S. Army Corps of Engineers. November 1983. Technical Assistance to United States Environmental Protection Agency for Times Beach, Missouri: Report on Advance Preparation/Emergency Response. St. Louis, Missouri: Department of the Army, St. Louis District, Corps of Engineers.
253. U.S. Army Corps of Engineers. June 1984. Technical Assistance to United States Environmental Protection Agency for Times Beach, Missouri: Flood Control Study. St. Louis, Missouri: Department of the Army, St. Louis District, Corps of Engineers.
254. U.S. Army Engineer Waterways Experiment Station. July 1979. Survey of solidification/stabilization technology for hazardous industrial wastes. Prepared for Municipal Environmental Research Lab, Cincinnati, Ohio. EPA report no. EPA-600/2-79-056. NTIS no. PB-299 206. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
255. U.S. Army Engineer Waterways Experiment Station. September 1980. Guide to the disposal of chemically stabilized and solidified waste. EPA report no. SW-872. Cincinnati, Ohio: Municipal Environmental Research Laboratory, Office of Research and Development, U.S. EPA.
256. U.S. considers burying deadly residues. 1976. The Sacramento Bee (Sept. 1).
257. U.S. EPA. 1975. Hazardous waste disposal damage reports. EPA report no. SW-151.2. Cincinnati, Ohio: Solid Waste Information, U.S. EPA.
258. U.S. EPA. 1978. Burning waste chlorinated hydrocarbons in a cement kiln. Prepared for the U.S. EPA. EPA report no. EPA/530/SW-147C. Montreal, Quebec, Canada: Environmental Protection Service. NTIS report no. PB-280 118. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.

259. U.S. EPA. 1980. Comprehensive environmental response, compensation, and liability act of 1980. Public Law 96-510.
260. U.S. EPA. 1980. Innovative and alternative technology assessment manual. EPA-430/9-78-009. Washington, D.C.: Office of Water Program Operations.
261. U.S. EPA. June 1981. PCB disposal by thermal destruction. EPA Report no. 906/9-82-003. NTIS no. PB-82-241 860. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
262. U.S. EPA. 1982. Handbook for remedial action at waste disposal sites. EPA Report no. 625/6-82-006. Cincinnati, Ohio: Municipal Environmental Research Laboratory, Office of Environmental Engineering and Technology, Office of Research and Development, U.S. EPA.
263. U.S. EPA. 1982. National oil and hazardous substances contingency plan. Final Rule. 40 CFR, Part 300.
264. U.S. Maritime Administration. 1980. Report of the Interagency Ad Hoc Work Group for the Chemical Waste Incinerator Ship Program. EPA report no. MA-SC-700-81022. NTIS report no. PB81-112849. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
265. VanNess, G.G., et al. 1980. Tetrachlorodibenzo-p-dioxins in chemical wastes, aqueous effluents, and soils. Chemosphere 9:553-563. Also, Publication no. 0045-6535/80/0901-0553, Pergamon Press Ltd., Great Britain.
266. Vermont Public Interest Research Group. 1977. Dioxins in Vermont. Summary of a report by David White entitled Pentachlorophenol and the chlorophenoxy herbicides in Vermont.
267. Veterans Administration. 1981. Review of literature on herbicides, including phenoxy herbicides and associated dioxins. Volume 1, Analysis of literature, and Volume 2, Annotated bibliography. Prepared by JRB Associates, McLean, Virginia. Washington, D.C.: Veterans Administration.
268. Walsh, John. 1977. Seveso: The questions persist where dioxin created a wasteland. Science 197(Sept. 9):1064-67.
269. Walt, Dorothy. 1976. Public affairs statement for the California Solid Waste Management Board regarding the proposed disposal of TCDD in a West Covina, California, landfill.
270. Ward, Claudia, and Fumio Matsumura. 1977. Fate of 2,4,5-T contaminant, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic environments. Prepared for the Office of Water Research and Technology, Washington, D.C. Report no. WIS-WRC-77-01; W7705170; OWRTA-058-WIS(1). NTIS no. PB-264 187. Springfield, Virginia: National Technical Information Service, U.S. Department of Commerce.
271. Weitzman, Leo. 1983. Cement kilns as hazardous waste incinerators. Environmental Progress 2(1):10-14.
272. Westinghouse opens plasma torch facility. 1983. The Hazardous Waste Consultant (November-December):pp(1)23-(1)24.
273. Whiteside, Thomas. 1979. The pendulum and the toxic cloud: The course of dioxin contamination. New Haven and London: Yale University Press.

274. Wilhelmi, A.R. and P.V. Knopp. 1979. Wet air oxidation: An alternative to incineration. Chemical Engineering Progress (August):46-52.
275. Wilkinson, R.R., G.L. Kelso, and F.C. Hopkins. 1978. State-of-the-art report: Pesticide disposal research. EPA report no. 600/2-78-183. Cincinnati, Ohio: Municipal Environmental Research Laboratory, Office of Research and Development, U.S. EPA. Available through the National Technical Information Service, Springfield, Virginia. Also in Disposal and decontamination of pesticides, ed. M.V. Kerred, 73-80. American Chemical Society Symposium Series 73. Washington, D.C.: ACS.
276. Wipf, H.K. et al. n.d. Field trials on photodegradation of TCDD on vegetation after spraying with vegetable oil. In Dioxin: Toxicological and chemical aspects, ed. F. Cattabeni, A. Cavallaro, and G. Galli, 201. New York: SP Medical & Scientific Books, Spectrum Publications, Inc.
277. Wipf, H.K., and J. Schmid. 1983. Seveso: An environmental assessment. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 255-274. Proceedings, international symposium on Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.
278. Wong, Anthony S. and Donald G. Crosby. n.d. Decontamination of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) by photochemical action. In Dioxin: Toxicological and chemical aspects, ed. F. Cattabeni, A. Cavallaro, and G. Galli, 185. New York: SP Medical & Scientific Books, Spectrum Publications, Inc.
279. Yezzi, James J. Jr., et al. 1982. The EPA-ORD mobile incineration system. Reprinted from the 1982 National Processing Conference. New York. ASME.
280. Yosim, S.J., K.M. Barclay, and L.F. Grantham. 1978. Destruction of pesticides and pesticide containers by molten salt combustion. In Disposal and decontamination of pesticides, ed. M.V. Kerred, 119-128. American Chemical Society Symposium Series 73. Washington, D.C.: ACS.
281. Young, Alvin, Charles E. Thalken, and William E. Ward. 1975. Studies of the ecological impact of repetitive aerial applications of herbicides on the ecosystem of test area C-52A, Elgin AFB, Florida. Prepared for the Air Force Armament Laboratory (DLV), Armament Development and Test Center, Elgin Air Force Base, Florida. Report AFATL-TR-75-142. Springfield, VA: National Technical Information Service, U.S. Department of Commerce.
282. Young, A.L., C.E. Thalken, and D.D. Harrison. 1981. Persistence, bioaccumulation, and toxicology of TCDD in an ecosystem treated with massive quantities of 2,4,5-T herbicide. Proceedings, West. Soc. Weed Sci 34:70-77.
283. Young, A.L. 1983. Long-term studies on the persistence and movement of TCDD in a natural ecosystem. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 173-189. Proceedings of an international symposium on Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.

284. Zack, Judith A., and William R. Gaffey. 1983. A mortality study of workers employed at the Monsanto Company plant in Nitro, West Virginia. In Human and environmental risks of chlorinated dioxins and related compounds, ed. Richard E. Tucker, Alvin L. Young, and Allan P. Gray, 575-591. Proceedings of an international symposium on Chlorinated Dioxins and Related Compounds, October 25-29, 1981, Arlington, VA. New York: Plenum Press.
285. Zirschky, J. and D. Harris. 1982. Cleaning up farm site 1. Pollution Engineering (July):19-23.
286. Zone II remedial planning/field investigation team management plan. April 1983. Prepared by CH2M HILL, Reston, VA, for U.S. EPA, Washington, D.C.

/CVR96/004

DRAFT

Appendix B
RISK ASSESSMENT OF THE NO ACTION ALTERNATIVE

DRAFT

Section 1 INTRODUCTION

1.1 OBJECTIVES

The objective of this assessment is to evaluate the actual and potential health and environmental effects if no remedial action is taken at hazardous waste sites. This section evaluates the consequences if no remedial action is undertaken at the sanitary and storm sewers of the Love Canal Emergency Declaration Area (EDA), Black, Bergholtz and Cayuga Creeks, and at the 102nd Street outfall in the Niagara River (Figure 1-1). This report makes no attempt to address the habitability of the EDA.

1.2 PREVIOUS HEALTH ASSESSMENT

In 1981, the U.S. Department of Health and Human Services (DHHS) obtained written opinions from eleven non-federal expert consultants on the health implications of the data obtained by the U.S. Environmental Protection Agency (USEPA) in its 1980 chemical testing at Love Canal. The letter from the DHHS Centers for Disease Control to the consultants asked them to review the data with respect to whether (1) the concentrations were significantly different from levels found in other areas of Niagara Falls, (2) the concentrations represent levels that could cause acute or chronic adverse health effects in people in the area beyond the "usual residential conditions" in the Niagara Falls area, (3) the data were sufficient to make a judgment, and (4) one could conclude the area is not habitable.

The consultants met for one day and were presented with a condensed form of the EPA monitoring data. The experts' opinions on the storm sewer and creek chemical concentrations were summarized as follows:

"Consultants agree that levels of chemicals detected in storm sewers and in Area 11 (Canal itself and the first two rings of houses surrounding the canal) exceed acceptable levels and represent a potential for increased health risk if remedial actions are not pursued and if human access is not controlled."

"Any judgment regarding the future habitability of the Love Canal area rests on two important requirements. The first reservation is that appropriate measures must be taken to clean up the obvious contamination of local storm sewers and their drainage tracts. Second, the security of Area 11 must be reevaluated to guarantee permanent containment of chemicals in the dump."
(Heath, et al., 1981).

Although the U.S. Congress Office of Technology Assessment criticized the general conclusions of the 1982 EPA monitoring report and the DHHS statement on habitability, no criticisms were directed at the identified need to clean the storm sewers and drainage tracts. This need was restated by DHHS on July 13, 1982. (Heath et al., 1982). The New York Department of Health has also supported this position (Huffaker, 1984).

1.3 FOCUS OF THIS STUDY

The area included in this evaluation is predominantly bounded on the east by the eastern boundary of the EDA (102nd Street), Bergholtz Creek on the north, Cayuga Creek on the west and the Niagara/Little Niagara Rivers on the south. Some discussion will include areas west of the Cayuga Creek.

On the basis of frequency of observation in the Malcolm Pirnie (1983) report, which included sampling of the sewers and creeks, and their toxicological properties. The following chemicals were chosen as target chemicals for assessment in this report: 2,3,7,8-tetrachlorodibenzo-p-dioxin, or TCDD, isomers of hexachlorocyclohexane, chlorinated benzenes, toluene, arsenic, cadmium and thallium.

Section 2 summarizes the concentrations and locations of these contaminants in the sewers, creeks and 102nd Street outfall. Section 3 describes the potential chemical migration of these chemicals in the sewers and creeks and the environmental fate of the target chemicals. In Section 4, a qualitative exposure and public health assessment is described. Attachment A details the detection limits associated with the 1980 EPA Monitoring Data. Attachment B provides a detailed listing of potential receptors in the Love Canal area. Appendix C discusses the toxicological and chemical properties of the target chemicals.

Other studies in addition to the 1980 EPA monitoring effort have been conducted. More recently, Malcolm Pirnie performed an extensive sampling program of the area sewers, creeks and 102nd Street outfall in January 1983. The objective of this report was to review available contaminant data and to examine potential human exposure to assist the U.S. Environmental Protection Agency in its determination of the risks associated with area contamination.

Section 2
SAMPLING RESULTS FOR SANITARY SEWERS, STORM
SEWERS, AND SURFACE WATERS

2.1 INTRODUCTION

This chapter summarizes chemical data collected at Love Canal for sanitary sewers, storm sewers, and surface waters (Black, Bergholtz and Cayuga Creeks, and the Little Niagara and Niagara Rivers). Nine sources were used in preparing this report:

- o Malcolm Pirnie, Inc. October 1983. Site Investigation and Remedial Action Alternatives-Love Canal. Prepared for the New York State Department of Environmental Conservation. Samples were taken in January and March 1983.
- o Office of Research and Development, U.S. Environmental Protection Agency. May 1982. Environmental Monitoring at Love Canal, Volumes 2 and 3. Samples were taken between August and October 1980.
- o Smith, M. P., O'Keefe, K. M., Aldous, D. R., Hilker, and J. E. O'Brien. 1983. "2, 3, 7, 8-tetrachlorodibenzo-p-dioxin in Sediment Samples from Love Canal Storm Sewers and Creeks." Environmental Science and Technology, Volume 17, No. 1. Samples were collected in 1979 and 1980 by the New York Department of Health.
- o Environment Canada and Ontario Ministry of the Environment. Environmental Baseline Report of the Niagara River. June 1980. Samples were taken in 1979.
- o Environment Canada and Ontario Ministry of the Environment. Environmental Baseline Report of the Niagara River--November 1981 Update. November 16, 1981. Samples were taken in 1981.
- o New York Department of Health. May 1984. Bergholtz Creek Preliminary TCDD Data. Samples were taken April 12, 1984.
- o New York Department of Health. February 8, 1985. Bergholtz Creek samples taken in July, 1984.
- o E. J. Kuzin, New York Department of Health. Cayuga Creek Dioxin Sampling of Fish and Sediment. Samples were taken in 1984.

- o Suns, K., G.R. Craig, G. Crawford, G.A. Rees, H. Tosine, and J. Osborne, "Organochlorine Contaminant Residues in Spottail Shiners (Notropis hudsonius) from the Niagara River." J. Great Lakes Research, Vol. 9, pp.335-340, 1983. Samples were taken in 1980 and 1981.
- o D.L. Stalling, Fish and Wildlife Service, Columbia, Missouri. Letter to L. Skinner, New York State Department of Environmental Conservation, January 18, 1982. Carp samples taken on June 5, 1980.

These data sources were selected because they represent relatively recent conditions at Love Canal. A detailed quality assurance assessment of these data was outside the scope of this study.

A total of 114 chemicals were identified at Love Canal in these five studies. Of these, eight compounds or groups of compounds were selected for consideration in greater detail in this report. These include 2,3,7,8-TCDD (dioxin), lindane and other BHC isomers, chlorinated benzenes, toluene, arsenic, cadmium, and thallium. These compounds were selected based on their toxicological properties and on their frequency of detection in chemical analyses of the Love Canal area. Other chemicals were identified in only a small number (less than ten) of samples.

The following sections of this chapter describe the distribution and abundance of these chemicals of interest with respect to sanitary sewer sediment and liquid samples, storm sewer sediment and liquid samples, and surface water sediment, liquid, and biota samples. The locations of sampling sites for the study used in this assessment are shown in Figures 2-1 to 2-5. Figure 2-1 shows sanitary sewer manhole locations sampled by Malcolm Pirnie. Figure 2-2 shows storm sewer manholes sampled by Malcolm Pirnie, EPA-ORD, and Smith, et al. Figures 2-3 to 2-5 show sampling sites in surface waters including Black, Bergholts, and Cayuga Creek and the Niagara River. These were sampled by EPA-ORD, Malcolm Pirnie, and the New York State Department of Health.

2.2 SANITARY SEWERS

EPA-ORD did not sample the sanitary sewers and, therefore, all data reported here are from the Malcolm Pirnie report. Figures 2-6 through 2-8 summarize the locations of the detected concentrations, and Table 2-1 summarizes the chemical concentrations found in the sewer sediments (wet weight concentrations for Malcolm Pirnie data). There were only four detected concentrations of TCDD (from 19 samples), ranging from 2.5 to 30 $\mu\text{g/kg}$. Concentrations of other organic

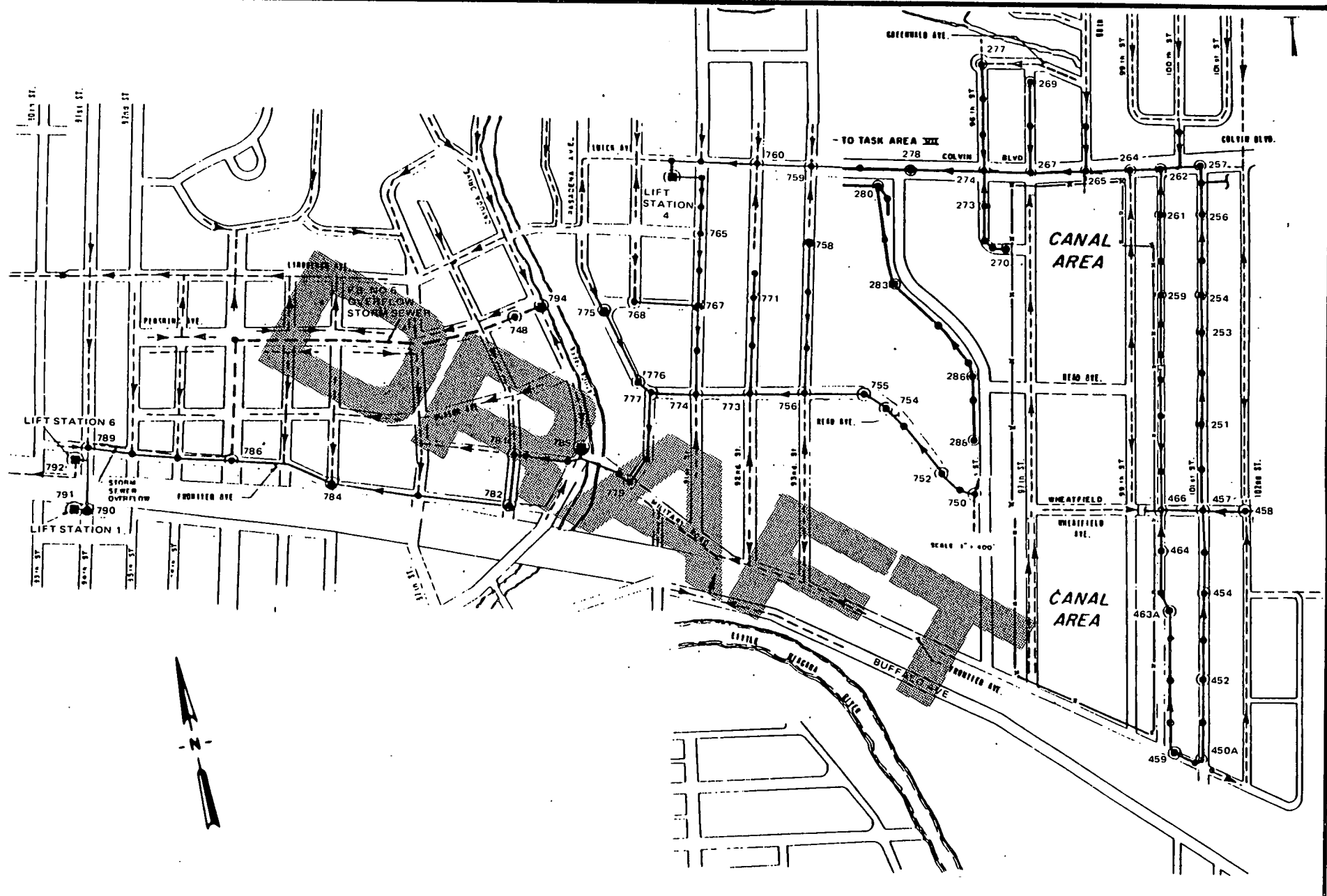


Figure 2-1
Sanitary Sewers of the
Love Canal Area



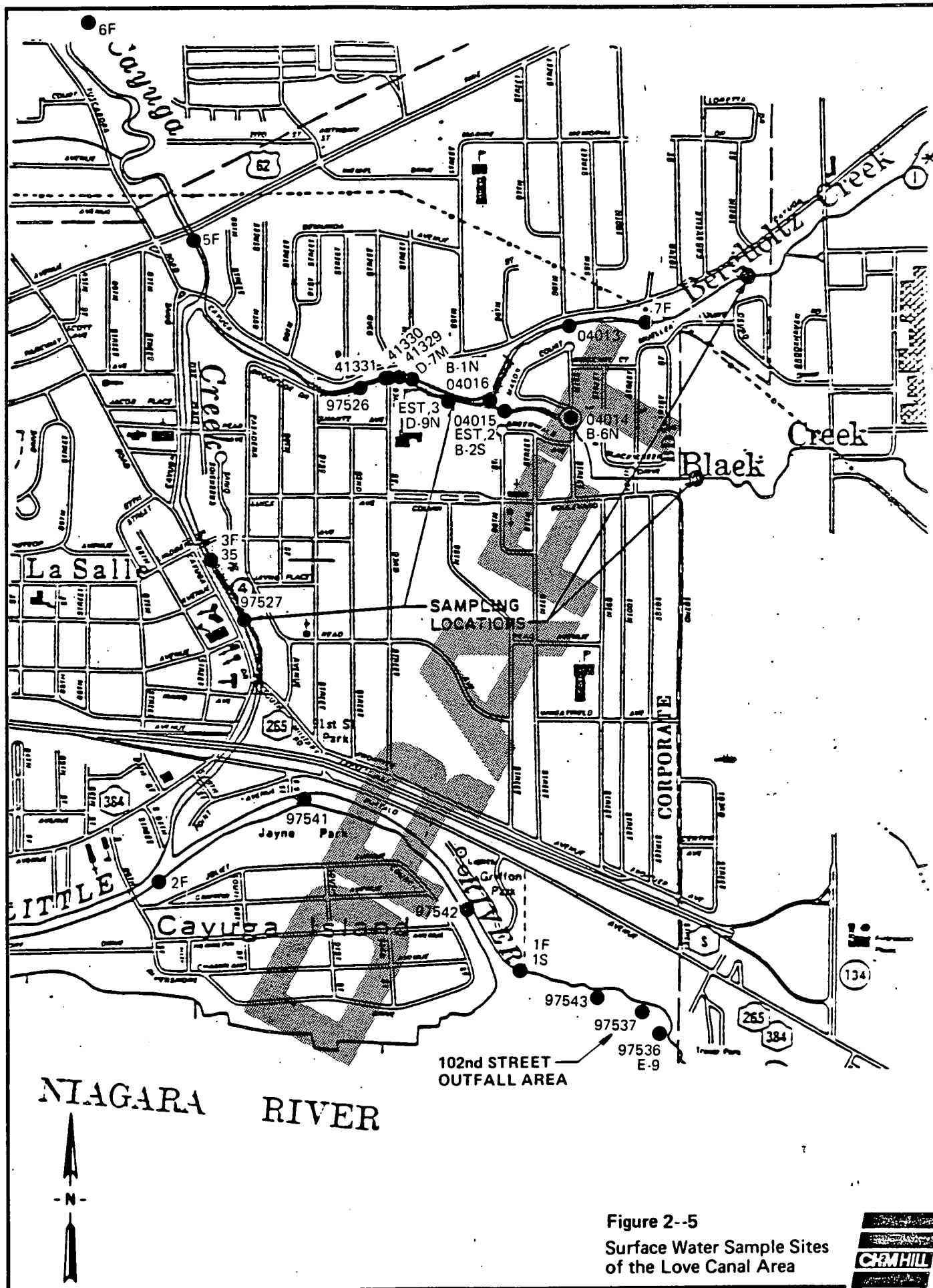


Figure 2--5
Surface Water Sample Sites
of the Love Canal Area



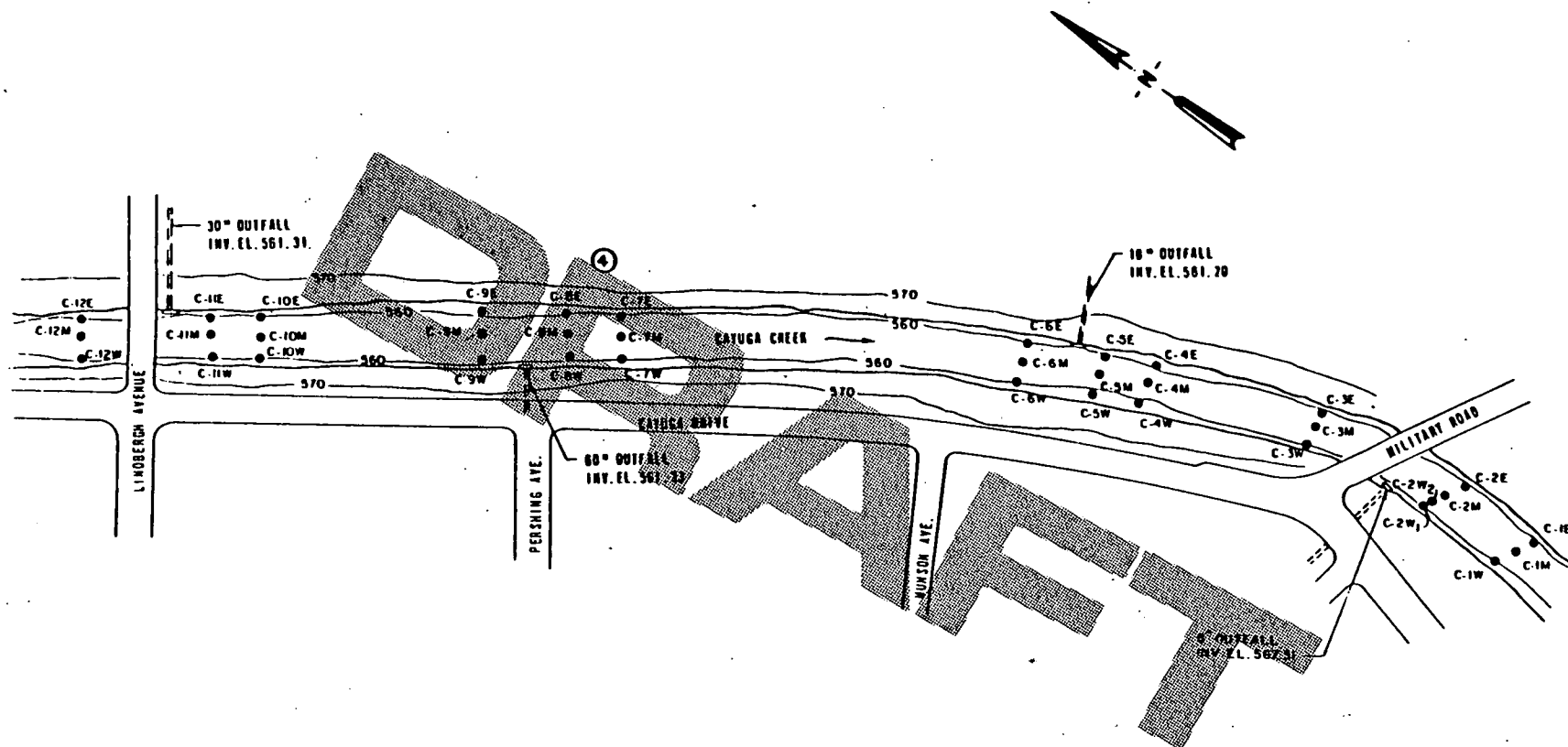
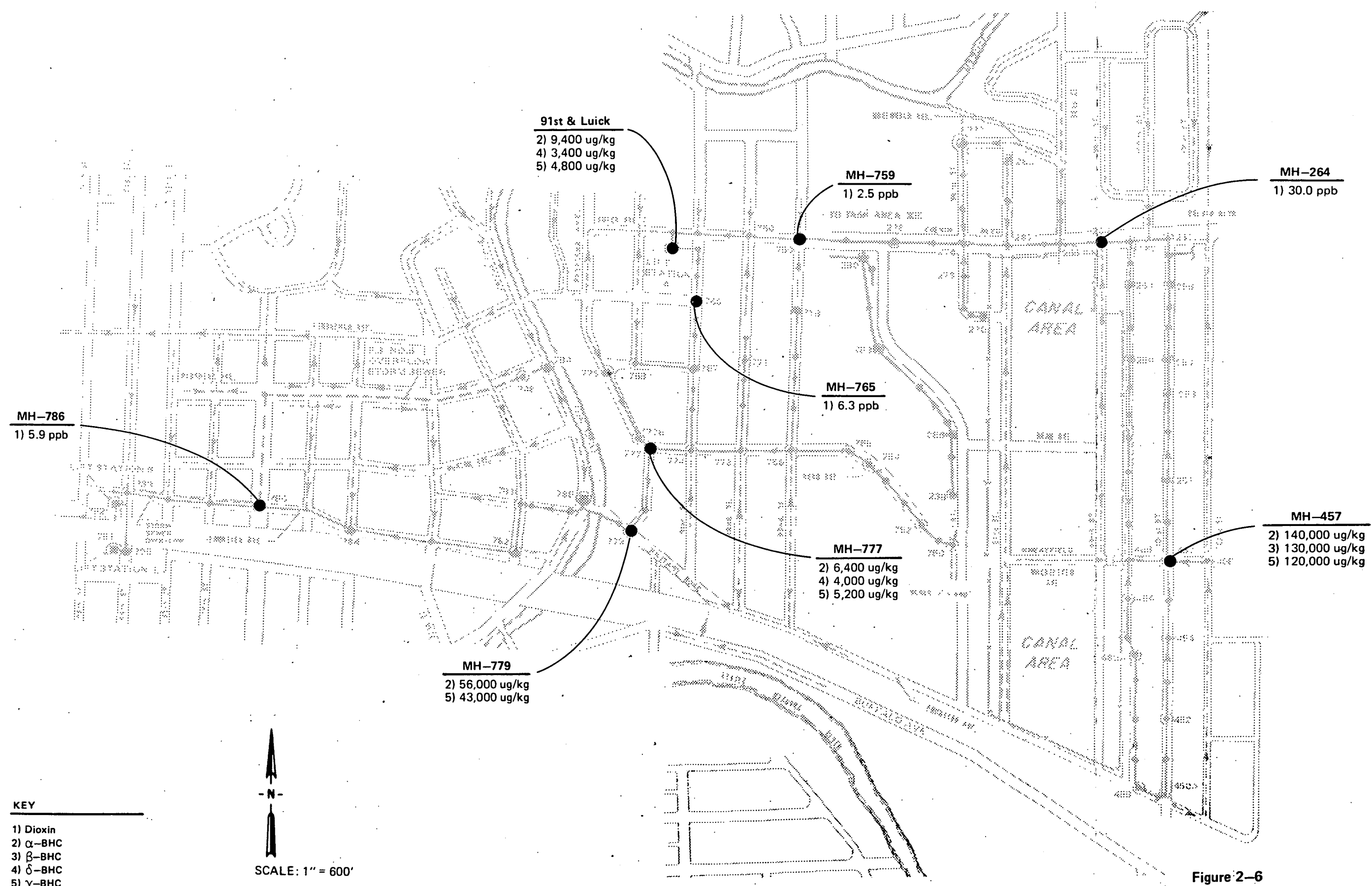
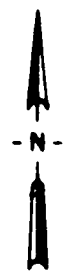
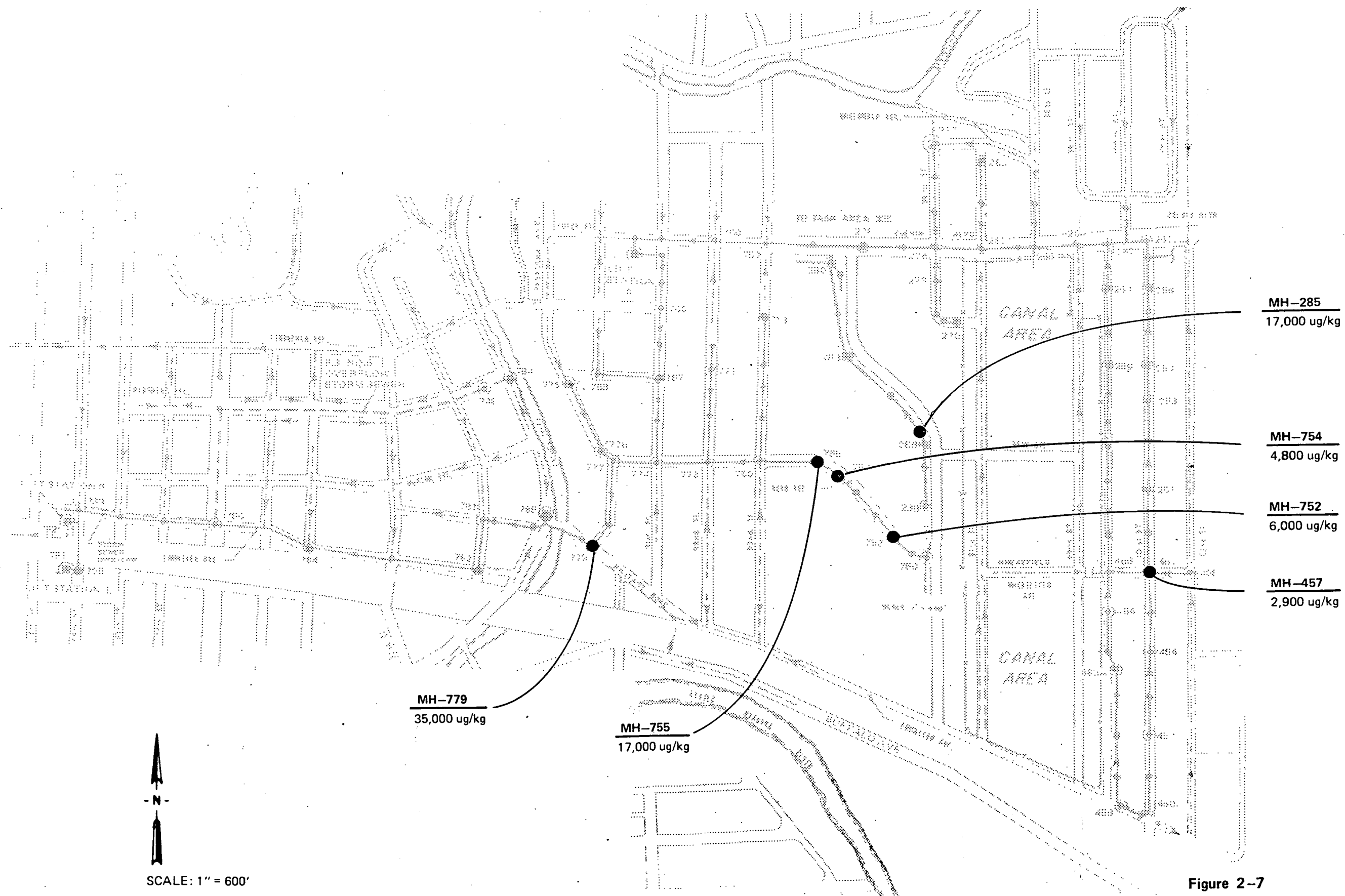


Figure 2-4
 Surface Water Sample Sites
 of the Love Canal Area
 Cayuga Creek



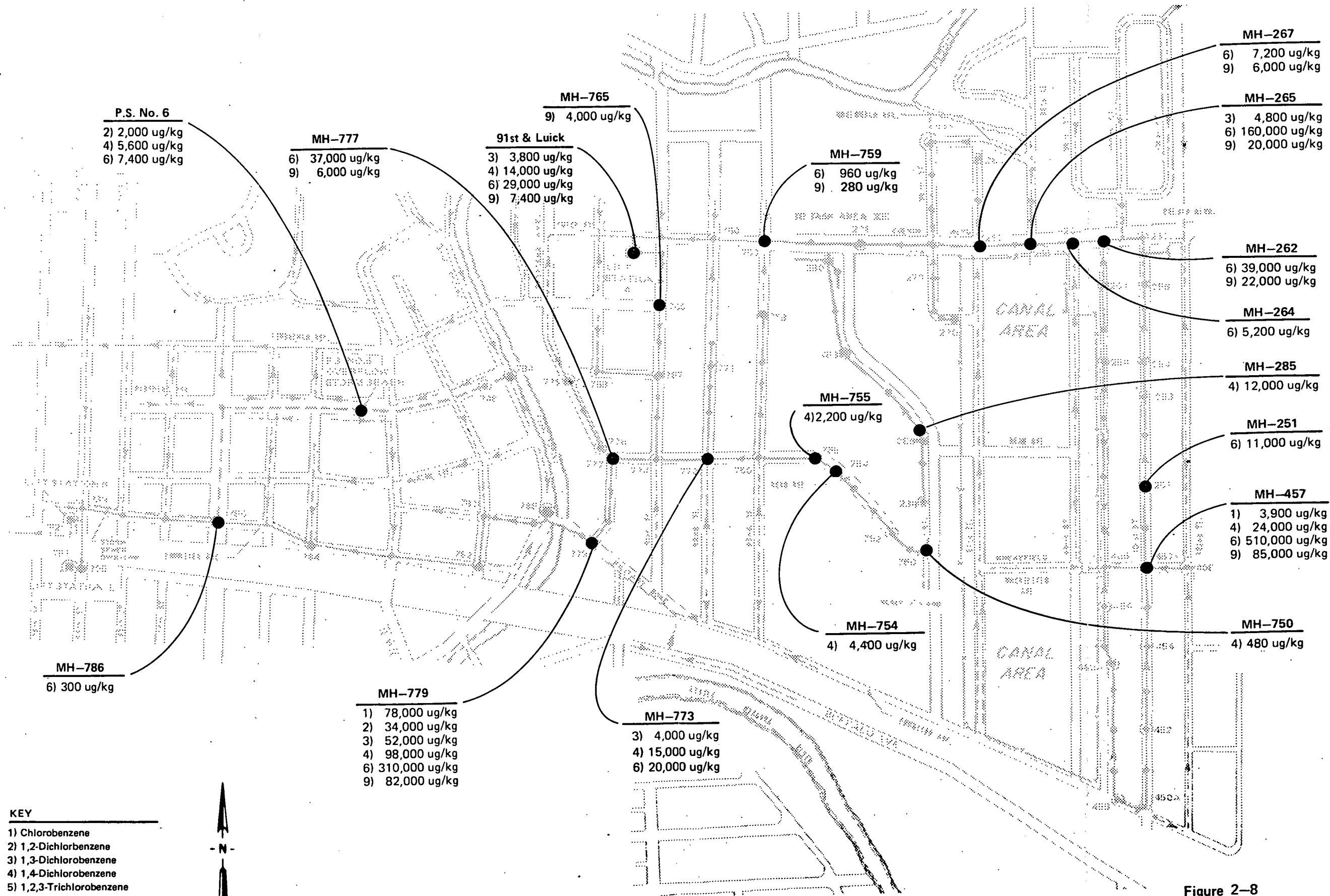




SCALE: 1" = 600'

Figure 2-7
Sanitary Sewer Sediment Samples
Containing Toluene





- KEY**
- 1) Chlorobenzene
 - 2) 1,2-Dichlorobenzene
 - 3) 1,3-Dichlorobenzene
 - 4) 1,4-Dichlorobenzene
 - 5) 1,2,3-Trichlorobenzene
 - 6) 1,2,4-Trichlorobenzene
 - 7) 1,2,3,4-Tetrachlorobenzene
 - 8) 1,2,4,5-Tetrachlorobenzene
 - 9) Hexachlorobenzene

SCALE: 1" = 600'

Figure 2-8
 Sanitary Sewer Sediment Samples
 Containing Chlorinated Benzenes

CH2M HILL

Table 2-1
Summary of Chemical Concentrations in the Sanitary Sewers
Malcolm Pirnie Data Taken in 1983

Chemical	Number of Samples	Nondetect Samples		Concentration (µg/kg)		
		Number	Detection Limit (µg/kg)	Maximum ^a	Mean ^b	Median ^b
TCDD	19	15	0.20-210 ^c	30	3	1
Hexachlorobenzene	31	24	200-20,000	85,000	10,000	4,000
α-BHC	31	28	200-20,000	140,000	9,800	4,000
β-BHC	31	31	200-20,000	20,000	4,100	4,000
λ-BHC	31	28	200-20,000	130,000	7,800	4,000
Chlorobenzene	31	29	2,000	78,000	4,500	2,000
Toluene	31	25	2,000	35,000	4,300	2,000
Arsenic	28	5	1,000	28,000	6,700	5,300
Cadmium	28	17	1,000	11,000	1,700	1,000
Thallium	28	17	1,000	13,000	2,300	1,000

^a Detected concentrations.

^b Calculated with the detection limit for nondetect samples.

^c One sample had a detection limit of 210 µg/kg. The next highest detection limit was 2 µg/kg.

B-5

chemicals ranged as high as 510,000 $\mu\text{g/kg}$ (1,2,4-trichlorobenzene at station MH-257 in Figure 2-8). In general, the median concentrations were close to the detection limit for all chemicals shown.

Detection of arsenic, cadmium and thallium was generally widespread throughout the sewer sediments. A summary of the concentrations is presented in Table 2-1. For comparison purposes, Table 2-2 presents concentrations of these inorganics for the EDA surface and average soils. As can be seen, the concentrations in the sanitary sewer sediments are about the same as the surface and average soils.

Only three samples of the sewer liquids were taken and all concentrations were reported as nondetects (organic detection limit of 10 $\mu\text{g/L}$ and inorganic detection limit of 1,000 $\mu\text{g/L}$), except for one sample that had reported concentrations of 1,2-dichlorobenzene, 1,4-dichlorobenzene, 1,2,4-trichlorobenzene, and hexachlorobenzene under 1 mg/L each.

2.3 STORM SEWERS

Both Malcolm Pirnie and EPA-ORD took samples in the storm sewers. Locations of detected concentrations are shown in Figures 2-9 through 2-13. The concentrations are summarized in Table 2-3 for the Malcolm Pirnie study and Table 2-4 for the EPA-ORD monitoring study (wet weight concentrations for sediment samples for Malcolm Pirnie data). A comparison of the two tables suggests that the concentrations of TCDD, BHC isomers and hexachlorobenzene have decreased, usually by at least an order of magnitude, in the 2.5 years between the two sampling periods. The maximum reported TCDD concentration went from 650 $\mu\text{g/kg}$ in the 1980 study to 1.9 $\mu\text{g/kg}$ in the 1983 study, and the mean concentration went from 49 $\mu\text{g/kg}$ to 0.82 $\mu\text{g/kg}$ in 1983. These decreased concentrations are consistent with the expected washout of the sediments from sewer water flow following the cutting of the sewer connection from the sewers immediately adjacent to the Canal to the remainder of the EDA. Other explanations for the concentration decrease (e.g., changes in the sampling locations, or analytical technique) cannot be completely ruled out, however. In contrast, concentrations of the volatiles toluene and chlorobenzene have increased in the Malcolm Pirnie data compared to the EPA-ORD monitoring study. Since both studies reported mostly nondetectable concentration for both volatiles, the apparent increase in mean and median concentrations is a reflection of the difference in detection limits. Increases in the median concentrations and the smaller decreases of the BHC isomers in the Malcolm Pirnie data compared to the EPA-ORD study also reflect the differences in detection limits.

Table 2-2
Soil Inorganic Concentrations ($\mu\text{g/kg}$)

Love Canal EDA Surface

Chemical	Maximum	Mean (a)	Median (a)	Average	Range
Arsenic	55000	15000	12,000	7000 (b,c)	1000-93,000 (b)
Cadmium	21000	330	10	350 (d,e)	10-2000 (d)
Thallium	8000	180	100	200 (d,e)	100-800 (d)

(a) Uses detection limit for nondetect concentrations.

(b) U.S. value (Kabata-Pendias and Pendias, 1984).

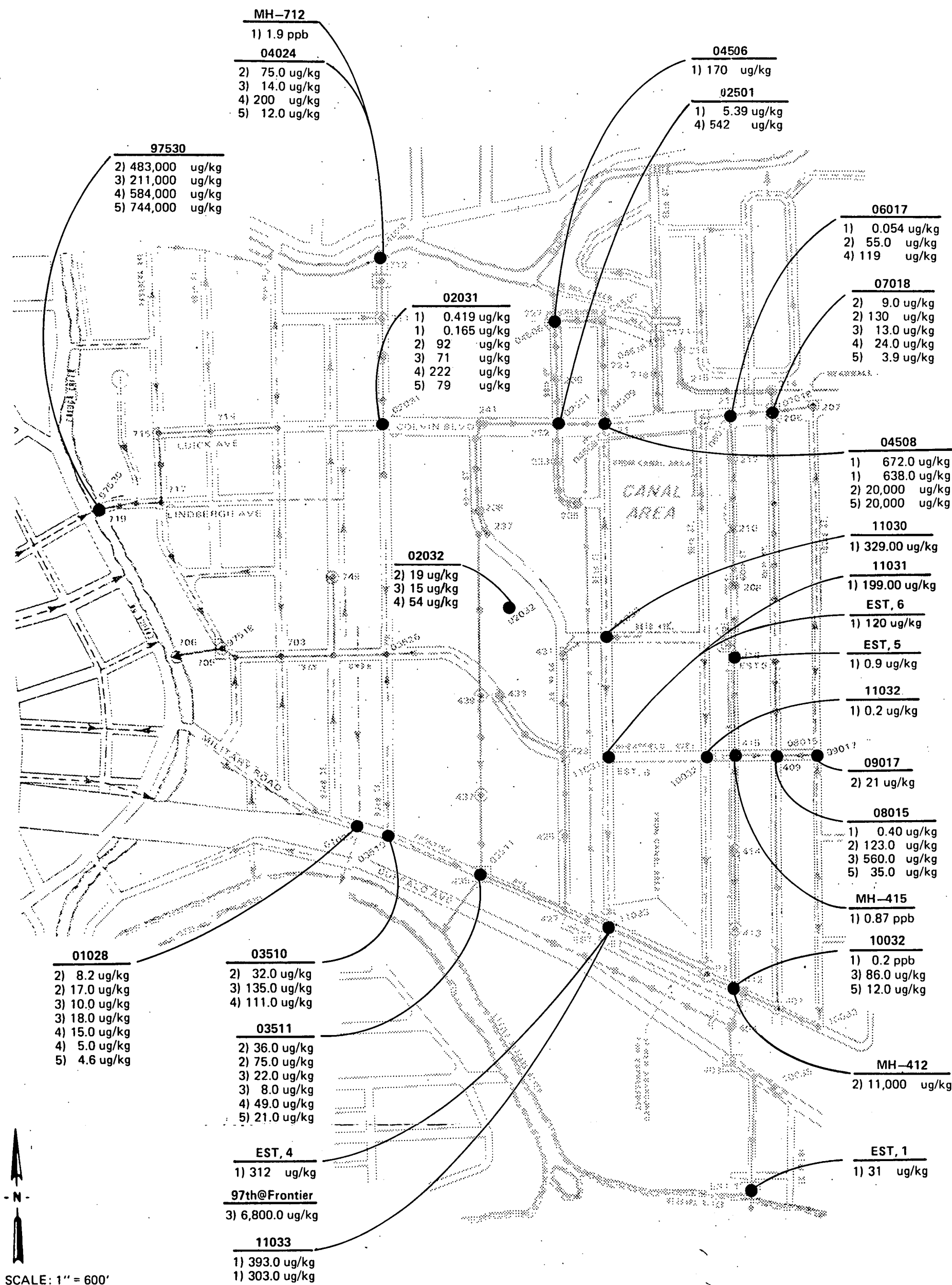
(c) Mean value.

(d) Global value (Bowen, 1979)

(e) Median value.

B-7

COM02/d.501



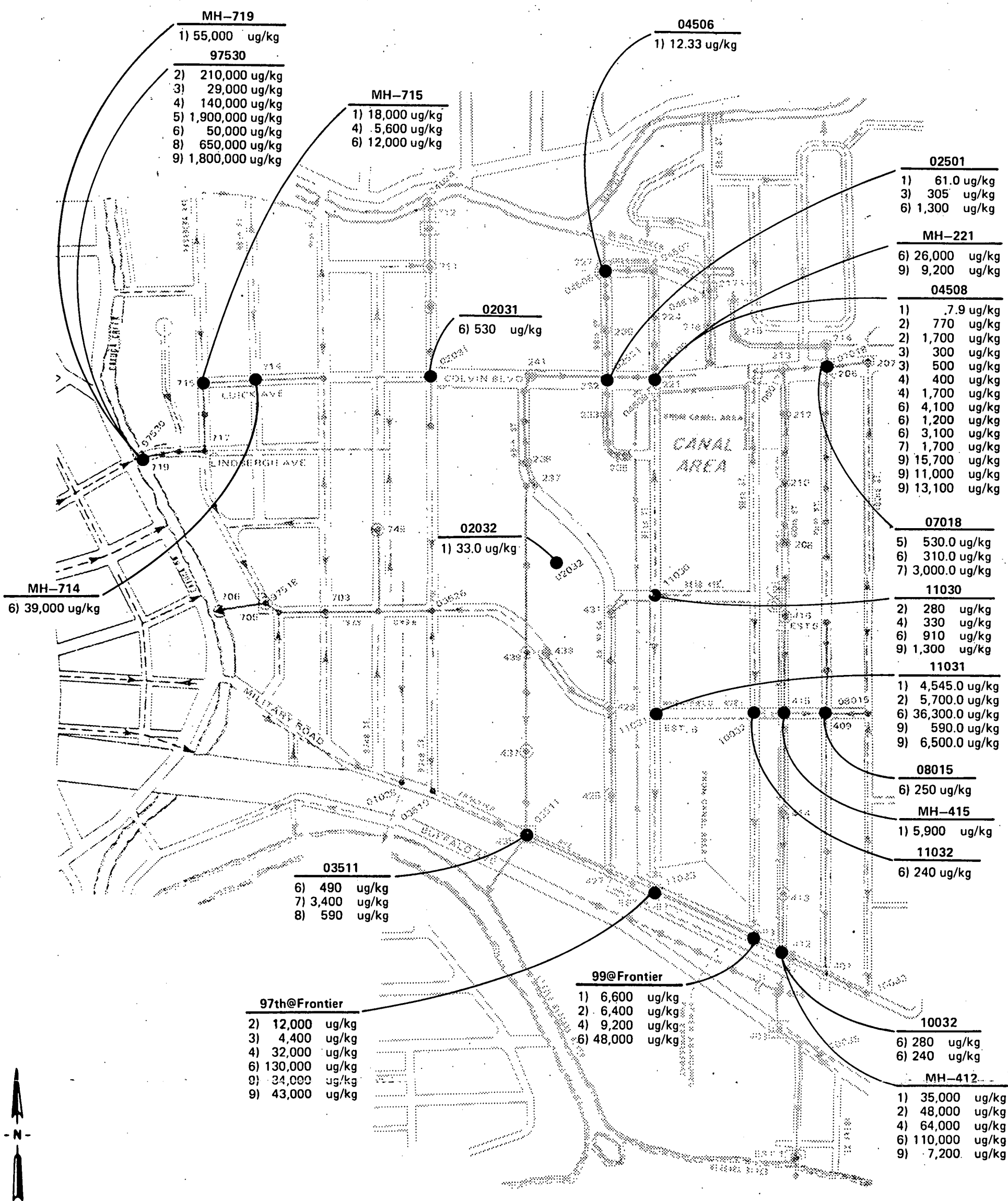
KEY

- 1) Dioxin
- 2) α -BHC
- 3) β -BHC
- 4) δ -BHC
- 5) γ -BHC

Figure 2-9

Storm Sewer Sediment Samples
Containing Dioxin & BHC Isomers

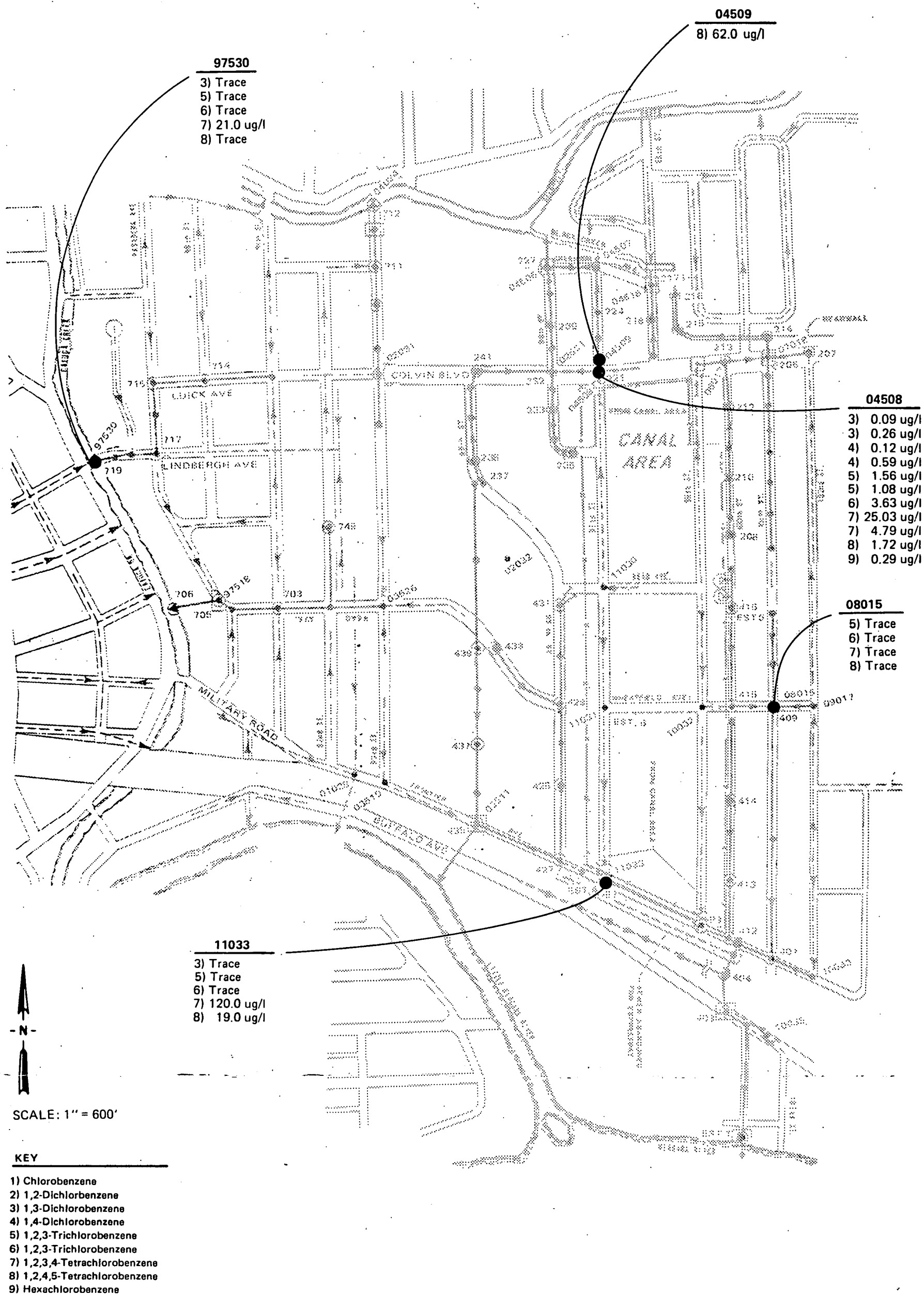




- KEY**
- 1) Chlorobenzene
 - 2) 1,2-Dichlorobenzene
 - 3) 1,3-Dichlorobenzene
 - 4) 1,4-Dichlorobenzene
 - 5) 1,2,3-Trichlorobenzene
 - 6) 1,2,4-Trichlorobenzene
 - 7) 1,2,3,4-Tetrachlorobenzene
 - 8) 1,2,4,5-Tetrachlorobenzene
 - 9) Hexachlorobenzene

Figure 2-11
Storm Sewer Sediment Samples
Containing Chlorinated Benzenes





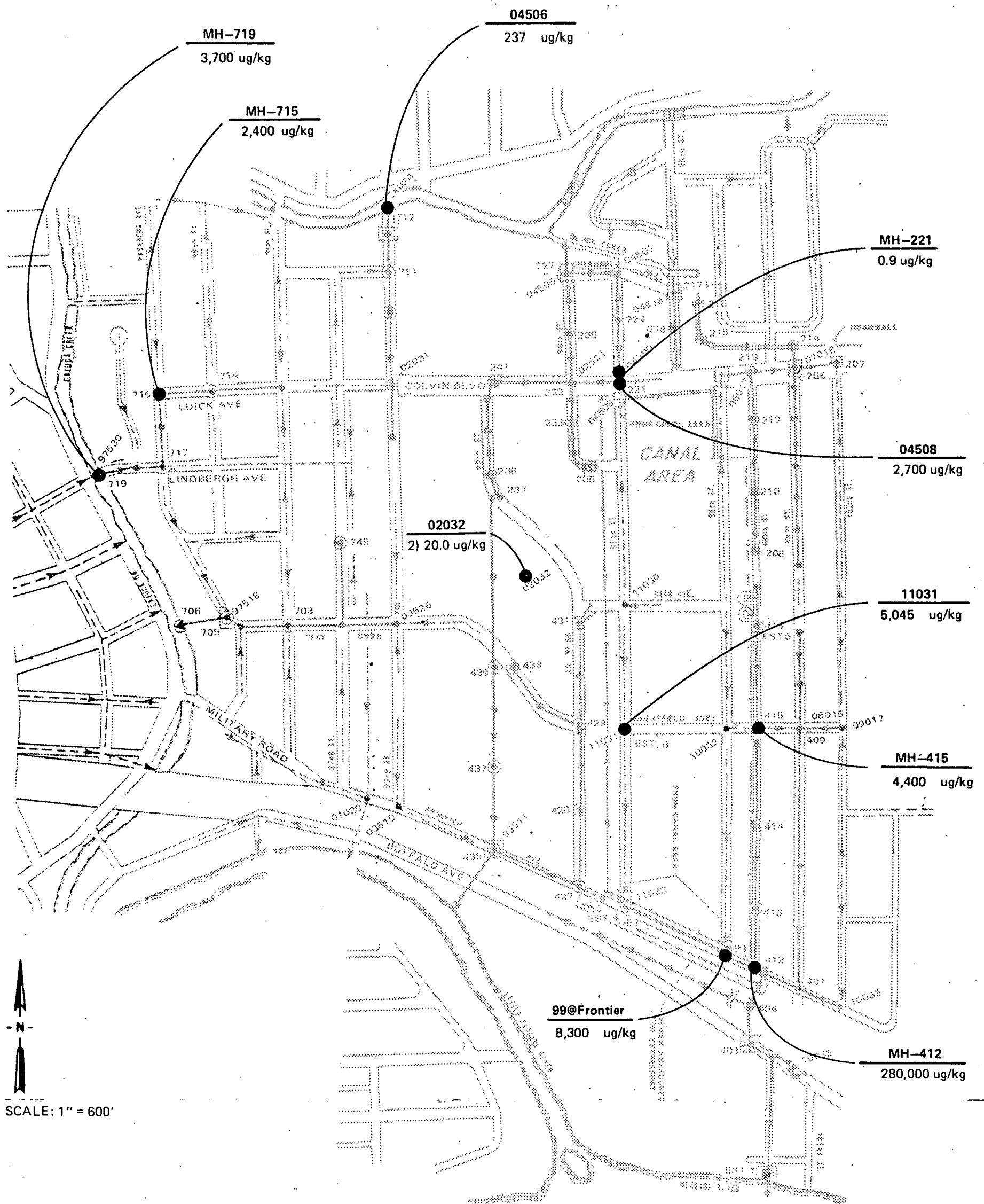


Figure 2-13
Storm Sewer Sediment Samples
Containing Toluene

Table 2-3
Summary of Chemical Concentrations in the Storm Sewers
Malcolm Pirnie Data Taken in 1983

Chemical	Number of Samples	Nondetect Samples		Concentration (µg/kg)		
		Number	Detection Limit (µg/kg)	Maximum (a)	Mean (b)	Median (b)
TCDD	12	10	0.01-3.80	1.9	0.82	0.45
Hexachlorobenzene	20	16	200-28,000	34,000	7,800	4,000
α-BHC	20	19	200-28,000	28,000	6,000	4,000
β-BHC	20	19	200-28,000	28,000	5,800	4,000
λ-BHC	20	20	200-28,000	28,000	5,600	4,000
Chlorobenzene	20	15	2,000	55,000	7,500	2,000
Toluene	20	14	2,000	280,000	16,000	2,000
Arsenic	18	0	1,000	18,000	10,000	10,000
Cadmium	18	13	1,000	1,700	1,100	1,000
Thallium	18	4	1,000	23,000	3,720	4,000

(a) Detected concentration.

(b) Calculated with the detection limit for nondetect samples.

Table 2-4
Summary of Chemical Concentrations in the Storm Sewer Sediment
EPA-ORD Data Taken in 1980

Chemical	Number of Samples	Nondetect Samples		Concentration (µg/kg)		
		Number	Detection Limit (µg/kg)	Maximum (a)	Mean (b)	Median (b)
TCDD	28	18	0.02	650	49	0.02
Hexachlorobenzene	28	24	85	1,800,000	65,000	85
α-BHC	28	15	6	483,000	18,000	6
β-BHC	28	18	6	211,000	7,600	6
λ-BHC	28	20	6	744,000	27,000	6
Chlorobenzene	28	23	7	4,600	220	7
Toluene	28	23	7	5,000	200	7
Arsenic	28	24	20	13,000	1,600	20
Cadmium	28	24	10	2,400	1,000	10
Thallium	28	28	100	100	100	100

(a) Detected concentration.

(b) Calculated with the detection limit for nondetect samples.

The concentrations of the inorganic chemicals remained approximately the same between the two sampling periods, with the difference in the mean and median concentrations being an artifact of the differences in the detection limits between the two studies. The concentrations in the sewers, creeks, and 102nd Street outfall (shown in later tables) are approximately the same. This and the similarity with other soils presented in Table 2-2 suggest that the inorganic chemicals do not originate from the Canal, but are indigenous to the area.

Only two samples of the storm sewer liquids were reported by Malcolm Pirnie and all concentrations were near the detection limit (10 to 20 $\mu\text{g/L}$ for organics and 1000 $\mu\text{g/kg}$ for inorganics), except for reported toluene concentrations of 2300 and 2900 $\mu\text{g/L}$. EPA-ORD took 28 samples of the storm sewer liquids and the highest reported concentration was 120 $\mu\text{g/L}$ (one sample of 1,2,3,4-tetrachlorobenzene) (Figure 2-12).

2.4 CREEKS

Sampling results for the creeks are shown in Figures 2-14 through 2-19, and the concentrations are summarized in Tables 2-5 through 2-7 (wet weight concentrations for sediment samples for Malcolm Pirnie data). No summary tables were prepared for the EPA-ORD data because of the limited number of samples: two sediment samples from Black Creek, three sediment samples from Bergholtz Creek, and one sediment sample from Cayuga Creek. The highest concentrations of TCDD in the sediment were near the confluence of Black and Bergholtz Creeks, where several sampling programs detected concentrations in approximately the same range of concentrations, with a maximum of 45.8 $\mu\text{g/kg}$. Concentrations at points away from this confluence generally approached or were lower than 1 $\mu\text{g/kg}$.

Only two liquid samples were taken by Malcolm Pirnie and only six liquid samples were taken in the EPA-ORD monitoring study from all creeks. Concentrations were reported as non-detects (about 10 $\mu\text{g/L}$ for organics, and less than 1000 $\mu\text{g/L}$ for inorganics). No volatile organics were reported in the creek sediments or water.

2.5 102ND STREET OUTFALL AND THE NIAGARA RIVER

Sampling results are summarized in Figures 2-20 and 2-21 (wet weight concentrations for Malcolm Pirnie data). TCDD was not detected in sixty-two samples taken by Malcolm Pirnie in the Little Niagara River. The concentration summary is shown in Table 2-8. No summary table was prepared for the EPA-ORD study because only five sediment samples were reported.

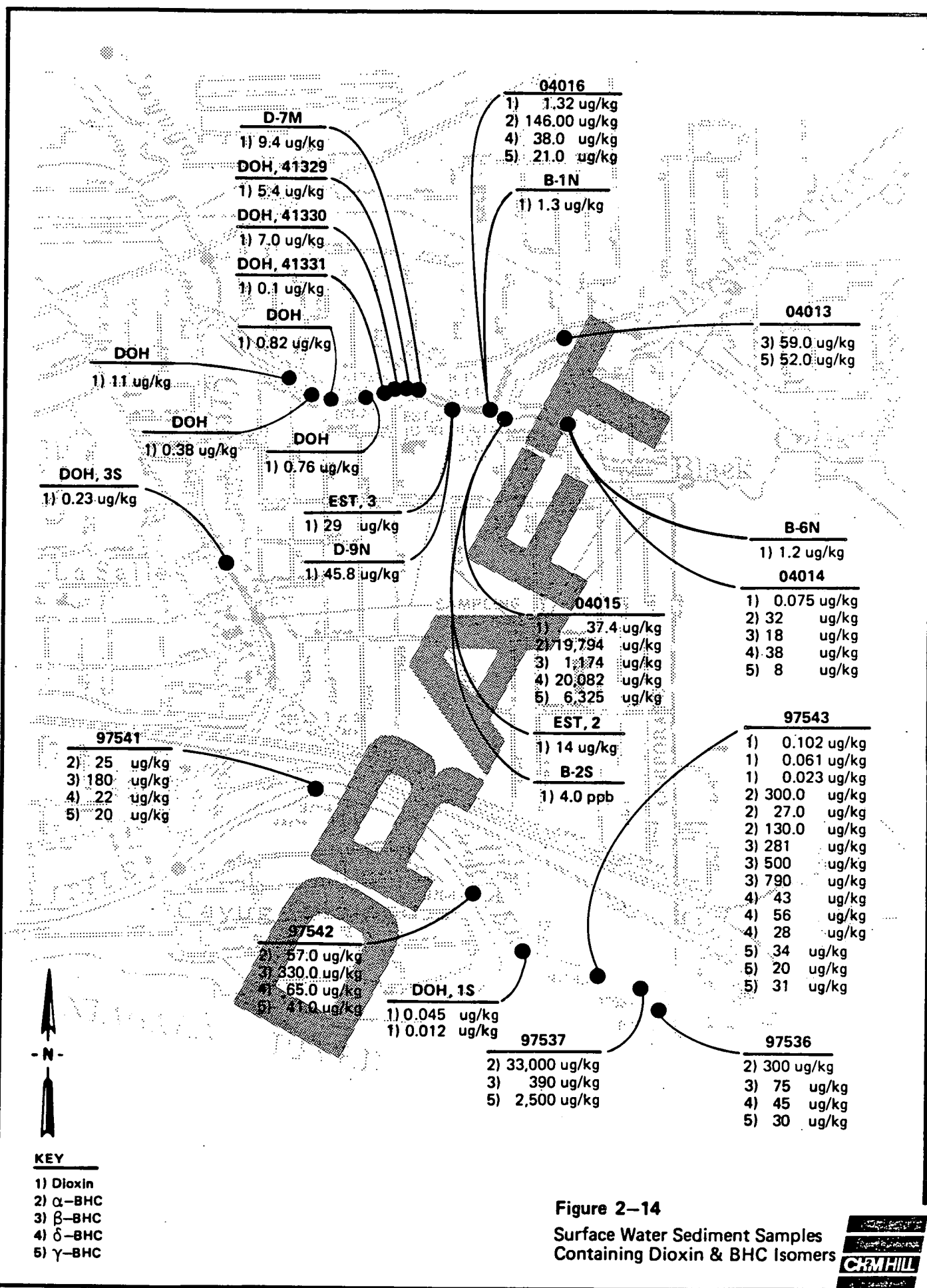


Figure 2-14

Surface Water Sediment Samples
Containing Dioxin & BHC Isomers



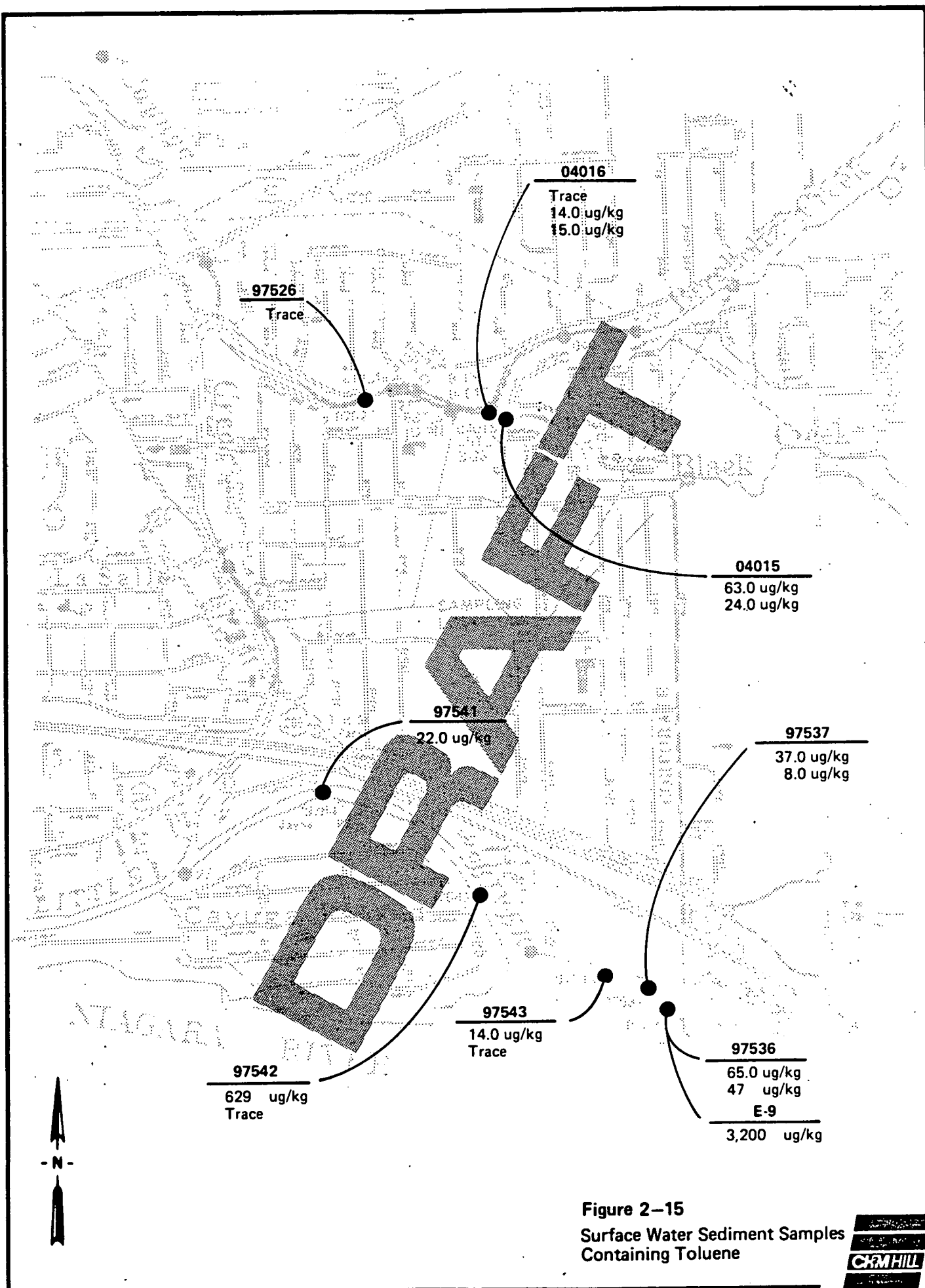
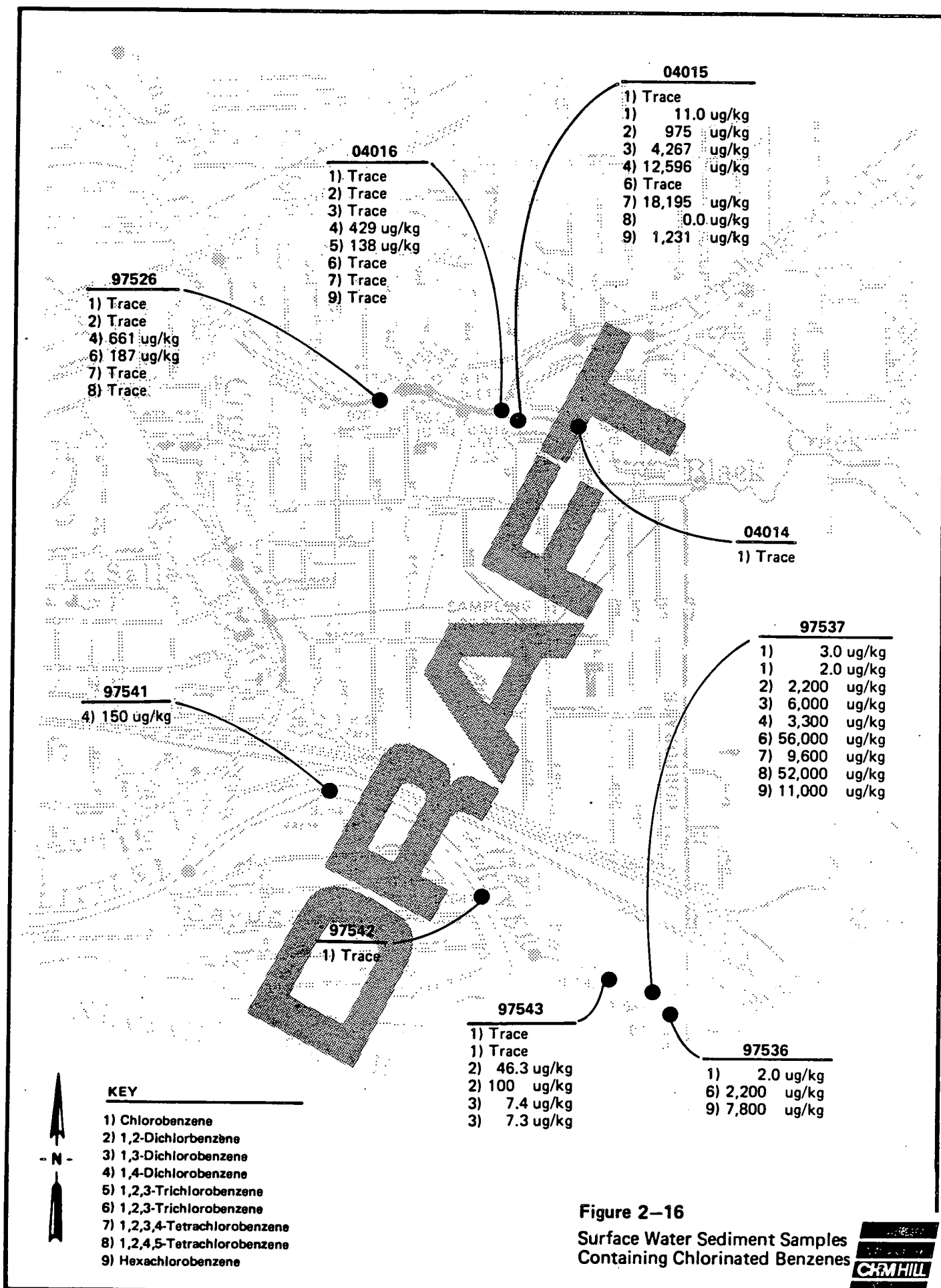


Figure 2-15
Surface Water Sediment Samples
Containing Toluene





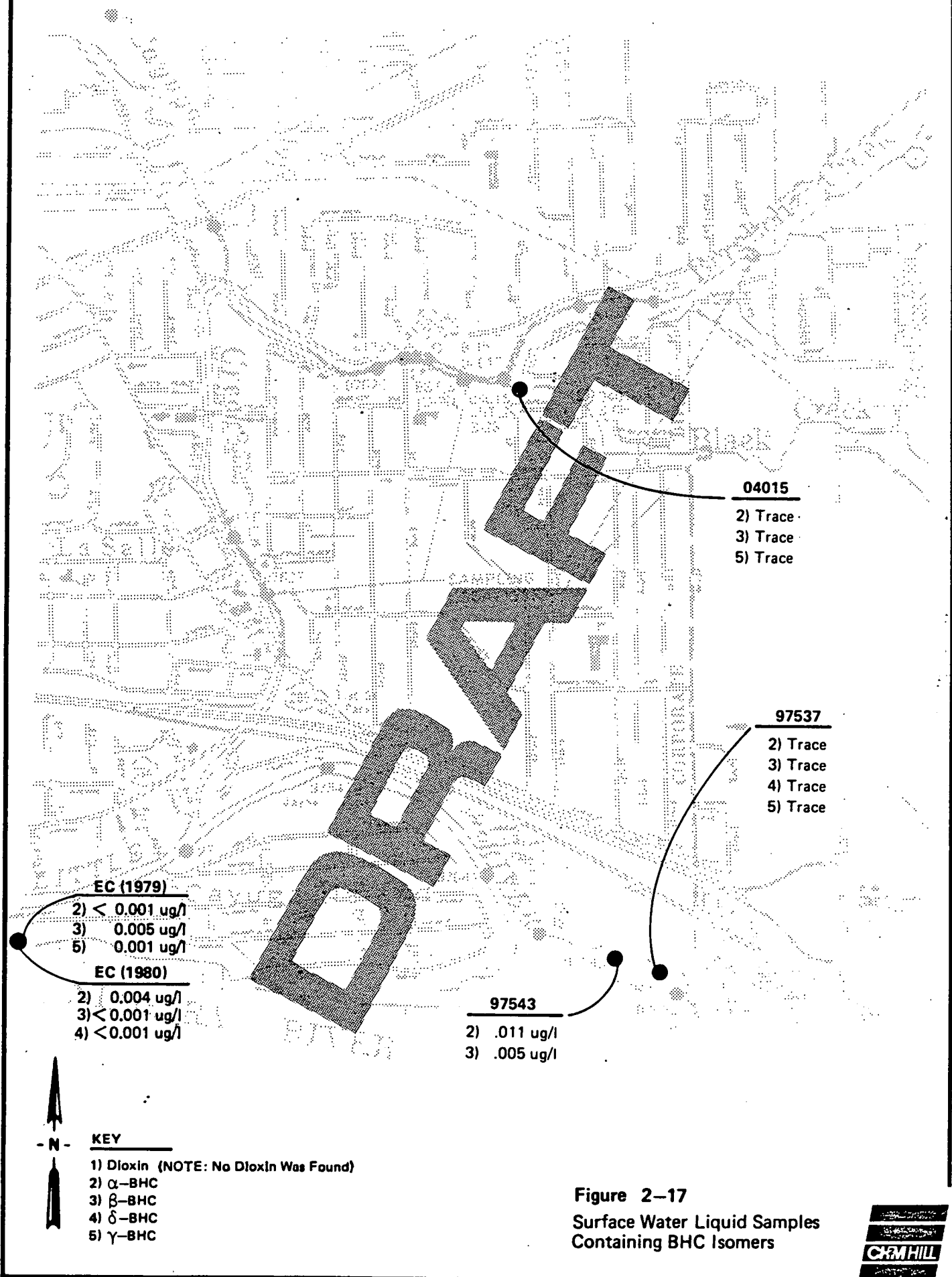


Figure 2-17
 Surface Water Liquid Samples
 Containing BHC Isomers

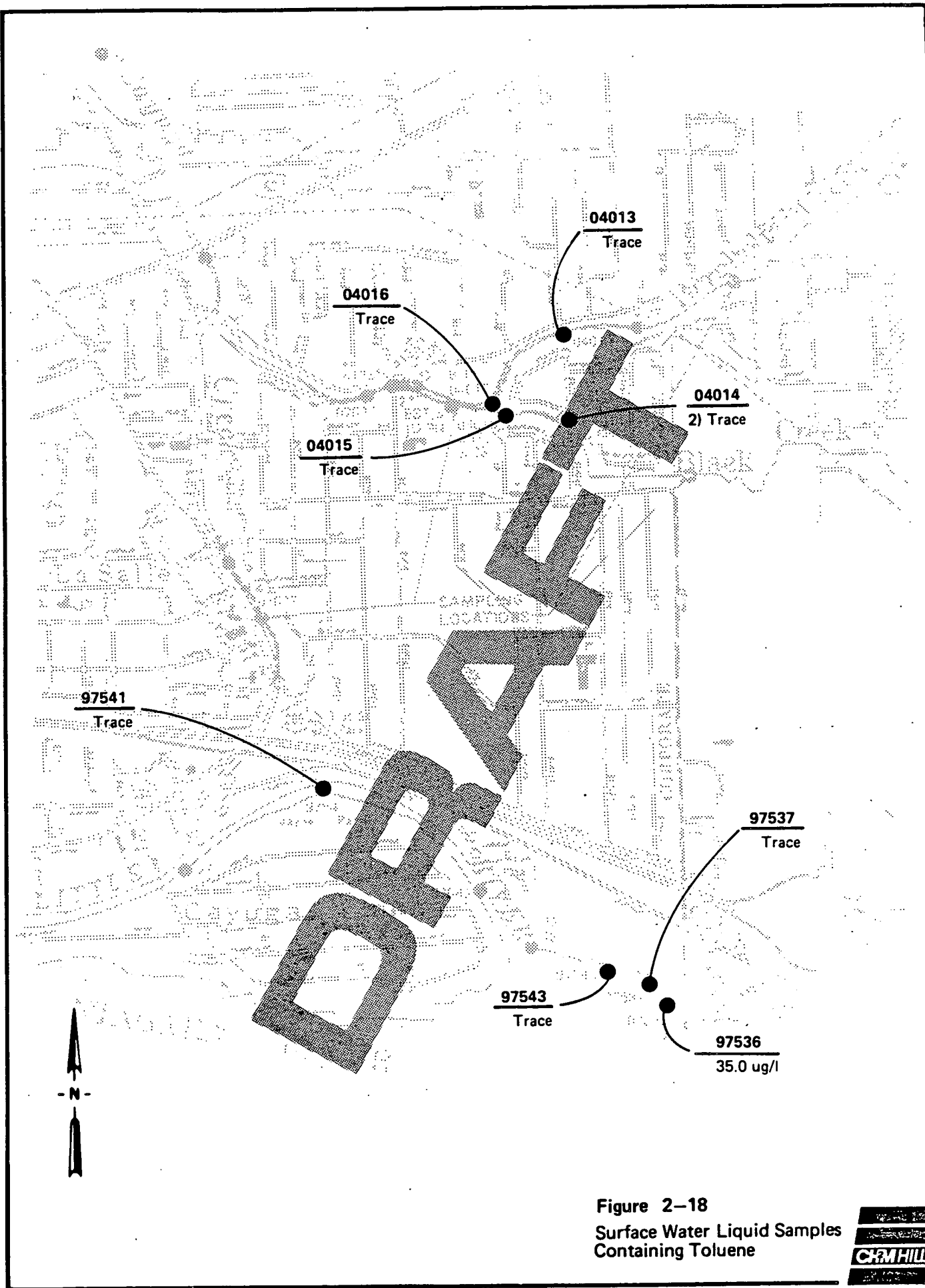
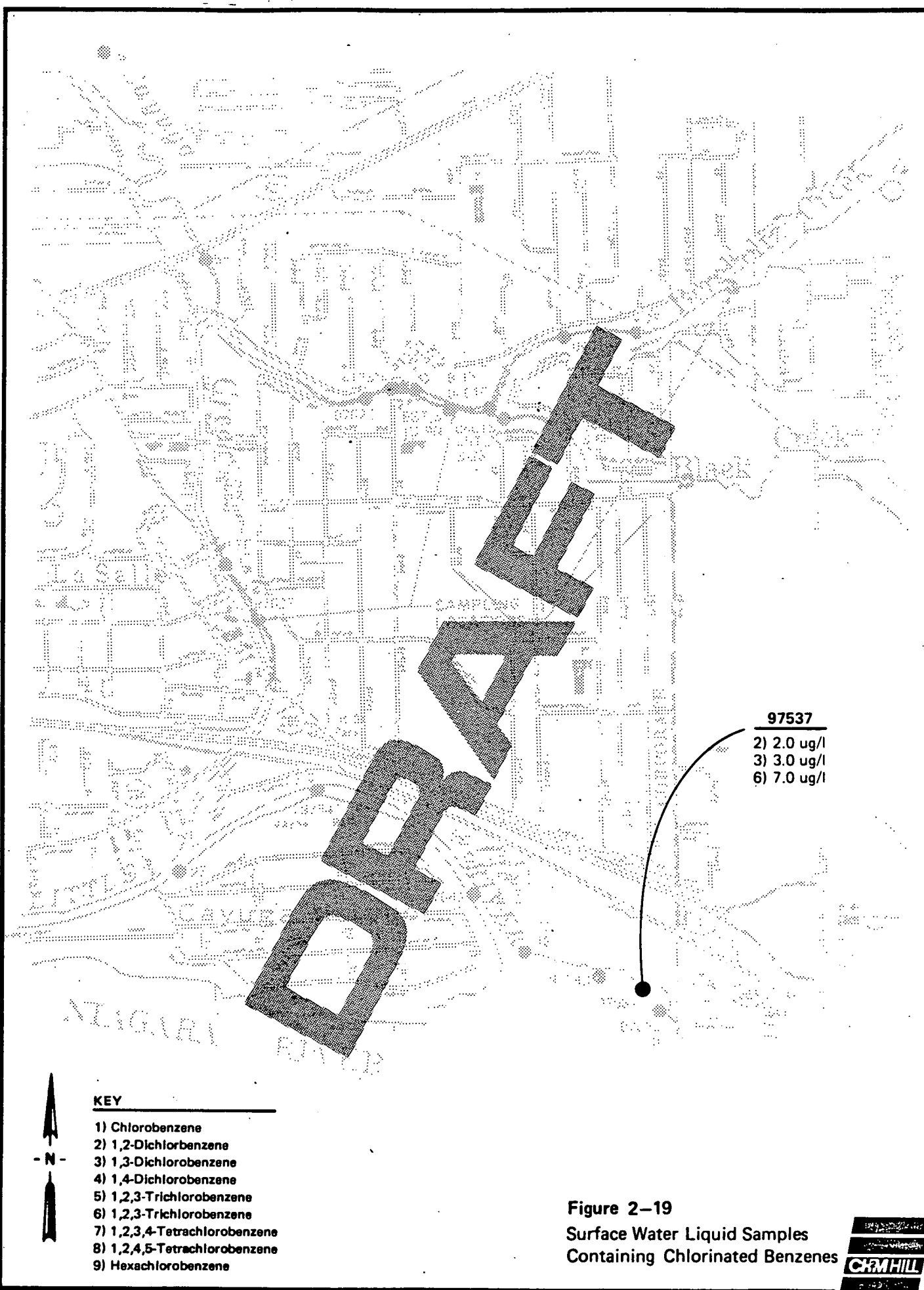


Figure 2-18
Surface Water Liquid Samples
Containing Toluene





97537

2) 2.0 ug/l
3) 3.0 ug/l
6) 7.0 ug/l

KEY

- 1) Chlorobenzene
- 2) 1,2-Dichlorobenzene
- 3) 1,3-Dichlorobenzene
- 4) 1,4-Dichlorobenzene
- 5) 1,2,3-Trichlorobenzene
- 6) 1,2,3-Trichlorobenzene
- 7) 1,2,3,4-Tetrachlorobenzene
- 8) 1,2,4,5-Tetrachlorobenzene
- 9) Hexachlorobenzene

Figure 2-19

Surface Water Liquid Samples
Containing Chlorinated Benzenes



Table 2-5
Summary of Chemical Concentrations in Black Creek Sediment
Malcolm Pirnie Data Taken in 1983

Chemical	Number of Samples	Nondetect Samples		Concentration (µg/kg)		
		Number	Detection Limit (µg/kg)	Maximum (a)	Mean (b)	Median (b)
TCDD	15	13	0.05-0.62	4	0.5	0.1
Hexachlorobenzene	15	15	200-2,000	2,000	690	0.2
α-BHC	15	15	200-2,000	2,000	690	0.2
β-BHC	15	15	200-2,000	2,000	690	0.2
λ-BHC	15	15	2,000	2,000	690	0.2
Chlorobenzene	15	15	2,000	2,000	2,000	2,000
Toluene	15	1	1,000	2,000	2,000	2,000
Arsenic	13	7	1,000	43,000	27,000	27,000
Cadmium	13	1	1,000	2,200	1,300	1,000
Thallium	13			17,000	9,800	9,200

(a) Detected concentration.

(b) Calculated with the detection limit for nondetect samples.

Table 2-6
Summary of Chemical Concentrations in Bergholtz Creek Sediment
Malcolm Pirnie Data Taken in 1983

Chemical	Number of Samples	Nondetect Samples		Concentration (µg/kg)		
		Number	Detection Limit (µg/kg)	Maximum (a)	Mean (b)	Median (b)
TCDD	15	12	0.03-1.00	46	3.9	0.2
Hexachlorobenzene	15	15	200-4,000	4,000	710	200
α-BHC	15	15	200-4,000	4,000	710	200
β-BHC	15	15	200-4,000	4,000	710	200
λ-BHC	15	15	200-4,000	4,000	710	200
Chlorobenzene	15	15	2,000	2,000	2,000	2,000
Toluene	15	15	1,000	2,000	2,000	2,000
Arsenic	14	0	1,000	40,000	25,000	32,000
Cadmium	14	11	1,000	2,200	1,100	1,000
Thallium	14	0	1,000	22,000	9,600	8,800

(a) Detected concentration.

(b) Calculated with the detection limit for nondetect samples.

B-12

COM02/d.504

Table 2-7
Summary of Chemical Concentrations in Cayuga Creek Sediment
Malcolm Pirnie Data Taken in 1983

Chemical	Number of Samples	Nondetect Samples		Concentration ($\mu\text{g/kg}$)		
		Number	Detection Limit ($\mu\text{g/kg}$)	Maximum (a)	Mean (b)	Median (b)
TCDD	13	13	0.07-4.50	ND	0.56	0.3
Hexachlorobenzene	13	13	200-400	400	220	200
α -BHC	13	13	200-400	400	220	200
β -BHC	13	13	200-400	400	220	200
λ -BHC	13	13	200-400	400	220	200
Chlorobenzene	13	13	2,000	2,000	2,000	2,000
Toluene	13	13	2,000	2,000	2,000	2,000
Arsenic	13	0	1,000	170,000	46,000	27,000
Cadmium	13	6	1,000	2,400	1,400	1,100
Thallium	13	0	1,000	16,000	10,000	12,000

(a) Detected concentration.

(b) Calculated with the detection limit for nondetect samples.

KEY

- 1) Dioxin
- 2) α -BHC
- 3) β -BHC
- 4) δ -BHC
- 5) γ -BHC

E-9

- 2) 4,100 ug/kg
- 2) 360 ug/kg
- 3) 49,000 ug/kg
- 3) 360 ug/kg
- 4) 260 ug/kg
- 5) 440 ug/kg

E-8

- 2) 5,800 ug/kg
- 3) 1,700 ug/kg

F-8

- 1) 3.3 ug/kg
- 2) 780 ug/kg
- 3) 300 ug/kg

E-4

- 2) 220 ug/kg

G-6

- 2) 45,000 ug/kg
- 3) 26,000 ug/kg
- 5) 2,400 ug/kg

K-6

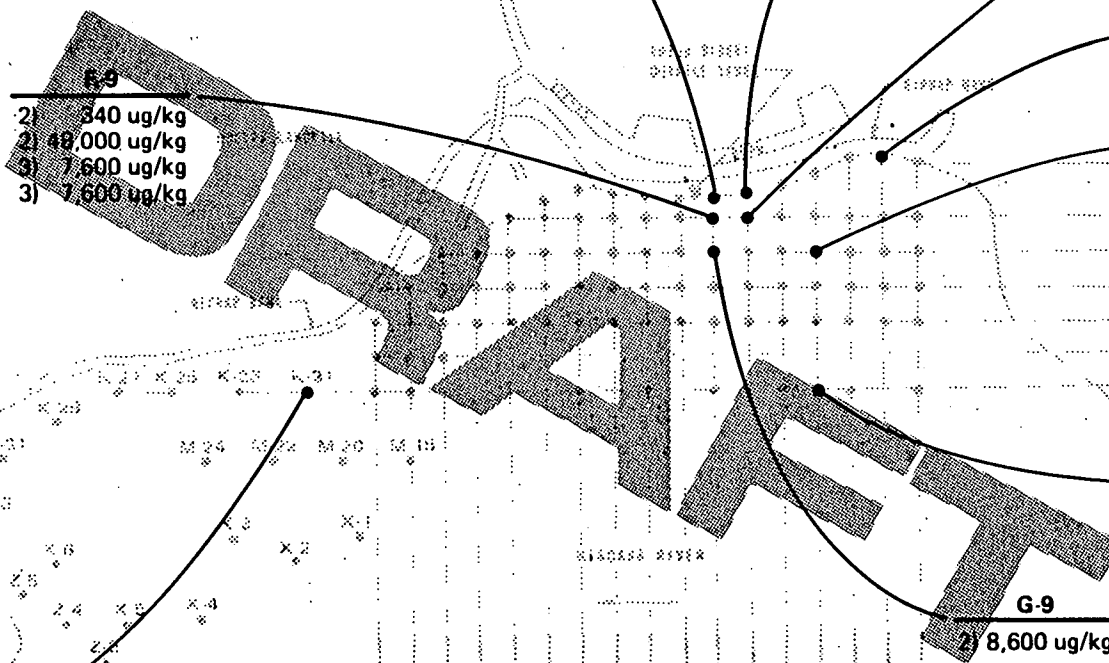
- 2) 350 ug/kg

G-9

- 2) 8,600 ug/kg

K-21

- 2) 2,600 ug/kg
- 3) 580 ug/kg



SCALE: 1" = 300'

Figure 2-20

Surface Water Sediment Samples
Containing Dioxin & BHC Isomers
102nd Street Outfall



KEY

- 1) Chlorobenzene
- 2) 1,2-Dichlorobenzene
- 3) 1,3-Dichlorobenzene
- 4) 1,4-Dichlorobenzene
- 5) 1,2,3-Trichlorobenzene
- 6) 1,2,3-Trichlorobenzene
- 7) 1,2,3,4-Tetrachlorobenzene
- 8) 1,2,4,5-Tetrachlorobenzene
- 9) Hexachlorobenzene

E-9

- 1) 9,100 ug/kg
- 2) 36,000 ug/kg
- 2) 300 ug/kg
- 3) 1,700 ug/kg
- 3) 64,000 ug/kg
- 4) 54,000 ug/kg
- 4) 300 ug/kg
- 6) 6,400 ug/kg
- 6) 300,000 ug/kg
- 6) 12,000 ug/kg
- 9) 960 ug/kg
- 9) 780 ug/kg
- 9) 52,000 ug/kg

E-10

- 1) 14,000 ug/kg

E-12

- 1) 2,200 ug/kg

E-11

- 1) 14,000 ug/kg
- 2) 660 ug/kg
- 2) 1,200 ug/kg
- 3) 360 ug/kg
- 4) 680 ug/kg
- 4) 1,800 ug/kg

E-8

- 1) 5,200 ug/kg
- 6) 1,800 ug/kg

E-7

- 1) 3,300 ug/kg
- 2) 330 ug/kg
- 4) 420 ug/kg

F-7

- 1) 2,800 ug/kg
- 1) 27,000 ug/kg

F-9

- 3) 6,400 ug/kg
- 4) 9,600 ug/kg
- 6) 88,000 ug/kg
- 6) 800 ug/kg
- 9) 6,800 ug/kg

G-6

- 2) 5,200 ug/kg
- 3) 3,000 ug/kg
- 4) 5,800 ug/kg
- 6) 21,000 ug/kg

G-7

- 1) 2,500 ug/kg

H-10

- 6) 560 ug/kg

F-11

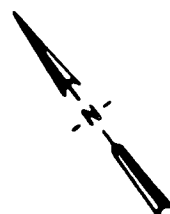
- 1) 13,000 ug/kg
- 2) 360 ug/kg
- 4) 260 ug/kg

G-11

- 1) 1,100 ug/kg

K-21

- 6) 280 ug/kg



SCALE: 1" = 300'

Figure 2-21

Surface Water Sediment Samples
Containing Chlorinated Benzenes
'102nd Street Outfall



Table 2-8
Summary of Chemical Concentrations in the 102nd Street Outfall Sediment (a)
Malcolm Pirnie Data Taken in 1983

Chemical	Number of Samples	Nondetect Samples		Concentration (µg/kg)		
		Number	Detection Limit (µg/kg)	Maximum (b)	Mean (c)	Median (c)
TCDD	31	30	0.07-2.2	3.3	0.4	0.2
Hexachlorobenzene	31	30	200-400	6,800	430	200
α-BHC	31	27	200-400	48,000	3,500	200
β-BHC	31	29	200-400	26,000	1,300	200
λ-BHC	31	30	200-400	2,400	280	200
Chlorobenzene	31	27	2,000	2,700	3,200	2,000
Toluene	31	31	2,000	2,000	2,000	2,000
Arsenic	31	3	1,000	21,000	11,000	12,000
Cadmium	31	30	1,000	1,100	1,000	1,000
Thallium	31	7	1,000	7,200	3,500	3,700

(a) Between rows F, J, 3 and 16 of Malcolm Pirnie.

(b) Detected concentration.

(c) Calculated with the detection limit for nondetect samples.

B-14

No outfall liquid samples were reported by Malcolm Pirnie and only five liquid samples were reported by EPA-ORD. Concentrations were reported as nondetects (detection limits were the same as for the liquid samples discussed in Section 2.4). Table 2-9 lists the concentrations detected in the Niagara River at the raw water intake at the Niagara Falls city water treatment plant.

2.6 BIOTA SAMPLES

Biota samples of spottail shiners, creek chubs, carp, or crayfish were taken from the creeks and rivers, and the results are shown in Figure 2-22. TCDD concentrations were as high as 417 ng/kg wet weight (sample taken in 1980), and there were several samples above 25 ng/kg wet. More recent samples from 1984 from the New York Department of Health had reported concentrations as high as 127 ng/kg wet weight.

TCDD concentrations in spottail shiners have also been measured at other areas of the Niagara River. In 1981, 15 ng/kg was measured near Fort Erie, but a nondetect (detection limit 1 ng/kg) was recorded in 1982. A concentration of 1 ng/kg was reported in 1982 just upstream of Grand Island, and nondetects were reported in nearby areas.

A concentration of 120 ng/kg was reported in 1982 about half way up the Grand Island in the Tonawanda Channel, which is upstream from the 102nd Street outfall. A nondetect was reported a short distance away (NRTC, 1984).

Spottail shiners are small fish (under three inches), and tend to school and stay in restricted areas. Their movements will depend on food availability, predators, water temperature and dissolved oxygen. Concentrations of TCDD in sport fish (trout, perch, salmon, bass) in the Niagara River were reported most often as nondetects (detection limit 10 ng/kg) in 1980, but bass samples in 1981 were reported as nondetects (detection limit 1 ng/kg) to 30 ng/kg in the Lower Niagara River near Queenston. Sport fish captured in Lake Ontario had concentrations that ranged from nondetect (detection limit 10 ng/kg) to 19 ng/kg in 1980 (NRTC, 1984).

Caged freshwater clams were exposed to the Niagara River for 16 days in 1980 at the downstream end of the Tonawanda Channel. The measured concentration of α -BHC in a composite of three was 12 ± 3 μ g/kg wet weight (Environment Canada, 1981).

Table 2-9
RAW WATER CONCENTRATIONS AT THE NIAGARA FALLS
WATER TREATMENT PLANT ($\mu\text{g/L}$)

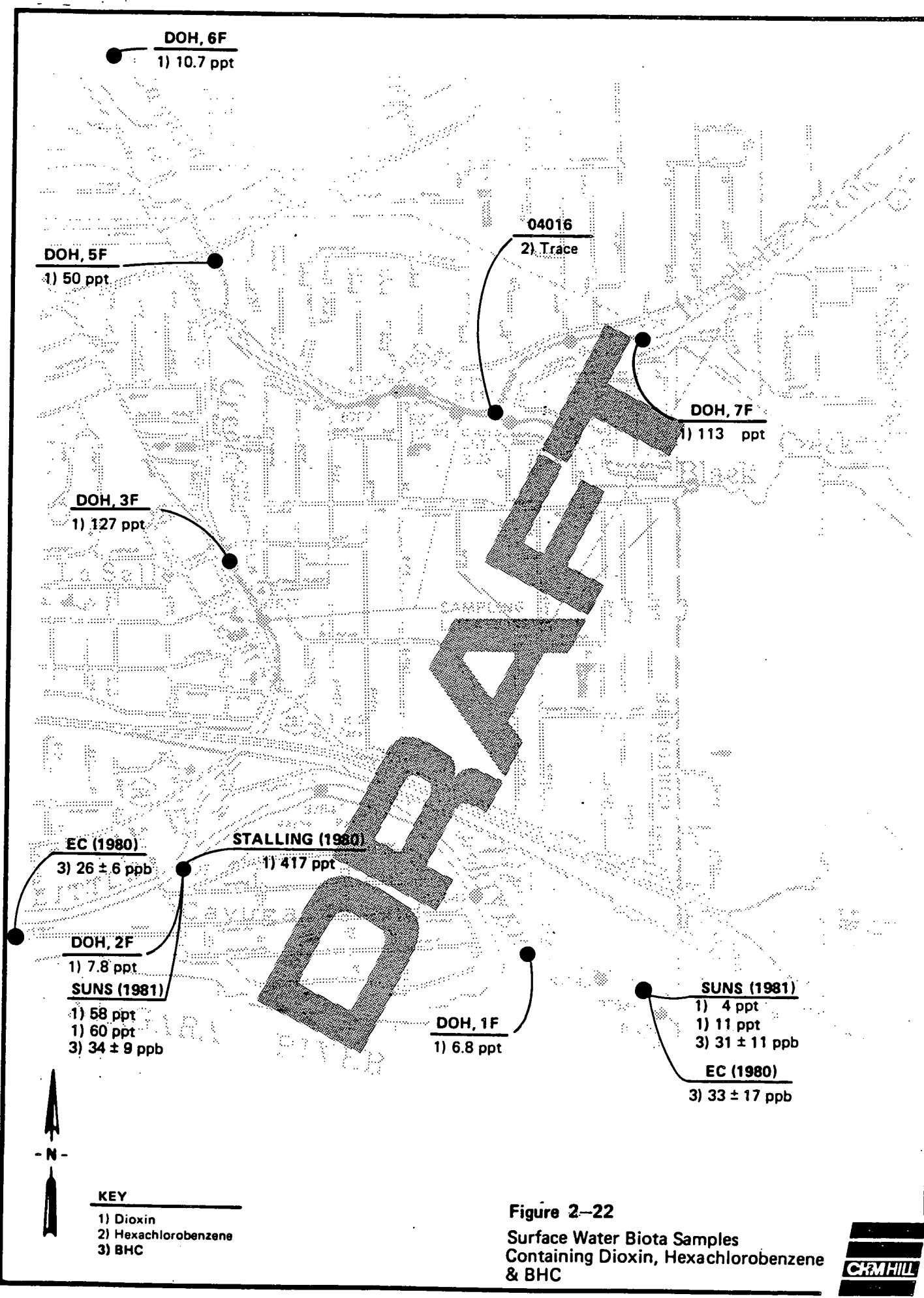
Chemical	1979 ^a	1980 ^b
TCDD		ND
α -BHC	ND-0.005	ND-0.005
β -BHC	ND	ND-0.001
γ -BHC	ND-0.003	ND-0.005
Toluene	ND-0.063	ND-0.3
Chlorobenzene	ND	ND
Arsenic	<1	

ND-not detected.

^aEnvironment Canada 1980. Six or seven samples.

^bEnvironment Canada 1981. Eleven or twelve samples.

WDR101/002



Section 3 CONTAMINANT MIGRATION

The objective of this section is to evaluate the potential for migration of contaminants from the sewers and creeks. Factors influencing potential migration include the physical characteristics of the sewers, the physical characteristics and hydrologic setting of the three creeks, the physical properties of the contaminants, the physical, chemical, and biological transformation processes by which a contaminant is transported and/or transformed in the environment.

3.1 FACTORS AFFECTING SEDIMENT MIGRATION

The degree to which sediments can be transported in any medium is dependant on several factors. Stream flow is a major factor with high flows scouring stream sediments and carrying (entraining) them downstream. The physical and chemical characteristics of the sediment and entraining water directly influence the type and volume of material eroded and transported and the condition of deposition when and where it occurs. Both entrainment and transport depend on the shape, size, and weight of the particle and the forces exerted on the particle by the flow of water. When these forces are diminished to the extent that the transport rate is reduced or transport is no longer possible, deposition occurs.

Sediment is transported in suspension (wash load) or as bed load, which rolls or slides along the bed of the creek. Sediment moves interchangeably by suspension and bed load. The rate of bed load transport depends on the availability of sediment and the influence of change in water flow conditions. If the supply of sediment is reduced by erosion control the amount of bed material or sediments will be reduced, especially during high flow conditions. Suspended sediment load includes both the bed material load in suspension and the wash load. Where the chief source of sediment is fine-textured soils, the wash load rather than the total bed material load usually constitutes the majority of the sediment discharge. Lower water velocities are required to keep the finer sediments in suspension and moving downstream. The coarser material requires higher water velocities before it is entrained or moves as bed load. Coarse material will not be moved as rapidly as the finer material.

3.2 PHYSICAL FEATURES IN THE SANITARY SEWERS

Most sanitary sewers in the assessment region were installed by the early 1930's, except for those connected to the Griffon Manor housing in 1973. All sanitary sewers in the eastern section of the EDA flow toward Colvin Boulevard, then west beneath the Boulevard to Lift Station No. 4. They

are constructed of vitrified clay pipe ranging from 8 to 18 inches in diameter with mortar joints. Their depths ranged from 6.5 feet below street level in the Griffin Manor housing project to 21 feet below Colvin Boulevard. Their condition has been described as good, with few major cracks or leaks. Sewers were plugged near Manholes 265, 267 and 466 in March, 1983 (see Figure 2-1).

When constructed, manholes had an exterior mortar coat, which was not in evidence at the time of the Malcolm Pirnie inspection.¹ The sanitary sewers were built without a drainage for channeling groundwater away from the pipe. This tends to produce a waterway around and along the pipe which provides for infiltration of groundwater into the pipe and exfiltration of water and material out of the pipe into the groundwater through defective joints and cracks (CDM, 1975). Also bedding material was found to be backfill of the original clay trenching material, not crushed gravel as was required. Many of the sanitary sewers in the study area have loose bricks in manholes which is a potential source of sediment accumulation.

The flow from Lift Station No. 4 is combined with the flows from the western part of the region (Figure 3-1). They cross over Cayuga Creek at Military Road and then move on to Lift Station No. 6. Most of the wastewater then moves in a westerly direction along Frontier Avenue. At 74th Street, the wastewater turns north and flows to Girard Avenue. The flow is then westerly to 66th Street.

Occasionally, wastewater from Lift Station No. 6 can overflow into Lift Station No. 1. From there, it flows westerly along Stephenson Avenue. At 66th Street, the wastewater flow turns north and joins the flow coming directly from Lift Station No. 6 at the intersection of Girard Avenue and 66th Street. Industrial wastewater from the northern industrial sector of Niagara Falls is combined with the domestic sewage at this point. The combined wastewater flows north to John Street and then west to 47th Street and Royal Avenue. The Southside Interceptor begins here and the flow during dry weather is to the City of Niagara Falls Wastewater Treatment Plant. During high flow periods, occurring most often during rainfall, the Southside Interceptor may overflow through regulators into the Falls Street Tunnel, which flows to the Gorge Pumping Station and then to the City of Niagara Falls Wastewater Treatment Plant or bypassed directly to the Niagara River.

¹ Discussion on sanitary and storm sewers is derived from Malcolm Pirnie (1983).

Table 3-1
FLOOD DISCHARGE ON CAYUGA AND BERGHOLTZ CREEKS

	Drainage Area (sq. miles)	Peak Discharge (cfs)		
		10-yr	50-yr	100-yr
Bergholtz at confluence with Cayuga	13.3	900	1,350	1,550
Cayuga at confluence with Little Niagara	28.2	1,650	2,650	3,050

Source: FEMA 1982

Table 3-2
FLOOD ELEVATIONS ON BERGHOLTZ CREEK AT SELECTED LOCATIONS

	Elevation (ft)			
	Confluence with Cayuga Creek (0 ft) ^a	91st St. (1325 ft) ^a	93rd St. (2500 ft) ^a	101st St. (4200 ft) ^a
Stream bed	559.8	560.25	561.0	559.5
10-year	567.75	569.0	569.25	569.5
50-year	569.5	570.75	571.0	571.5
100-year	570.75	571.5	571.75	572.0

^aDistance from confluence with Cayuga Creek.

Source: FEMA 1982

WDR101/003

Lift station Nos. 4 and 6 have overflow bypasses that discharge to the storm sewers in the west during high flow periods. The Lift Station No. 4 surcharge pump discharges into a storm sewer that empties into Cayuga Creek at the Lindberg Avenue outfall. The surcharge pump at the Lift Station No. 6 discharges into a sewer that empties into Cayuga Creek at Pershing Avenue. An additional sanitary overflow into Cayuga Creek during high rainfall periods can occur from the overflow pump station at the intersection of Cayuga Drive and Military Road.

During the sampling program by Malcolm Pirnie in January 1983, no surcharged sanitary manholes were observed in the northern section. Manholes 256 on 101st Street and 283 on 95th Street, however, appeared to have previously surcharged to a depth of about 5 feet. Manholes on 95th Street upstream of 283 also showed evidence of high water marks and/or excessive sediment on benches, suggesting past surcharging.

No signs of past surcharging in the southern area near Frontier Avenue were observed during the Malcolm Pirnie investigation. It has been suggested by Niagara Falls City personnel, however, that all sanitary sewers in the area are subject to surcharging. Surcharging is typically caused by insufficient hydraulic capacity, pipe constriction either from root intrusion, sediment deposition, or collapsed pipe, structural problems, excessive groundwater infiltration, or inflow from storm sewer interconnections, illegal house connections, etc.

Surcharging has been observed in the area of 91st Street, 92nd Street, 93rd Street, and Read Avenue during high rainfall periods. Portable pumping equipment has been used to relieve the hydraulic overload on the sanitary sewers during high rainfall at Pasadena and Read Avenues, Colvin Boulevard and 93rd Street, and 93rd and Cayuga Creek by discharges to the adjacent storm sewers.

During the Malcolm Pirnie investigation, most of the sewers in the western section along Read Avenue east of 92nd Street were surcharged. The cause was likely a clogged pipe on Read Avenue between 91st and 92nd Streets because surcharging was not observed downstream of this area.

3.3 PHYSICAL FEATURES IN THE STORM SEWERS

Many storm sewers were initially used for sanitary sewer disposal prior to the installation of sanitary sewers which began shortly after annexation. After the canal was filled in 1953 and Read and Wheatfield Avenues were built across the landfill, the City of Niagara Falls installed a storm sewer line under Read Avenue, which entered the canal from

97th Street and ended in a catch basin at approximately 98th Street. Storm sewer lines were also built along Wheatfield Avenue from 97th Street and 99th Street running toward the canal. Apparently, all storm sewer trenches were backfilled with excavated soils as were most of the sanitary sewers (CDM, 1975; Malcolm Pirnie, 1983). Many of the manholes were noted as in poor condition during the Malcolm Pirnie field investigation. Loose brick with sediment accumulation on the walls and benches was common in approximately 40 percent of the manholes inspected.

Storm sewers in the northern section of the EDA flow northward to three outfalls, the 96th, 98th, and 101st, into Black Creek (see Figure 2-2). The pipes are constructed of several materials, including vitrified clay (8- to 18-inch diameter), concrete (18- to 36-inch diameter), and corrugated steel (48- and 72-inch diameter). Depths ranged from 3.5 to 10 feet below street level in the northwest with the outlet to the 96th Street outfall, and from 4.5 to 6.5 feet in the northeast to the 101st Street outfall.

Storm sewers in the southern section flow southward to two outfalls, the 102nd Street outfall (42-inch diameter) into the Niagara River, and the Little Niagara outfall southwest of 95th Street. It is estimated that the 102nd Street outfall and its sewer network were built in the early 1920's. The originally discharged stormwater collected from 97th, 99th, 100th, 101st, and 102nd Streets and part of Frontier Avenue. The 97th and 99th Street storm sewers were cut and plugged in March, 1983 to prevent further discharge from Love Canal. Between the Love Canal EDA and the outfall, the sewer passes under the LaSalle Expressway and Buffalo Avenue, and then through the 102nd Street Landfill. The sewers vary in size from 6 to 42 inches in width. Depth below the surface varied from approximately 3 feet below street level in the Griffon Manor housing development to 10 feet below street level along Buffalo Avenue.

In the western area of the Love Canal region, storm sewer outfalls are at 93rd Street into Bergholtz Creek, and at Lindbergh Avenue (30-inch) and Read Avenue (18-inch) to Cayuga Creek.

Manhole 232A was the only location that showed any signs of past surcharging in the northern section during the Malcolm Pirnie investigation. The storm sewer along 93rd Street is reported to overflow frequently.

3.4 SURFACE WATERS

Black Creek flows westerly from the eastern edge of the EDA to a point near Colvin Boulevard and 102nd Street where it enters an underground pipe. Prior to its piped section, the

creek is approximately 2- to 4-foot wide with less than a foot of water at very low flows. The bottom material is clay extending to 21 inches below the creek bed. Access is readily available to this section of the creek as it is open and flat and only a short distance from the street. The flow in the pipe is to the west, then north, then west near the intersection of Greenwald Avenue and 98th Street, where it receives stormwater. The Creek emerges from the pipe just north of Greenwald Avenue and flows westerly to its confluence with Bergholtz Creek in the vicinity of 96th Street. After surfacing from the piped section, the creek is fenced on both sides to the confluence with Bergholtz Creek. The bottom material is clay down to 3 feet overlain with organic material. Disturbance of the sediments caused an oily film to surface during the Malcolm Pirnie investigations (Malcolm Pirnie, 1983).

Bergholtz Creek flows southwesterly from the northwest corner of the EDA to its confluence with Black Creek. After joining Black Creek it flows west to the confluences with Cayuga Creek near 88th Street. The bottom composition is clay down to approximately 4 feet overlain by organic material. Some portions of the bed are composed of coarse material of gravel and cobbles. A bed roughness coefficient used in hydraulic computation for Bergholtz Creek was estimated at 0.03 for the channel and 0.06 for the overbank (FEMA, 1982). These values indicate the ease at which water moves along the surface of the creek or bank. The higher the roughness coefficient, the greater surface disturbance. Disturbance of the sediments resulted in an oily film surfacing during the Malcolm Pirnie investigations. Creek banks vary in width, from 5 to 60 feet. Bank height and slope also vary with location. Flows are moderate and the depth averages 2 to 3 feet deep. Flooding on Bergholtz Creek occurs primarily in the early spring as a result of snowmelt (average of 57 inches), heavy rains (average 25 inches) and ice jams in the Niagara River. Shallow flooding can happen during heavy rains from ponding. Table 3-1 shows discharge rates for Bergholtz and Cayuga Creeks. Table 3-2 gives stream bed elevation and flood elevations for the 10-, 50-, and 100-year events at two locations (FEMA 1982).

Cayuga Creek flows southerly to its confluence with the Little Niagara River near South 87th Street. Width of the creek varies from 35 to 100 feet and the banks are moderately steep. The creek bottom is clay to approximately 4 feet overlain with organic material. A bed roughness coefficient used in hydraulic computations was estimated to be 0.03 for the channel and 0.06 for the overbank. Water depth at the time of the Malcolm Pirnie investigation was to 5 feet and averages 4-5 feet with moderate flows (FEMA, 1982).

The most severe flooding on Cayuga Creek occurs during early spring as a result of snowmelt, heavy rains and ice jams in the Niagara River. Conditions of strong southwesterly winds, power releases and ice jams on the Niagara can back water up into Cayuga Creek for 2,500 feet from its confluence with the Little Niagara. Table 3-3 gives stream bed elevations and flood elevations for the 10-, 50-, 100-year events at several locations upstream of its confluence with the Little Niagara 1982).

The Little Niagara River flows into the Niagara River at the west end of Cayuga Island. Because of the flat stream gradient, the Niagara River can significantly affect the flow patterns in the creeks. Weather conditions, such as strong wind from the southwest, can produce local and temporary reversal of flows in the three creeks. Wide fluctuations of depths in the creeks can be experienced depending on the quantities of water being withdrawn from the Niagara River by power companies. Flow rates of the Niagara River have a mean of 200,000 cfs and a lowest monthly mean flow of 140,000 cfs. Water flows at about 1.5 to 2.3 ft/sec, depending on the volume flow rate of the river. The Tonawanda Channel of the Niagara River carries about 40 percent of the river's flow.

3.5 GROUNDWATER CHARACTERISTICS

The regional groundwater hydrology is summarized by four characteristics.

- (1) There is a shallow, seasonably saturated system that consists of silt fill and silty sand (see Figure B-1).
- (2) There is a bed of confining clay and till below the shallow system.
- (3) Below the confining layer is the Lockport Dolomite.
- (4) The local system is bounded at the lower end by the Rochester Shale (GEOTRANS, 1981).

The Dolomite is probably bounded hydrogeologically in the Love Canal area in the west by the lower Niagara River and in the south by the upper Niagara River.

The shallow groundwater system is bound to the north by Bergholtz Creek, on the west by Cayuga Creek and toward the south by the Little Niagara River. The system has some low permeabilities and a low gradient. Before the Love Canal remedial actions, the slight water mound in the fill resulted in a potential migration in all directions. The effect of

Table 3-3
FLOOD ELEVATIONS ON CAYUGA CREEK AT SELECTED LOCATIONS

	Elevation (ft)					Confluence w/Bergholtz (5750 ft) ^a
	Mouth (0 ft) ^a	88th St. (800 ft) ^a	South Military Rd. (1593 ft) ^a	Lindbergh Ave. (3375 ft) ^a	Pear Ave. (4475 ft) ^a	
Stream bed	555.5	559.4	557.5	559.5	560.0	560.0
10-year	567.5	567.5	567.5	567.5	567.5	567.7
50-year	567.75	567.75	567.75	568.0	569.5	569.5
100-year	568.0	568.0	568.0	569.5	570.2	570.5

^aDistance from confluence with Niagara.

Source: FEMA 1982

WDR101/004

DRAFT

fill around the sewers and swales is uncertain. Exfiltration, if any, from the sewers is likely to connect with this shallow system.

3.6 CONTAMINANT MIGRATION POTENTIAL

3.6.1 Definitions

Several definitions will be useful in the discussions below:

- o Solubility. The solubility of a chemical is a measure of its tendency to dissolve in water. Many physical properties influence the solubility of a chemical in the environment: valence, state, temperature, soil adsorption, and presence of other chemicals.
- o Vapor pressure. Vapor pressure represents the tendency of a chemical to volatilize from the pure substance. Although this ideal condition is not likely to be present in the environment, the parameter provides an indication of the importance of volatilization.
- o Henry's law constant. This is the ratio of concentrations in air to that in water for a chemical at equilibrium. It thus represents the tendency of a chemical to escape from aqueous solutions.
- o Octanol-water partition coefficient. This parameter expresses the tendency of a chemical to concentrate in either the organic or aqueous phase. The coefficient correlates with the bioconcentration factor.
- o Soil-water partition coefficient. This parameter is the ratio of the soil: chemical concentration to the soil: water concentration divided by the soil organic carbon content at equilibrium. The coefficient provides an indication of the leachability of a chemical in the soil.
- o Bioconcentration factor. This parameter is the ratio of the chemical concentration in fish to the concentration in water at equilibrium. The coefficient represents the tendency of a chemical to concentrate in the tissue of aquatic species.
- o Intestinal absorption factor. This parameter is the fraction of ingested chemical that is absorbed in the human gut. This coefficient is used in estimating the human chemical dose.

3.6.2 Mobility of Contaminants

Appendix C contains a discussion of the chemical characteristics of the contaminants and the partitioning of the contaminant into various environmental media (see Table 3-4 for a summary of the physical properties of the contaminants which may be important in the transport and transformation of the contaminant through the environment). The mobilities of the contaminants vary. The inorganics arsenic, cadmium and thallium, exhibit low aqueous solubility at neutral pH, such as would be found in sewers and creeks. Therefore, they should be strongly bound to sediments and not available for uptake by aquatic organisms. The migration potential of the metals would then be linked to the migration of sediments through the sewers and creeks. TCDD is most likely to be found adsorbed onto sediments and transported with them also. TCDD is not a volatile compound nor is it readily soluble in water. It will persist in the sediments for a long period of time. λ -BHC is water soluble but has a limited residence time in aquatic systems. The predominant portion of λ -BHC released to the environment would be found in sediments. If the hypothesis that λ -BHC is isomerized to alpha and delta isomers in sediments by microbial activity is correct, a greater portion of BHC compounds detected in the sediments should be of the alpha and delta isomers, both of which are more persistent in the environment than the gamma isomer (U.S. EPA, 1980).

The chlorinated benzenes all have relatively high tendencies to adsorb onto sediments, with adsorption increasing as chlorination increases. Transformation processes slow with increasing chlorination. Persistence in the environment would be expected, especially of the higher chlorinated compounds. Toluene is highly volatile and relatively insoluble. That portion solubilized tends to persist in the aquatic environment. Toluene will adsorb to sediment high in organic matter, although its octanol:water coefficient is relatively low.

Table 3-4
PHYSICAL CHARACTERISTICS OF CONTAMINANTS^a

Contaminant	Molecular Weight	Melting Point (°C)	Boiling Point (°C)	Freezing Point (°C)	Flash Point (°C)	Solubility in mg/l	Vapor Pressure mm Hg at 25°C	Henry's Law Constant ^b	Octanol: Water Partition Coefficient	Soil-Water Partition Coefficient	Bioconcentration Factor	Intestinal Absorption Factor
2,3,7,8-TCDD	321.9	302-305	--	--	--	0.2	0.00001	2.1	6.9-19 x 10 ⁶	3.8 x 10 ⁶	3,000-68,000 ^d 7,000-900,000 ^d	fish: 0.5 - 0.86 ^e soil: 0.2 - 0.26 ^e
γ-BHC	290	65 or 112.5	--	--	--	7.3	0.0008	0.05	5200	4270	130	0.35
Chlorinated benzenes												
Chlorobenzene	112.6	-46.5	132	--	29.4	499	11.8	2.6	690	380	10.3	
1,2-Dichlorobenzene	147.01	-17.0	180.5	--	68.1	145	1.28	1.3	2400	--	--	
1,3-Dichlorobenzene	147.01	-24.7	173.0	--	68.1	143	1.89	--	2400	--	--	
1,4-Dichlorobenzene	147.01	53.1	174.0	--	65.6	75	1.0	2.4	2400	--	--	
1,2,4-Trichlorobenzene	181.46	16.9	213.5	--	110.0	34.6	0.29	4.3	13000	--	--	
Hexachlorobenzene	284.76	230	322.9	--	242.2	0.005	0.0000168	0.12	--	3900	8690	
Toluene	92.13	-95	110.6	-94.9	6-10	524.9	28.7	--	310	310	10.3 ^c	
Arsenic	74.92	817	NA	NA	NA	NA	NA	NA	NA	NA	44	
Cadmium	112.4	320.9	765	NA	NA	NA	NA	NA	NA	NA	3-12,000	
Phallium	204.37	303.5	1,457±10	NA	NA	NA	NA	NA	NA	NA	NA	

^aData from ambient water quality criteria documents (USEPA, 1980) unless otherwise specified.

^bX 0.001 log part. coeff.

^cThree percent lipids in fish.

^dTwo ranges are based on measured and calculated values, respectively. U.S. EPA has used 5,000 (U.S. EPA, 1984a).

^eRange USEPA, 1984c.

NA = Not Applicable.

Section 4 QUALITATIVE EXPOSURE PUBLIC HEALTH ASSESSMENT

This section provides a brief summary of the toxicology effects of the target chemicals and then examines the environmental pathways by which populations at risk might become exposed to the chemicals. A set of potential exposure scenarios is developed for each sources of contamination; sewers, creeks, 102nd Street outfall and the Niagara River. Each scenario is discussed qualitatively in terms of the events that must occur for human exposure, the potential duration of exposure, and the potential chemical dose levels that might result from this exposure. Significant uncertainties in the evaluation are identified.

4.1 TOXICOLOGY SUMMARY

A summary of the toxicological properties of the contaminants of concern is presented below. For a more detailed discussion of the toxicology of the contaminants refer to Appendix C.

2,3,7,8-Tetrachloro-dibenzo-p-dioxin. TCDD has been shown to be extremely toxic in experimental animals and has teratogenic, mutagenic and carcinogenic effects. Sensitivity and target organs differ for various species. Chloracne and hyperkeratosis are distinctive symptoms of TCDD exposure in animals and humans. A number of reports suggest an association of soft tissue sarcomas and TCDD exposure in humans.

Hexachlorocyclohexane isomers. (HCH or BHC). λ -BHC is stored in fat tissue of experimental animals, but is cleared from the system after cessation of exposure. Target organs include the brain, liver, and kidney. Chronic exposures may cause blood disorders. The α , β , and γ isomers of BHC have been demonstrated to induce cancer in experimental animals.

Chlorinated benzenes. The main sites affected by acute exposure to high concentrations are the hepatic, renal and nervous systems. Lower levels of exposure for some of the chlorinated benzenes may have adverse effect on the nervous system. Hexachlorobenzene has been demonstrated to produce fetotoxicity in experimental animals and has been shown to be a animal carcinogen.

Toluene. The primary effect of exposure to humans is dysfunction of the central nervous system. Levels required to induce effects (100-300 ppm in air for short-term exposures) are substantially in excess of typical environmental levels. No evidence of mutagenicity, teratogenicity or carcinogenicity has been found in animal experimentation.

Table B-1
NUMBER OF OCCUPIED LOTS OR BUILDINGS WITHIN THE EMERGENCY DECLARATION AREA (EDA) BOUNDARY
AS OF MAY 1984

	<u>Between Bergholtz Creek (Northern EDA Boundary) and Colvin Boulevard</u>	<u>Between Colvin Boulevard and Frontier Avenue</u>	<u>Between Frontier Avenue and Southern EDA Boundary</u>
Between western EDA boundary and 93rd Street	7 lots	14 lots	--
Between 93rd and 96th Streets	4 lots	Senior citizen housing (4 units) LaSalle Development Courts 1-12 (18 full or partially- full units) Saraceni Drugs LaSalle Community Center	--
Between 96th and 100th Streets	28 lots Wesley Methodist Church	4 lots Church of God	3 lots The Establishment Restaurant Jerry's Garage Fama's Welding
Between 100th Street and eastern EDA boundary	7 lots	54 lots Fire house	--

WDR65/80

Attachment B
DETAILED LISTING OF POTENTIAL RECEPTORS

Potential receptors are those persons living or working in and adjacent to the EDA, populations that may come into primary or secondary contact with contaminated surface waters of sediments, downstream populations, recreationalists on the Niagara River in the vicinity of the 102nd Street outfall, users of the public docks and private marina immediately downstream of the 102nd Street outfall, recreationalists on the Little Niagara River at the confluence of Cayuga Creek and downstream users.

As of May 1984, 121 residences inside the EDA including two homes inside Ring 2, were still occupied. Additional buildings that were occupied or being used at that time included two churches, one restaurant, one garage, one welding shop, one drugstore, one fire house, one community center, four units of a senior citizen housing development and 18 full units of a housing development court. The number of occupied lots and buildings are summarized by location in Table B-1. According to the Love Canal Revitalization Agency, approximately 325 persons work and live within the EDA.

Potential receptors are those persons living or working adjacent to the three creeks. The status of properties which are adjacent to the creeks was determined by the CH2M HILL May 1984 property map and a 1980 aerial photograph supplied by NYDEC.

Black Creek flows west from 98th Street to its confluence with Bergholtz Creek, a distance of approximately 550 feet. Nine lots border the north creek bank. Four of these have occupied houses; the other five houses are unoccupied. Nine lots also border the south creek bank. Two of these contain occupied houses; four have unoccupied houses, and three are vacant lots. Approximately 2,400 feet of Bergholtz Creek is within the EDA. Creek length from the eastern most boundary of the EDA (near Mueller Court) to the confluence with Black Creek is about 1,500 feet. Twenty lots border the south creek bank within this reach; four have occupied houses; 13 have unoccupied houses, and three are vacant lots. Bergholtz Creek flows an additional 900 feet from its confluence with Black Creek before reaching the western most EDA boundary. The 93rd Street school property (unoccupied) and two vacant lots border the south creek bank within this reach. The school property is located between the two vacant lots and borders about 800 feet of Bergholtz Creek. A total population of 20 persons currently (May 1984) occupy homes along the creeks within the EDA.

DRAFT

Attachment A
DETECTION LIMITS FOR THE 1980 EPA MONITORING STUDY

Detection limits for soil and sediment samples were obtained for the 1980 monitoring study and are shown in Table A-1. They represent the average reported detection limit across the analytical laboratories used in that study.

Table A-1
SOIL AND SEDIMENT DETECTION LIMITS (a)

Chemical	Average Reported Detection Limit (µg/kg)
TCDD	0.001-0.020 (a)
Hexachlorobenzene	85
BHC isomers	6
Chlorobenzene	7
Dichlorobenzenes	85
1,2,4-trichlorobenzene	85
Toluene	7
Cadmium	10
Thallium	100

(a) Depends on organic content of sample. The value
0.02 µg/kg was used in Section 2.

Source: Black, 1984.

WDR101/28

4.8 FISH AQUATIC TOXICITY IN THE NIAGARA RIVER

Chemical concentrations of the Niagara River water shown in Table 2-9 are below the aquatic life criteria shown in Table 4-1, and therefore little effect on the river biota would be anticipated.

WDR101/001

DRAFT

exposure because of the general absence of volatile organic chemicals at the 102nd Street outfall.

Niagara River water (after treatment) is used for drinking water purposes. Table 2-9 shows the chemical concentrations in the raw water (before treatment) at the Niagara Falls water treatment plant. These concentrations are below the drinking water criteria shown in Table 4-1. The quantitative contribution of the sewers, creeks and 102nd Street outfall on the chemical loading of the river has not been determined. Data on the concentrations in the Tonawanda Channel upstream and downstream from Love Canal suggest little effect. Concentrations of a-BHC in 1979 were 0.007 ug/L at river range 23.3 (upstream from Cayuga Island) and 0.005 ug/L at river range 19.3 (downstream at the end of Grand Island). Concentrations of g-BHC and cadmium were unchanged (Environment Canada, 1980).

4.7 INGESTION OF FISH

Based on its evaluation of TCDD toxicity, the U.S. Food and Drug Administration (FDA) has stated that "fish containing more than 50 ppt [ng/kg] should not be consumed and those containing more than 25, but less than 50 should not be consumed more than twice a month (Miller, 1983)." FDA did not provide any recommendation of concentrations below 25 ppt. The FDA cancer potency for TCDD is 17,800 kg-day/mg, which is less than EPA's Carcinogen Assessment Group (CAG) by a factor of nine. In contrast to the EPA, FDA used a rat-to-man extrapolation based on body weight, did not include some data that CAG did, and did not compensate for early morbidity in the animal studies. FDA, in its exposure assessment, assumed that only a limited quantity of fish having TCDD concentrations near the advisory level would be consumed (USEPA, 1984a).

The consumption rate of local fish caught in the creeks or near the 102nd Street outfall is not known, although anecdotal stories suggest summertime fishing. Section 2.6 discussed the concentrations of TCDD found in the local fish. Concentrations as high as 417 ng/kg wet weight were reported from in a 1980 sample. Samples of spottail shiners and creek chubs collected by the New York Department of Environmental Conservation in 1984 in the creeks below Porter Road showed TCDD concentrations from 6.8 to 127 ng/kg wet weight (2 of 7 samples exceeded 50 ng/kg and 3 of 7 samples exceeded 25 ng/kg). As the chemical concentrations decrease in the creek and 102nd Street outfall sediments, the concentrations in the fish should decrease. These fish, however, are not usually consumed because of three small sizes.

downstream on Bergholtz Creek and up to 197th Street on Black Creek. Although fences can be breached or gone around, this barrier should further reduce the potential human exposure to the contaminated areas of Black and Bergholtz Creeks. Access to Cayuga Creek, however, is open. Several homes abut the creek and recreational use has been observed.

During recreational activities, creek sediment and water may be ingested. Section 4.4.3 discussed the uncertainty associated with the ingestion of residential soil during outdoor activities. The ingestion rate of creek sediments would be expected to be lower, although even more uncertain, because of the reduced period of contact and the intervening layer of water during activities in the water. The amount of water ingested during recreational activities is not known, although one draft study has estimated that 50 ml are ingested during a swimming period. Wading would likely result in a lower water ingestion rate compared to swimming.

Cayuga Creek is the most easily accessed creek because of its length and absence of fencing. The sediment concentrations, however, are the lowest of the three creeks. The concentration of arsenic in Cayuga Creek is increased (mean of 46,000 ug/kg) compared to Black and Bergholtz Creeks (mean about 26,000 ug/kg) because two samples in Cayuga Creek had reported concentrations of 160,000 and 170,000 ug/kg. Except for those two samples, other reported concentrations for arsenic in the three creeks are comparable.

4.5.2 Migration to Residential Yards

A high rate of rainfall and/or a high stage of the Niagara River could produce flooding of the creeks onto the yards of nearby residents. This would lead to deposition of contaminated sediments and mixing with residential soils. The qualitative nature of the human exposure potential is much the same as discussed for surcharged sewer sediments in Section 4.4.3. Soil ingestion and dermal contact could be expected if the material were not immediately removed. The contaminant concentrations, however, would decrease over time through actions of deliberate human removal, natural rain and snow-melt flows, and organic chemical degradation.

4.6 POTENTIAL FOR HUMAN EXPOSURE TO 102ND STREET OUTFALL AND THE NIAGARA RIVER

Potential human exposure routes to the 102nd Street outfall and the Niagara River are much the same as the creek sediments and water. Recreational activities, such as wading or swimming, may lead to the ingestion of contaminated sediment or water, and dermal absorption through contact. Inhalation of volatiles is not considered to be a route of potential

that sanitary sewer sediments may backflow to the houses, and be discharged into the house. Concentrations would have been reduced by the mixing with the sewer liquid. If the discharged material remain undetected, volatile organics will be released within the house and contact with the material may occur through normal activities.

4.4.5 Exfiltration to Groundwater

As discussed in Section 3.2, the potential for exfiltration of water from the sewers has been enhanced by the absence of a drainage system to channel the groundwater away from the pipe. The presence of a shallow groundwater system was discussed in Section 3.5. The rate of exfiltration, if any, is unknown. The effect of fill around the sewers and swales and the Love Canal remedial actions on the flow of groundwater is very uncertain. It is, thus, not possible to carry an assessment of this pathway any further. Several of the target chemicals, including TCDD, are tightly adsorbed to the soil (Table A-4), and therefore will be limited migration with groundwater.

4.5 POTENTIAL FOR HUMAN EXPOSURE TO CREEK CONTAMINANTS

Chemical concentrations in the creeks were discussed in Section 2.4. No volatile organics were detected in creek sediments or water, so inhalation is not a major exposure route for volatile organics. Discharging of storm sewer sediments, which did contain some volatile organics, to the creeks has the potential for localized chemical concentrations. Migration to the atmosphere appears to be rapid, as sewer discharges have not led to detectable volatile concentrations in the creeks in the Malcolm Pirnie study. Atmospheric dispersion in the open air would reduce concentrations substantially.

Human contact with contaminated sediments would occur during swimming, wading, or other recreational use of the creeks. It is also possible that flooding could transport creek sediment to residential yards, as many residences abut the creeks. Dried sediments on the creek banks may become entrained into the atmosphere, leading to potential human exposure through inhalation. The chemical contribution of creek sediments to the Niagara River will be discussed in Section 4.6. Over time, the concentrations in the creek sediments would be expected to decline because of downstream transport and degradation of the organic chemicals.

4.5.1 Recreational Activities

Access is limited along Black and Bergholtz Creeks because fencing extends from the confluence of Black and Bergholtz Creeks from 150 feet upstream to 650 feet

has been estimated that the sanitary sewers have 200 cubic yards and the storm sewers have 80 cubic yards of sediment. With the mean TCDD concentrations from Tables 2-1, 2-3, and 2-4, the total quantity of TCDD in the sanitary sewers is estimated to be 0.03 ounces (0.8 g), and the total in the storm sewers is estimated to be 0.003 ounces (0.09 g) using the Malcolm Pirnie concentrations, and 0.02 ounces¹ (0.5 g) using the EPA-ORD monitoring study results.

The human chemical dose would also depend on the length of time that the chemical remained in the yard or on the street. The concentrations would decrease through the actions of: deliberate washing by residents or city services after the surcharging is noted; natural washout through rain and snow-melt runoff; and the natural degradation of organic chemicals.

The soil ingestion rate is difficult to estimate and will likely vary with age because of changes in behavior (e.g., pica in young children), and the length of contact. This uncertainty is reflected in the range of soil ingestion rates for children recommended by the USEPA: 0.1 to 5 g soil

per day for children ages 2 to 6 years (USEPA, 1984c). Kimbrough, et al., (1983) have recommended age specific rates that vary from 10 g/day for children 1.5 to 3.5 years old to 0.1 g/day for people over 5 years. The USEPA has recommended a dermal absorption fraction of 0.0007 to 0.03 for TCDD contained in the dust on skin (USEPA, 1984c). The total amount absorbed would also be a function of the area of exposed skin on which dust could settle. Dried sediment may become entrained into the atmosphere, leading to potential exposure through inhalation.

For both the ingestion and dermal absorption routes, the magnitude of the dose depends on the duration of exposure, which is a function of the period outdoors in contact with contaminated materials. If contaminated soil enters the home (e.g., dirty shoes and clothes, entrained dust in the atmosphere), then the exposure will continue in the house.

If a garden soil becomes contaminated, then the contaminants may be taken up by the plants and ingested by humans if vegetables or fruits are grown.

4.4 Backflow of Sanitary Sediments to Basements

Backflow preventers were probably not installed in houses of the age around Love Canal. Therefore, the potential exists

¹ Assuming 3,000 lb/yd³.

data collected in 1983. EPA-ORD data from 1980 on the storm sewers gave maximum and mean sediment concentrations of 5,000 and 200 ug/kg. Sediment chlorobenzene maximum and mean concentrations were 78,000 and 4,500 ug/kg in the sanitary sewers. In the storm sewers, the maximum and mean concentrations were 55,000 and 7,500 ug/kg with Malcolm Pirnie data, and 4,600 and 200 ug/kg with EPA-ORD data. Other detected volatiles in the sanitary sewer include one sample that had 1,1,1-trichlorethane and trichlorofluoromethane at less than 4,000 ug/kg each. In the storm sewer, other detected volatiles included one sample with ethylbenzene and benzene (both at the detection limit of 2,000 ug/kg).

Sewers do not receive regular maintenance, and workers would enter the system only if problems are reported. Prior to sewer entry, the manholes would be opened and air circulated through to reduce volatile gas concentrations. Normal health and safety practice for typical sewers would not have respirators and special suits in use. Higher level protection would be used, however, for entry into the EDA sewers to minimize worker exposure.

Section 4.4.1 discussed the presence of volatile organics in the sewers. Volatile emissions through manholes, or the outfalls will be dispersed by winds and atmospheric turbulence. This will reduce the potential for the public to inhale the volatile organics in the sewers.

4.4.3 Surcharge of Sediments to the Surface

Sections 3.2 and 3.3 discussed the evidence for past surcharging of the sewers. Surcharging of sanitary sewers to the surface has been observed in the area of 91st, 92nd, and 93rd Street and Read Avenue during high rainfall periods. Surcharging within manholes has occurred elsewhere. Surcharging to the surface has been observed frequently from the storm sewers along 93rd Street.

Surcharging to the surface results in the possibility that the material may run onto a residential yard or remain in the street. No TCDD was detected in the area of the reported surface surcharging around Read Avenue (Section 3.2), but TCDD (6.3 ug/kg at MH-765) was reported at an upgradient manhole. During the process of surcharging, the concentration of the contaminants would be expected to become diluted as the materials mix with the water, and therefore, the chemical concentration of the deposited material would be lower than that of the sewers. It has not been possible to estimate a range of dilution factors within the scope of this project.

The size of the sediment deposit on a yard or street would depend on local flow and topographical characteristics. It

- o Sewer maintenance. Workers may be exposed to chemicals through inhalation, ingestion or dermal contact.
- o Migration of volatile organics. Volatile emissions through manholes or outfalls could expose the public through inhalation.
- o Surcharge of sewer sediments. Surcharging would transport contaminated sediments to the surface soil and roads. Public exposure could occur through inhalation, ingestion or dermal absorption.
- o Backflow of sanitary sewer sediments to basements. This route would transport sediments to the basements of local residents. Public exposure could occur through inhalation, ingestion or dermal absorption.
- o Exfiltration to groundwater. The loss of contaminated material through sewer leaks would contaminate the surrounding soil and the shallow groundwater. Subsequent public exposure could occur through inhalation, ingestion or dermal absorption if the groundwater was used.

Each of these routes will be examined qualitatively in the following subsections. Discharge of materials to the creeks will be discussed in Section 4.5 and to the 102nd Street outfall in Section 4.6.

Sediment chemical concentrations in the sewers are expected to decrease with time because of the transport of contaminated sediment downgradient and dilution by clean sediments, as discussed in Section 3.1. Love Canal specific chemical contributions to the sewers are not expected because the lines from the immediate Love Canal area to the EDA have been plugged. Degradation of organic chemicals would also be expected, although photolytic degradation would not occur. As discussed in Section 2.3, the concentrations of TCDD, BHC isomers and hexachlorobenzene have apparently decreased during the 2.5 years between the two sampling periods.

4.4.1 Sewer Maintenance

The detected sediment concentrations of the volatile organics toluene and chlorobenzene were summarized in Sections 2.2 and 2.3 for the sanitary and storm sewers, respectively. The maximum and mean concentrations for toluene were 35,000 and 4,300 ug/kg in the sanitary sewers, and 280,000 and 16,000 ug/kg in the storm sewers with the Malcolm Pirnie

Houses abut the west side of Bergholtz Creek in the study area except for a large open field which is part of the 93rd Street School, just below the confluence with Black Creek. Recreational use of the creek by children was observed during the Malcolm Pirnie investigation.

Cayuga Creek presents a similar set of exposure routes as described above. The length of the creek, the accessibility and increased number of inhabitants along the creek increases the potential for human exposure from this pathway. The west bank of Cayuga Creek abuts Cayuga Drive. At the southern end, a commercial area has several buildings built into the creek bank itself. Backyards abut the east bank with houses set back from the bank. Access to the creek is available and it is used for recreation (Malcolm Pirnie, 1983).

Recreational use of the Little Niagara River may provide for dermal contact with, or ingestion of, contaminated waters or sediments. The potential for ingestion of contaminated drinking waters by the City of Niagara Falls exists since the city water supply intake is 2.5 miles downstream of the Cayuga Creek/Little Niagara confluence.

Potential routes of exposure from the outfall at 102nd Street and the Niagara River include dermal contact with sediments and waters from recreational use, ingestion of aquatic organisms from the area and inhalation of volatiles from disturbance of sediments near the shore and from waters of the outfall.

4.3 POTENTIAL RECEPTORS

Potential receptors are those persons living or working in and adjacent to the EDA, populations that may come into primary or secondary contact with contaminated surface waters of sediments, downstream populations, recreationalists on the Niagara River in the vicinity of the 102nd Street outfall, users of the public docks and private marina immediately downstream of the 102nd Street outfall, recreationalists on the Little Niagara River at the confluence of Cayuga Creek, and downstream users. Attachment B contains a detailed listing of potential receptors. Some people, such as local residents, may be exposed to site chemicals through multiple sources. For example, a resident may be exposed to contaminants from creek sediments and fish.

4.4 POTENTIAL FOR HUMAN EXPOSURE TO SEWER MATERIALS

The sediment chemical concentrations were summarized in Sections 2.1 and 2.2 for the sanitary and storm sewers, respectively. Several chemical migration pathways offer the potential for human exposure to chemicals in the sewers.

- o Contact with surcharged sewer material;
- o Contact with creek sediments and water;
- o Contact with contaminated material at the 102nd Street outfall;
- o Ingestion of contaminated creek (recreational activities) or river water (ingestion of drinking water);
- o Ingestion of fish, which may bioaccumulate contaminants deposited in creek or river sediments.

Chemical concentrations in the creeks and rivers may also present exposures potentially harmful to aquatic species.

Surcharging of sewer lines, particularly sanitary sewer lines, may back up flow into residences or onto surface soils in the EDA. In that event, inhalation, dermal contact and ingestion would be the primary exposure routes.

Sanitary sewer lift stations 4 and 6, which ultimately receive all flows from the area, are also a potential source of volatilization of contaminants and a potential route of inhalation exposure. Pipes further downstream may be contaminated from the flow of material. Storm sewer catch basins within the EDA are a potential source of volatilization of contaminants during surcharging and therefore are a potential inhalation exposure route.

Access is limited along Black Creek because of fencing on both banks. Potential primary exposure routes would be inhalation of volatile contaminants at the outfall on 96th street, and dermal contact with waters and sediments from flooding, by breaching the fence, or by going around the fence in areas where the fence is not secure.

Bergholtz Creek from its confluence with Black Creek presents several exposure routes. It is fenced from 150 feet upstream of the confluence to 600 feet downstream. Dermal contact with sediments from swimming, wading, or other recreational use of the creek is possible along portions of its 3,900 feet length to the confluence with Cayuga Creek or dermal contact with floodwaters. Secondary exposure routes include dermal contact with pets or objects that may have been in contact with contaminated waters or sediments. Ingestion of small amounts of water from swimming, ingestion of contaminated aquatic life caught from the creek or ingestion of sediments in the creek, as with children that exhibit pica, are viable exposure routes. Inhalation of volatilized contaminants from the outfall at 93rd Street is also a potential exposure route.

Table 4-1
HEALTH CRITERIA, GUIDELINES, AND STANDARDS FOR VARIOUS CONTAMINANTS

Contaminant	Drinking Water Standard (µg/L)	EPA Water Quality Criteria (µg/L)			Residential Soil Concentration (µg/kg)	Acceptable Daily Intake (mg/day)	Cancer Potency (d) (kg-day/mg)
		Drinking Water	Fish and Drinking Water ^c	Freshwater Aquatic Life			
2,3,7,8-TCDD		2.2×10^{-7} (f)	1.3×10^{-8} (e,f)		1 (g)		156,000
α-BHC		0.013 (f)	0.0092 (f)	0.08 ave., 2.0 max.			11.12
β-BHC		0.023 (f)	0.013 (f)				1.84
γ-BHC	4	0.026 (f)	0.0186 (f)				1.33
Chlorinated benzenes				250			
Chlorobenzene		20 (h)				1	
Dichlorobenzenes				1,120 acute, 763 chronic			
1,2,4-Trichlorobenzene							
Hexachlorobenzene		0.021 (f)	9.2×10^{-4} (f)				1.67
Toluene		15,000	14,300	17,500		30	
Arsenic	50	0.0025 (f)	0.0022 (f)	72 ave., 140 max.			15
Cadmium	10	1.2 (f)	1 (f)	(1.16 In(hardness)--3.841)		0.17	
Thallium		18	13			0.037	

(a) EPA, 1980.

(b) For ingestion of 2 L per day.

(c) For ingestion of 2 L and 6.5 g of fish and shellfish per day.

(d) U.S. EPA, 1984d.

(e) U.S. EPA, 1984a.

(f) 10⁻⁶ excess lifetime cancer risk.

(g) Level of concern. Kimbrough, et al., 1983.

WDR101/006

Arsenic. Mutagenic changes have been noted in animal cells following exposure to arsenic. Ingestion of contaminated water can result in changes in immune function, cardiovascular disorders, peripheral vascular disorders, and nerve degeneration in the peripheral nervous system. Arsenic exposure has been linked to cancer and reproductive effects in humans. Dermal exposure can lead to skin reddening and formation of skin eruptions.

Cadmium. Primary effects of chronic exposure to inhaled cadmium are pulmonary emphysema and renal tubular damage. Hypertension may be an early symptom of exposure. Recent evidence indicates inhaled cadmium acts to induce lung cancer.

Thallium. Thallium has been used for medicinal purposes, but can produce toxic effects when large doses (200 to 1,000 mg/kg body weight) are ingested. Effects of overexposure include neurological disorders, gastrointestinal disorders, hair loss, kidney damage, liver damage and death. Hair loss is a symptom of low dose exposure.

The toxicological profiles above are based on exposure to only one chemical, and synergistic and antagonistic effects have not been considered. The state of toxicological knowledge is insufficient to describe the potential effects of multiple exposure to a variety of contaminants, and this assessment assumes that the effects are additive.

The contaminants may be grouped into two primary health effects categories, those that are known or suspected carcinogens and those that are not. TCDD, hexachlorobenzene, the α , β , and γ isomers of BHC and arsenic belong in the carcinogen group and the rest of the chlorinated benzenes, toluene, ingested cadmium, and thallium were evaluated as noncarcinogens. It is believed that an incremental increase in exposure to a carcinogen will add an incremental increase in the probability of getting cancer. In contrast exposure to other chemicals is often a threshold phenomenon, in which a specific dose, or threshold, must be exceeded before a biological effect will occur.

Table 4-1 summarizes the various standards, criteria and recommendations on acceptable levels of contaminants in various environmental media.

4.2 EXPOSURE PATHWAYS AND ROUTES

The principal pathways of human exposure to contaminated waters and sediments are:

- o Inhalation of volatile organics from sewers, creeks, or rivers;

The potential population that could occupy present housing within the EDA numbers is 2,250 persons. This figure is based on the number of residences currently standing and an assumed four persons per household. Of these, 128 persons could occupy housing adjacent to either the free flowing portion of Black Creek or Bergholtz Creek. This potential population would be receptors if no action is taken on contaminants in sewers and creeks and the area is rehabited.

Along the north bank of Bergholtz Creek, outside the EDA, Cayuga Drive roughly parallels the creek about 50 to 300 feet to the north. Two houses are located roughly opposite the upstream EDA boundary (101st Street) and are within about 60 to 100 feet of the creek. Six houses are located approximately opposite the confluence of Black Creek with Bergholtz. All of these buildings are within at least 200 feet of the creek. Two houses are located opposite the school property, one about 50 feet from the creek and the second about 100 feet. In total, ten houses are located between Cayuga Drive and Bergholtz Creek adjacent to the creek.

Bergholtz Creek flows approximately 1,800 feet past the western EDA boundary (93rd Street) before entering Cayuga Creek. Cayuga Drive continues to parallel the north creek bank and is no more than about 50 feet from the creek. One commercial building is located on the north bank near the intersection of Cayuga Drive and 91st Street. Within this 1,800 feet reach, three houses are located adjacent to Bergholtz Creek on the south bank. Five other are about 70 feet or more from the creek bank. Eight other houses located within about 70 feet of the south creek bank are separated from Bergholtz Creek by Brookside Avenue.

A potential population of 104 persons lives adjacent to Bergholtz Creek outside the EDA. This number is derived from a total of 26 residences and an average of four persons per residence.

Cayuga Creek flows approximately 4,900 feet south from its confluence with Bergholtz Creek before entering the Little Niagara River. This creek is outside the EDA. Cayuga Drive parallels much of the west creek bank, and upstream of Military Road is no more than 50 feet from the creek itself. There are no buildings between Cayuga Drive and the creek in this 3,400 feet reach. In the remaining 1,500 feet of Cayuga Creek, downstream of Military Road, 11 houses and 11 commercial buildings are located adjacent to the west creek bank. Many are within about 10 feet of the creek.

A total of 42 houses and three commercial buildings are located along the east bank of Cayuga Creek downstream of its confluence with Bergholtz Creek. Approximately

32 houses and one commercial building are located along the 3,400 feet reach upstream of Military Road. Most of these buildings are within about 80 feet of the creek. Ten houses and two commercial buildings are located along the east bank of Cayuga Creek in the 1,500 feet reach downstream of Military Road. Several of these buildings abut the creek and are built into the bank.

A potential population of 212 persons live adjacent to Cayuga Creek. This number is derived from a total of 53 residences and an average of four persons per residence.

The population of Cayuga Island are potential receptors because of the location of the island between the 102nd Street outfall and the mouth of Cayuga Creek. Cayuga Island is easily flooded; contamination could be brought ashore with flood waters. Approximately 90 percent of the island is in the 100-year flood plain.

An aerial photograph showing the eastern two-thirds of the island indicates no buildings are present between Joliet Avenue, which extends along the north shore, and the Little Niagara River. Distance between the river and Joliet Avenue varies from about 125 to 475 feet. Several baseball diamonds and other recreational facilities are present in this area. Council Avenue extends about 1,200 feet along the island's eastern shore. Distance between the avenue and the Little Niagara River varies from about 70 to 170 feet. Sixteen residences are located between Council Avenue and the river. West Rivershore Drive is the main road on the western half of the island, extending approximately 3,600 feet. No estimates are available on the number of residences along this drive.

Many homes fronting the Niagara River on the south shore of the island have small docks. No estimates are available on the population of Cayuga Island.

Workers for the City of Niagara Falls are potential receptors. Maintenance workers who service the sewer lines, repair possibly surcharged manholes, or who service ground utilities, are potential receptors.

The City of Niagara Falls has a 1980 population of 71,384 persons which are potential receptors for potentially contaminated water supplies. No estimates are available on the numbers of recreationalists that utilize the areas adjacent to the 102nd Street outfall, or the public dock or private marina on the Niagara downstream of the outfall. No estimates are available on the number of persons who may consume fish from the creeks and rivers and would be potential receptors of contamination.

WDR101/10

REFERENCES

- Black, S. C. December 1984 and January 1985. U.S. Environmental Protection Agency, Las Vegas, Nevada. Personal Communication.
- Blair, H., Ed., 1973, Chlorodioxins - Origin and Fate, American Chemical Society, Washington, D.C.
- Bowen, H.J.M. 1979. Environmental Chemistry of the Elements. Academic Press, New York, N.Y.
- Camp, Dresser & McKee. 1975: Report to City of Niagara Falls, New York on LaSalle infiltration/inflow analysis. C-36-747, Boston, MA. U.S. Environmental Protection Agency. 1982. Environmental Monitoring at Love Canal, Vol. 1. Office of Research and Development, EPA-600/4-82-030a, Washington, D.C. 296p.
- Clayton, George D. and Florence E. Clayton, Eds., 1981, Patty's Industrial Hygiene and Toxicology Third Revised Edition Volume IIB Toxicology. John Wiley & Sons. New York.
- Crosby, D. G. 1983. "Methods of Photochemical Degradation of Halogenated Dioxins in View of Environmental Reclamation." In F. Coulston and F. Pocchiari. Accidental Exposure to Dioxin, Human Health Aspects. Academic Press, N.Y.
- Environment Canada and the Ontario Ministry of the Environment. June 1980. Environmental Baseline Report of the Niagara River.
- Environment Canada and the Ontario Ministry of the Environment. November 16, 1981. Environmental Baseline Report of the Niagara River--November 1981 Update.
- Federal Emergency Management Agency. 1982. Flood Insurance Study, City of Niagara Falls, New York. FEMA Community No.-360506, Washington, D.C.
- GEOTRANS. 1981. Final Report on Groundwater Flow Modeling Study of the Love Canal Area, New York.
- Gosselin, Robert E., Smith, Roger P., Hodge, Harold C., and Braddock, Jeanette E., 1984, Clinical Toxicology of Commercial Products, Fifth Edition, Williams and Wilkins, Baltimore, Maryland.
- Heath, C. W., R. Kimbrough, J. Liddle, D. P. Rall, T. Damstro and W. Rogan. 1981. HHS Evaluation of Results of Environmental Chemical Testing Performed by EPA in the

Vicinity of Love Canal: Implications for Public Health.
U.S. Department of Health and Human Services.

Heath C. W., R. D. Kimbrough, J. A. Liddle, D. P. Rall and W. J. Rogan. July 13, 1982. HHS Evaluation of Results of Environmental Chemical Testing Performed by EPA is the Vicinity of Love Canal--Implications for Human Health--Further Considerations Concerning Habitability. U.S. Department of Health and Human Services.

IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man, Some Malogenated Hydrocarbons
Volume 20, 1979, World Health Organization, Geneva, Switzerland.

Kabata-Pendias, A. and H. Pendias. 1984. Trace Elements in Soils and Plants. CRC Press, Boca Raton, Florida.

Kimbrough, R.D., Henry Falk, Paul Stehr and Geroge Fries. 1983. Health Implication of 2,3,7,8-Tetrachlorodibenzodioxin (TCDD). Center for Environmental Health, Center for Disease Control, Atlanta, GA.

Kolbye, Jr., A. 1983. "Mechanisms of Carcinogenesis Related to TCDD. In F. Coulston and F. Pocchiari. Accidental Exposure to Dioxin, Human Health Aspects. Academic Press, N.Y.

Malcolm Pirnie, Inc. 1983. Environmental Information Document, Site Investigation and Remedial Action Alternatives Love Canal. Division of Solid and Hazardous Waste, New York State Department of Environmental Conservation, Albany, NY.

Miller, S.A., Director of Bureau of Foods, Food and Drug Administration. June 30, 1983. Statement to the House Subcommittee on National Resources, Agriculture Research and Environment.

National Toxicology Program, 1983. Third Annual Report on Carcinogens, Summary, NTP BJ-010, Research Triangle Park, NC.

Olie, K., P.L. Vermeulen, O. Hotzinger, 1977, "Chlorodibenzo-p-dioxins and Chlorodibenzofurans are Trace Components of Fly Ash and Flue Gas of Some Municipal Incinerators in the Netherlands", Chemosphere No. 8, pp 455-459.

Sittig, Marshall, Ed., 1980. Priority Toxic Pollutants: Health Impacts and Allowable Limits, Noyes Data Corporation, Park Ridge, New Jersey.

Stalling, D. L. U.S. Fish and Wildlife Service, Columbia, Missouri. January 18, 1982. Letter to L. Skinner, New York Department of Environmental Conservation.

Suns, K., G. R. Graig, G. Crawford, G. A. Rees, H. Tosine .. and J. Osborne, 1983. Organochlorine Contaminant Residues in Spottail Shiners (*Notropis Hudsonius*) from the Niagara River. J. Great Lake Research, Vol. 9, pp. 335-340.

Threshold Limit Values for Chemical Substances in the Work Environment Adopted by ACGIH for 1984-85, American Conference of Governmental Industrial Hygienists Cincinnati, Ohio.

U.S. Department of Labor - Occupational Safety and Health Administration. Occupational Exposure to Inorganic Arsenic; Supplemental Statement of Reasons for Final Rule. Friday, January 14, 1983, Vol. 48, No. 10, United States Department of Labor, Washington, D.C.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Criteria for Arsenic. EPA-440/5-80-021, Washington, D.C.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Criteria for Cadmium. EPA-440/5-80-025, Washington, D.C.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Criteria for Thallium. EPA-440/5-80-059, Washington, D.C.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Criteria for Hexachlorocyclohexane. EPA-440/5-80-041, Washington, D.C.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Criteria for Chlorinated Benzenes. EPA-440/5-80-014, Washington, D.C.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Criteria for Toluene. EPA-440/5-80-075, Washington, D.C.

U.S. Environmental Protection Agency. 1982. Environmental Monitoring at Love Canal. EPA 600/4-82-030a, Washington, D.C.

U.S. Environmental Protection Agency. 1983. Revised Section B of Ambient Water Quality Criteria for Arsenic. Washington, D.C.

U.S. Environmental Protection Agency. 1983. Revised Section B of Ambient Water Quality Criteria for Cadmium. Washington, D.C.

U.S. Environmental Protection Agency. 1983. Health Assessment Document for Toluene. Office of Research and Development, EPA-600/8-82-008F, Research Triangle Park, NC.

U.S. Environmental Protection Agency. 1984a. Ambient Water Quality Criteria for 2,3,7,8-Tetrachloro dibenzo-p-dioxin. Environmental Protection Agency, Office of Water Regulations and Standards, EPA-440/5-84-007, Washington, D.C.

U.S. Environmental Protection Agency. 1984b. Health Assessment Document for Inorganic Arsenic. EPA-600/8-83-021F, Washington, D.C.

U.S. Environmental Protection Agency. 1984c. Risk Analysis of TCDD Contaminated Soil. Office of Research and Development, Washington, D.C.

U.S. Environmental Protection Agency. 1984d. Health Assessment for Epichlorohydrin. EPA-600/8-83-032F. Washington, D.C.

Walter, N. February 8, 1985. New York Department of Environmental Conservation. Personal communication.

WDR65/67

Appendix C
CHEMICAL AND TOXICOLOGICAL PROPERTIES OF THE
CHEMICALS OF CONCERN

INTRODUCTION

This section discusses the chemical and toxicological properties of seven chemicals or groups of chemicals that were selected as target chemicals for assessment. These target chemicals were selected based on the frequency with which they were found during the 1983 Malcolm Pirnie sampling, and the concentrations of these chemicals in the environment at Love Canal. These seven target chemicals or groups of chemicals follow:

- o 2,8,7,8-tetrachlorodibenzo-p-dioxin, (TCDD or dioxin);
- o isomers of hexachlorocyclo hexane
- o chlorinated benzenes;
- o toluene;
- o arsenic;
- o cadmium; and
- o thallium

CHEMICAL PROPERTIES

TCDD

There are theoretically 75 different chlorinated dibenzo-p-dioxin structures with varying degrees of chlorination. The most toxic dioxin that has been isolated and tested is 2,3,7,8-tetrachlorodibenzo-p-dioxin, commonly referred to as "TCDD" or simply "dioxin."

TCDD is inadvertently synthesized in concentrations of a few parts per million or less during industrial processing of chlorinated phenols, a widely used class of industrial organic chemicals. Elevated temperatures during chlorinated phenol processing have been shown to increase the formation of TCDD. TCDD is also generated during the combustion of chlorinated phenols. Some research suggests that TCDD may form in refuse incinerators, fossil-fueled power plants, gasoline and diesel-powered motors, fireplaces, charcoal grills, and cigarettes; however, more research is needed to verify these findings.

TCDD was first intentionally prepared in 1872. It was not until 1957 that TCDD was synthesized and isolated from other chlorinated dioxin isomers. Since then, researchers have investigated various aspects of TCDD generation, environmental behavior, toxicity, and destruction. Most of this background data, especially the toxicity data, pertains to solubilized TCDD (a relatively mobile form), with only limited data applicable to TCDD complexed in soil. Several studies have indicated that the behavior of TCDD in the environment can change substantially depending on the nature of the TCDD-containing matrix.

GENERAL CHARACTERISTICS OF TCDD

TCDD is a highly symmetrical and stable nonpolar chlorinated organic compound that lacks chemically reactive functional groups. Pure TCDD is a colorless crystalline solid with an approximate melting point of 305°C and is believed to have a low vapor pressure of roughly 10^{-6} mm of mercury at room temperature. It is commonly accepted that TCDD has a low water solubility at room temperature of approximately 0.2 parts per billion. TCDD is categorized as a lipophilic compound, which means that it has a relative affinity for fats, waxes, and related organic compounds. TCDD solubilities have also been reported at 10 ppm in methanol, 40 ppm in lard, 570 ppm in benzene, and 1,400 ppm in ortho-dichlorobenzene.

SUMMARY OF TCDD BEHAVIOR IN THE ENVIRONMENT

This summary of TCDD behavior in the environment focuses on TCDD in soil. Several studies have indicated that the behavior of TCDD in the environment can change substantially depending on the nature of the TCDD-containing matrix. Research in the past 5 years has shown that TCDD binds with soil and becomes increasingly difficult to extract with time. This binding mechanism is not precisely understood, but is generally believed to be either a physical adsorption on soil particle surfaces or a chemical bonding with the soil humus fraction.

Leachate characterization studies conducted by the EPA have verified the soil binding phenomenon. In the studies, five TCDD-contaminated soil samples with TCDD concentrations ranging from 118 to 780 ppb were vigorously mixed with deionized water for 24 hours, decanted, and sequentially filtered to 0.45 microns. The filtrate samples were analyzed for their TCDD concentrations after each filtration stage. Of the five samples analyzed after the 0.45-micron filtration, four samples had nondetectable levels of TCDD. Duplicate analyses for the remaining sample showed TCDD concentrations of 6 ppt and 12 ppt. EPA is conducting additional leachability tests to determine more accurately TCDD's

leachability from various soil matrixes and to develop appropriate leachate treatment methods for TCDD contamination storage facilities.

Physical Movement in the Environment

The potential for TCDD in soil to leach into groundwater appears to be low due to its strong soil binding and its low solubility in water. Evaporation of TCDD is not well understood, but appears to be quite low, especially when in soil.

It appears that the primary mechanism for movement of TCDD in soil is displacement of contaminated soil particles. This can occur by wind or water erosion, contact with people, animals, or vehicles, or by intentional movement of the soil. In these ways, dioxin in soil may spread across an area and into the air or surface water.

BIOLOGICAL PATHWAYS

It appears that plant uptake of TCDD has shown mixed experimental results that have not been resolved. However, several researchers believe that plant uptake of TCDD that is bound in a soil matrix is not significant.

TCDD-contaminated soil is introduced into animals or humans through the respiratory tract, the skin, and the digestive tract. Recent animal research has shown that the absorption of TCDD into the body through the skin or the intestinal tract is reduced when it is mixed with soil. This absorption was found to further decrease as the time of contact between TCDD and soil was increased.

Studies have shown that TCDD in aquatic systems can be bioconcentrated in fish to approximately 6,000 times the surrounding water concentration. According to Dr. Stalling of the Columbia National Fisheries Research Laboratory, research indicates that TCDD in soil that is translocated into streams may be available at significant levels for fish uptake and concentration.

ENVIRONMENTAL DEGRADATION

Biodegradation of dioxin in soil has not been demonstrated to any significant level. Although several previous studies indicated that appreciable biological degradation of TCDD in soil did occur, more recent studies have shown, and several researchers of previous studies have also concurred, that many of these results were likely due to inadequate analytical procedures and experimental controls.

Thermal degradation of TCDD in soil is essentially nonexistent in the environment. Pure TCDD is not appreciably

decomposed by laboratory incineration at 700°C. TCDD bound to particulates has been said to resist thermal decomposition at incinerator temperatures up to 1000°C.

TCDD bound in soil does not exhibit any significant ultraviolet degradation. However, substantial ultraviolet degradation of TCDD will occur in several hours to a few days in the surface soil layers if TCDD is solubilized in a thin light-transmitting phase with a hydrogen donor present and exposed to sunlight or artificial ultraviolet light.

In summary, TCDD is very persistent. Natural environmental degradation does not occur for all practical purposes. This is evidenced by its presence at a constant level in the soils in Times Beach and other Missouri sites after more than 10 years.

PREVIOUS DIOXIN CASE STUDIES

Since 1949, more than 23 major industrial accidents involving solubilized dioxin have been reported around the world. In addition, many people have been exposed to solubilized dioxin in the chemical manufacturing industry and through transportation accidents, herbicide applications, waste handling, and chemical laboratory work. The following paragraphs summarize some of the major reported dioxin-exposure incidents and the public response to these hazards.

Seveso, Italy. On July 10, 1976, an accident at the Givaudan factory ICMESA 2,4,5-TCP plant released a dioxin-contaminated chemical cloud that showered down on a farmland area of about 6,000 acres, inhabited by approximately 40,000 people. Thousands of animals died and approximately 134 people in the three designated zones of contamination developed chloracne (a skin disorder believed to be the most sensitive indicator of dioxin exposure in humans; it is most prevalent on the face, neck, and arms).

Some of the initial remedial measures implemented at Seveso included collecting and isolating contaminated materials, evacuating residents in contaminated areas, implementing measures to avoid contact with contaminated materials, and decontaminating affected materials. Numerous measures were studied for ultimate destruction of the contaminated material, including incineration, ultraviolet degradation, biological degradation, and chemical destruction. None of these options were considered viable for effectively treating the large volumes of contaminated soil. Removing the contaminated materials to offsite disposal site(s) was not considered acceptable either. Onsite disposal of contaminated soil in secured basins was considered the most practical option. This method has been used for disposing of the soil in the more highly contaminated zones.

Missouri Horse Arenas. In 1971, waste oils containing TCDD were sprayed for dust stabilization in three east-central Missouri horse arenas. In the most affected arena, 54 out of 57 horses exposed to the waste oil developed similar illness symptoms and died. Dead birds, cats, dogs, rodents, and insects were also found in and around the arena. A 6-year-old girl who played regularly in the arena became very sick and lost 50 percent of her body weight. Five years later, she had apparently recovered.

The arena co-owners and their 10-year-old daughter have experienced severe headaches, nausea, and other symptoms. Human illness and animal illnesses and deaths occurred at the other two horse arenas. All three horse arenas were sprayed within one month of each other with waste oil from Bliss Waste Oil Company. That same company later sprayed dioxin-contaminated oil on unpaved streets in Times Beach. The source of the TCDD-contaminated oil was traced to the Northeast Pharmaceutical and Chemical Company (NEPACCO) in Verona. Analysis of a distillation residue, similar to that mixed in with the waste oil, revealed the residue contained 306 to 356 ppm TCDD. The Shenandoah Stables horse arena may have been sprayed with undiluted still bottoms.

Soil from one of the arenas was later analyzed, and contained 31 to 33 ppm TCDD. The soil from each of these arenas was excavated and landfilled at various other Missouri sites. No further TCDD-related animal deaths or human illnesses have been reported since the soils were excavated.

Syntex--Verona, Missouri. In 1974, Syntex Agribusiness discovered a tank containing 4,300 gallons of distillation bottoms sludge contaminated with 343 ppm dioxin. The sludge was a byproduct formed in the production of hexachlorophene by NEPACCO, the previous facility occupants. Syntex implemented initial measures to provide security and safety for the storage tank and began investigating alternatives for destroying the dioxin, including treatment in a high-pressure reactor, incineration, treatment with a chemical process, and ultraviolet photolysis. After extensive review, a solvent extraction-ultraviolet photolysis process was selected and implemented for destroying dioxin in the waste sludge. The cost of equipment and installation exceeded \$500,000. After processing, the sludge contained 0.1 to 0.5 ppm dioxin, six thousand gallons of process solvent were contaminated with 0.15 ppm dioxin, and salt crystals from the evaporated neutralized brine were contaminated with 20-80 parts per trillion. All of these wastes are currently stored onsite, awaiting final destruction or disposal.

Other dioxin contamination has been discovered and is suspected around the Verona facility. This site is currently on the National Priority List.

Denney Farm Site, Missouri. In 1979-80, a trench at the Denney Farm site in southwest Missouri was found to contain approximately 90 barrels of dioxin-contaminated wastes disposed of by NEPACCO. Syntex Agribusiness, the EPA, and the Missouri Department of Natural Resources developed and implemented a cleanup program. The drums and soils contaminated from drum leakage were excavated and placed into storage in onsite concrete vaults at a cost of over \$1.8 million. The contaminated materials currently await final disposal or destruction.

Neosho Digester--Neosho, Missouri. A digester at the Neosho municipal wastewater treatment plant was used to store wastewater from NEPACCO during 1970 and 1971. In 1977, during a plant renovation, this digester was filled with gravel, soil, and debris. The digester overflowed and contaminated the ground nearby. The contaminated soil was excavated and put in a trench near the digester.

The EPA in 1981 sampled the digester and found concentrations as high as 60 ppb of TCDD and 1,000 ppm of TCP. In June 1984, the EPA issued an administrative order to the City of Neosho to cap the site and monitor groundwater.

Neosho Tank Spill Site--Neosho, Missouri. The Neosho Water and Wastewater Technical School conducted treatability studies on NEPACCO wastewater. The tank used to store the wastewater leaked onto the ground.

In 1981, the EPA found a TCDD concentration of 1.9 ppm in samples of the tank's contents. The highly contaminated soil next to the tank was excavated and put in 15 drums. The tank and drums were put in an Army ammunition bunker on the school property. The tank site was capped and fenced.

An EPA administrative order, issued in August 1982 and amended in June 1984, requires proper disposal of the drums and soil from the tank area. This waste is scheduled to be destroyed in EPA's mobile incinerator in the fall of 1984.

Rail Accident--Sturgeon, Missouri. In 1979 a tank car of orthochlorophenol contaminated with 37 ppb dioxin was derailed in Sturgeon, Missouri. Two workers who were involved in cleanup operations were later found to have low levels of dioxin in their blood. In 1982, \$57 million in damages were awarded to 75 workers exposed to dioxin contamination during this cleanup operation. This award was recently repealed and the suit is now awaiting further court action.

Union Carbide--Sydney, Australia. In 1978, dioxin wastes generated by Union Carbide in trichlorophenol manufacturing processes were discovered in garbage sites in three Sydney suburbs. The Australian government implemented a massive health record review of residents in proximity to these sites. This review apparently did not reveal any definite health problems associated with these landfills. It appears that no further remedial actions were implemented.

Agent Orange. Dioxin was found to be a contaminant in Agent Orange, a defoliant used in Viet Nam. At least 4,800 veterans have asked for treatment because of their exposure to the herbicide, and considerable litigation has resulted from these incidents. An out-of-court settlement was recently reached between the veterans and several manufacturers of Agent Orange.

In 1977 more than 2 million gallons of Agent Orange were incinerated in the Pacific Ocean on the incinerator ship Vulcanus. Calculations on the dioxin combustion efficiency showed it exceeded 99.9 percent.

Activated coconut charcoal has been used by the Air Force to reduce the TCDD level from 8 ppm to 0.1 ppm in Agent Orange. The contaminated carbon has been stored with no designated final disposal.

Coalite Chemicals--England. In 1968, dioxin-contaminated chemicals were released in an explosion in a trichlorophenol reactor inside the Coalite Chemicals plant in Derbyshire, England. Seventy-nine workers around the plant reportedly showed signs of chloracne during the next half year. Equipment and other materials found to be seriously contaminated were buried far down an abandoned coal mine shaft. In 1969, two workers developed chloracne during construction of a new building at Coalite. The source of their exposure was traced to a metal vessel that had been salvaged from the contaminated facility. The vessel reportedly had been extensively cleaned.

Phillips-Duphar--Amsterdam, Netherlands. In 1963, dioxin contaminated chemicals were released from a trichlorophenol reactor inside the Phillips-Duphar 2,4,5-TCP plant in Amsterdam. Twenty-eight employees were reported to have chloracne. Extensive measures were implemented to decontaminate and reconstruct contaminated equipment and areas in the building. However, animal toxicity testing showed that dioxin contamination was still present and the plant was demolished. The rubble was embedded in concrete and placed into three barges whose holds were hermetically sealed. The barges were towed out into the Atlantic ocean near the Azores and sunk in deep water.

Badische Anilin and Soda-Fabrik (BASF)--Ludwigshafen, Germany. In 1953, dioxin-contaminated chemicals were released from a trichlorophenol reactor inside the BASF plant in Ludwigshafen, Germany. This resulted in 75 cases of chloracne. Decontamination of the building was attempted. This was determined to be unfeasible and the building was demolished in 1968.

Dow Chemical--Midland, Michigan. In 1964, 49 out of 61 Dow workers exposed to dioxin-contaminated trichlorophenol developed chloracne. Dow is currently being investigated for dioxin contamination around its Midland, Michigan, chemical manufacturing facility.

Vertac--Jacksonville, Arkansas. In 1979, approximately 3,000 barrels of dioxin-contaminated trichlorophenol wastes were found at this site. Some of the barrels were leaking and had contaminated soil and nearby surface water. In 1979, Vertac moved the barrels into an EPA approved shelter at a cost of approximately \$500,000. The final disposition of these wastes has not been determined. The Vertac facility is currently on the National Priority List.

Monsanto--Nitro, West Virginia. In 1949, dioxin-contaminated chemicals were released after a pressure buildup in a trichlorophenol reactor inside Monsanto's Nitro facility. One hundred twenty-two workers reportedly developed chloracne as a result of this contamination.

A study was released in May 1984 of 204 workers exposed to TCDD in cleanup operations at Nitro. The study concluded that, in the 35 years since their exposure, these workers had not developed more health problems than workers not exposed to TCDD.

γ-BHC

Lindane is the common name of the gamma isomer of 1,2,3,4,5,6-hexachlorocyclohexane (BHC), a broad spectrum insecticide of the cyclic chlorinated hydrocarbons. Five isomers of hexachlorocyclohexane exist, but the gamma isomer possess all the insecticidal activity. Prior to 1978, all companies making BHC formulations had various percentages of BHC isomers. After that date, all switched to gamma formulations. Lindane is synthesized by the direct action of chlorine on benzene in the presence of ultraviolet light. This produces a technical grade formulation from which the gamma isomer is isolated by selective crystallization. Lindane is fairly water soluble but has a low residence time in the natural aquatic environment. It is principally removed by sedimentation, metabolism by microorganisms and volatilization. It has, however, been detected in the

finished drinking waters of a small city in Illinois at 4 µg/l. It is not susceptible to photolysis, but can be dehydrochlorinated by alkalies to 1,2,4-trichlorobenzene. In soils and sediments, lindane is slowly degraded by microbial action with a 10 percent degradation after 6 weeks. It is hypothesized that the gamma isomer is changed to the alpha or delta isomers by microbial activity or from uptake by plants, but this has yet to be shown (USEPA, 1980)¹.

CHLORINATED BENZENES

The chlorinated benzenes are a group of cyclic aromatic compounds in which one to six H atoms have been replaced with up to six chlorine substitutes. Twelve different compounds are possible. All have relatively low water solubility, solubility decreases with increasing chlorination, low to moderate vapor pressure, with vapor pressure decreasing with increasing chlorination and low flammability. Their octanol:water partition coefficient is moderate to high and increases with increasing chlorination. This means the higher chlorinated compounds, such as hexachlorobenzene, have a high tendency to adsorb onto sediments. All are considered to be volatile, except for hexachlorobenzene and all are only slightly reactive. The chlorinated benzenes are readily transported in the atmosphere. They are likely to enter the atmosphere as a result of volatilization and/or evaporation from soil or

water. Once in the atmosphere they may be degraded by chemical or sunlight catalyzed reactions to nitro benzenes and/or nitrophenols. Atmospheric residence time is greater for the more highly chlorinated compounds. Chlorinated benzene compounds in the air may also become adsorbed onto particles that then settle or are removed from the atmosphere by rain. Transport of chlorinated benzenes in aqueous systems is limited. Most are readily evaporated from both aerated and nonaerated waters. In a controlled experiment greater than 99 percent of monochlorobenzene, 1,2-dichlorobenzene, 1,4-dichlorobenzene and 1,2,4-trichlorobenzene evaporated in four hours from aerated water and in 72 hours from nonaerated waters. The half-life of evaporation has been calculated using Henry's Law constant and assumptions on water depth, wind speed, ambient temperature, etc. Monochlorobenzene, 1,2-dichlorobenzene and 1,2,4-trichlorobenzene have calculated half-lives of 4.6 minutes, 8.1 minutes, and 0.75 hours, respectively. The fate of chlorinated benzenes left in water has not been completely characterized. It is believed that microbial degradation occurs on the lower chlorinated compounds, but has yet to be demonstrated. All of the chlorinated benzenes have a medium to strong tendency to adsorb onto soils and sediments. This adsorption potential increases with

¹References can be found at the end of Appendix B.

increasing chlorination and increasing organic matter content of the soils and sediments. Once adsorbed, movement through soils is dependant on the soil type and temperature and the characteristics of the leachate. The potential for downward migration is high. Volatilization from porous soils to the atmosphere is a potential transport route. Again, this depends on soil type and temperature (each 10°C increase in temperature increases volatilization by 3.5 times) and the volatility of the compound, keeping in mind that vapor pressure decreases with increasing chlorination. The fate of chlorinated benzenes in soils and sediments is similar to water, only reaction times differ. Little microbial degradation occurs in soils. Chlorophenols are the likely primary product of degradation. Chlorinated benzenes are persistent in soils once adsorbed, with persistence a function of chlorination (USEPA, 1980).

TOLUENE

Toluene is a clear colorless liquid that ranks fourth among all agents listed in terms of the number of people exposed to any single agent. It is the most prevalent aromatic hydrocarbon in ambient air, primarily from auto emissions. Atmospheric concentrations of toluene average 37 ppb in the Los Angeles area. The high volatility and low solubility of toluene leads to its presence and persistence in the atmosphere. It is chemically quite stable in air and its residence time in the atmosphere has been estimated at 1.9 days depending on solar intensity, temperature and local trace gas composition. It is removed primarily by the free radical chain process where OH radicals are added to the ring. Reaction products include acetylene, acetaldehyde, acetone, formaldehyde and formic acid of unknown yield. Photolysis of toluene in a polluted atmosphere (containing NO_x) yields ozone and peroxyacetylnitrate, a strong eye irritant and oxidizer. Toluene is not removed from the atmosphere to any significant degree by rain. Toluene may be persistent in aquatic environment. No data are available on hydrolysis of toluene in aquatic media. Evaporation and volatilization from water surfaces transports it into the atmosphere at a relatively rapid rate. Evaporation half-life for toluene from water 1m deep has been calculated at 5 hours. Volatilization occurs over a few days time. Sedimentation occurs, depending on the organic matter content of the sediments as the octanol:water partition coefficient is moderately low. Adsorption capacity is increased as pH decreases. The fate of toluene in soils has not been thoroughly investigated. It is anticipated a portion of the toluene (38-66 percent) in soils will undergo intermedia transport to the atmosphere by volatilization. The part that remains in the soil may undergo chemical transformation or biodegradation but the relative importance of each is not known. It is known that toluene is susceptible to bacterial

decomposition in soils, dependant on soil properties. One strain of bacteria has been isolated that uses toluene as its sole carbon source. A half-life of 20 to 60 minutes in soil containing degrading bacteria was observed. Toluene may be leached through soil, depending on soil and leachate characteristics. The fate of leached toluene is not known (USEPA, 1980).

ARSENIC

Arsenic can exist in a variety of chemical forms, each with differing physical properties and solubility. It is most commonly found in the pentavalent (arsenate) and trivalent (arsenite) states. In oxidizing conditions arsenite is converted to arsenic acid or arsenate by biological oxidation. Arsenate hydrolyzes in water. Arsenate is found in more basic, aerated soils. Formation of arsenite is favored under conditions of low pH, low dissolved oxygen and low oxidation potential as is found in water logged soils and sediments. Arsenic persists in the environment in some form. Both of the common forms may be precipitated from water by adsorption onto soil colloids. Iron and aluminum strongly fix arsenic to soil colloids. Waters containing high quantities of organic matter may bind arsenic compounds to humic matter. The extent of adsorption by clays and organic matter decreases as pH increases over neutral.

CADMIUM

The most common valence of cadmium in nature is 2+. It occurs principally as a sulfide salt. In water it hydrolyzes to form hydroxide complexes. Precipitation from solution is dependant on pH and the concentration of cadmium. The cadmium ion is precipitated from solution by carbonate, as hydroxide or sulfide ions. The solubility of cadmium compounds in water depends on the nature of the compound and on water quality. Relatively insoluble, cadmium may be mobilized by complexing with anions. Cadmium is strongly adsorbed to clays, muds, and organic matter. Sorption processes account for removal of dissolved cadmium to sediments. The process is more effective as pH increases (USEPA, 1980).

THALLIUM

Thallium exists in nature in either the monovalent, thallos or trivalent, thallic state. The monovalent form is more common and stable with a large variety of compounds. A large percentage of the thallos salts are soluble in water. Thallic salts are readily reduced by common reducing agents to thallos salts. Thallium is chemically reactive with air and water, oxidizing slowly in air at 20°C. As temperatures increase so does the rate of reaction. Moisture enhances

the reaction at any temperature. Thallium may be leached through soils into the groundwater. Cycling of thallium through environmental media is not well understood nor well documented (USEPA, 1980).

TOXICOLOGICAL PROPERTIES

TCDD

Exposure to TCDD is possible through ingestion of food or water, inhalation, or dermal application. Because TCDD is strongly sorbed to soils and sediment, drinking water contamination is very unlikely, especially in groundwater supplies. Surface waters can be contaminated from contaminated industrial effluents or washouts from contaminated disposal sites, however, even in these cases, TCDD was found to be strongly sorbed to sediments and biota (USEPA, 1984a).

Possible TCDD contaminated food items can include plant crops sprayed with weed killing herbicides such as Silvex and 2,4,5-T, livestock raised on TCDD contaminated forage and other organisms that have bioconcentrated the chemical through the food chain. Studies on animals indicate that TCDD is readily absorbed through the gastrointestinal tract, but there is little evidence that TCDD is taken up or absorbed in food crops (USEPA, 1984a). Research indicates that when TCDD is a contaminant in an organic herbicide, rapid photochemical degradation occurs during the application process (Crosby, 1983).

TCDD can bioaccumulate in aquatic and terrestrial organisms. Bioconcentration studies using a variety of fish species have resulted in measured bioconcentration factors (BCF). BCF relates the concentration of a chemical in aquatic species to the concentration of the chemical in water. Bottom feeders, carnivores, and species with high fat content had higher BCF values. These species include catfish, carp, trout, and salmon. Calculated BCF values using the octanol:water partition coefficient resulted in a range of 7,000 to 900,000 (USEPA, 1984a). Currently, the USEPA's best estimate for the BCF of TCDD in aquatic organisms is 5,000. TCDD also apparently accumulates in fat tissue of cattle grazing on contaminated pasture (Kimbrough 1983). Levels of TCDD in cattle were found to range from 4 to 70 parts per trillion (ppt). TCDD can also be found in mammalian milk.

Inhalation of TCDD can occur during agricultural spraying, industrial incineration, industrial accidents, or as dust borne particles. No analytical information is available concerning effects of inhaling TCDD (USEPA, 1984a).

Dermal exposure to TCDD is most likely to occur during the manufacture and application of contaminated chlorinated herbicides. Many people were exposed to dioxins from the application of Agent Orange during the Vietnam war. Researchers have attempted to quantify human health effects from possible exposure to TCDD by studying health histories of manufacturing and agricultural workers and Vietnam veterans most likely exposed to this chemical. The most common and obvious effect reported was chloracne, or skin lesion. Other possible effects are included in the following discussions of acute and chronic toxicities, mutagenicity, teratogenicity, and carcinogenicity.

After absorption of 2,3,7,8-TCDD into most animal species studies, this chemical is most often found in the liver and adipose tissues. Biological metabolites of TCDD are not considered to be toxic compared to TCDD itself. Elimination routes for TCDD in mammals include lactation, direct intestinal elimination, and sebum.

The toxic effects of TCDD have been extensively studied in animals. These studies indicate that the compound is toxic from both an acute and chronic standpoint. Animal studies indicate that TCDD is potent hepatic enzyme inducer in most species. Beside the liver, other target organs include the thymus, testicles, spleen, gall bladder, skin, and urinary tract (Olie, et al, 1977). Of special interest from an acute toxicity standpoint is the wide species variation. The oral LD50 values for TCDD range from 0.6 $\mu\text{g/kg}$ body weight for the guinea pig to 5,051 $\mu\text{g/kg}$ body weight for the hamster. Toxicities vary with age, sex, and strain of test animal with young animals more susceptible than older animals. Six differences varied with animal tested showing no apparent trend (USEPA, 1984a). The most common toxic responses included loss of body weight, thymic atrophy, and increase in liver weight. Often toxic responses were delayed.

Chronic effect studies indicate that TCDD exposure may produce chloracne, liver damage, kidney damage, immunological alterations, hematological alterations, gastrointestinal tract changes, and neuropsychiatric effects in test animals. Subchronic effects reported in rats include lethargy, decreased body weights, liver pathology, biochemical evidence of liver damage, thymic atrophy, decreased lymphatic tissues, disturbance of porphyrin metabolism, slight alterations in the hematopoietic system and mild adverse effects on both male and female reproductive systems. Long recovery times were seen in subchronic studies (USEPA, 1984a). Subchronic effects were observed in humans after the accidental exposure to TCDD in Seveso, Italy. Over 200 cases of chloracne were reported with the most severe fully recovered after 18 to 24 months (with one exception). Other symptoms included signs of liver damage,

raised serum transaminase and glutamyl transferase, and some neurological effects (USEPA, 1984a). Before chloracne appears, overexposure may be indicated by burning sensations in the eyes, nose, and throat, headache, dizziness, and nausea. Other symptoms include joint pain, fatigue, insomnia, irritability and nervousness. Emotional disorders, difficulties with muscular and mental coordination, blurred vision, and loss of taste or smell may occur. Deaths related to TCDD induced liver damage have occurred (Olie, et al, 1977).

Animal studies also indicate that TCDD is a developmental toxin. It acts as a teratogen in mice and hamsters, but not primates. TCDD produces fetotoxicity at doses higher than doses causing teratogenic effects. The most common teratotoxic responses in rats and mice are increased cleft palate and kidney abnormalities. TCDD appears to be acting synergistically with 2,4,5-T with respect to the increase in cleft palate occurrences. Many attempts have been made to link fetotoxicity and teratotoxicity to TCDD or herbicide exposure in pregnant women. Studies thus far have not been statistically conclusive regarding any toxic effects in human reproduction (USEPA, 1984a). Animal testing and human health histories have shown no increase in fetotoxicity or teratotoxicity resulting from male exposure to TCDD.

Genotoxicity testing of TCDD has been extensive, however, the results of these studies have indicated little potential for mutagenic effects (USEPA, 1984a). While some studies indicate that TCDD may be a bacterial mutagen and cause cytogenetic damage (Olie, et al, 1977), overall, the data indicate little potential for the interaction of TCDD with nucleic acids or the ability of TCDD to produce chromosomal aberrations (USEPA, 1984a).

Several animal studies using TCDD have indicated an oncogenic effect. This effect has been seen in both mice and rats. Also, studies have been performed that have led to the conclusion that TCDD is a tumor promoter. Recently, the National Toxicology Program (NTP) bioassay program concluded that TCDD is a carcinogen when studied in rats and mice (NTP, 1983). Kolbye (1983) states that dioxins are secondary carcinogens only able to promote tumors in already initiated cells after continuous, consistent, and selective pressures on that cell.

Researchers have tried to link human cancers of various types to TCDD exposure. The most significant association appears to be between TCDD and soft tissue sarcoma. The most recent studies prepared by the U.S. Air Force involving 1,247 people exposed to TCDD in Vietnam showed no significant cancer increase. Small sample size or inadequate

study plans have discounted many human health surveys attempting to link cancer to TCDD or herbicide exposure.

TCDD is suspected of being a human carcinogen because of multiple positive animal carcinogenicity studies. The USEPA's position is that there is no recognized safe level for a human carcinogen and the recommended concentration in water for maximum protection of human life is zero. Because attaining a zero concentration may not be feasible at this time, the concentrations corresponding to incremental increased lifetime cancer risk levels have been estimated (USEPA, 1984a) and are shown below:

Exposure	Concentration of TCDD in water resulting in 10^{-6} incremental increase for lifetime cancer risk ($\mu\text{g/L}$)
Consumption of 2 L drinking water per day and 6.5 g of fish and shellfish.	1.3×10^{-8}
Consumption of 2 L drinking water per day only	2.2×10^{-7}

As can be seen in this example, the water contamination level must be 17 times as high for the same risk level as when no contaminated fish are consumed.

The Center for Disease Control has recommended that residential soils contain TCDD at average concentrations no greater than 1 ppb. Higher levels in commercial areas may represent an acceptable risk level to non-occupationally exposed persons (Kimbrough, 1983). The U.S. FDA has recommended against consumption of food containing TCDD at levels greater than 50 ppt or more than twice per month at greater than 25 ppt. No tolerance levels have been established for TCDD on food crops (Miller, 1983).

In summary, TCDD is considered to be an unusually toxic compound with demonstrated acute, subchronic, and chronic effects in man and animal. Reported adverse effects include chloracne and damage or changes to the liver, nervous system, immune system, and reproductive system. Special groups at risk are those employed in the manufacture of chemicals which may contain 2,3,7,8-TCDD as a contaminant, women of child-bearing age, and especially the fetus.

HEXACHLOROCYCLOHEXANE

BHC is the common name for a mixture of configurational isomers of 1,2,3,4,5,6-hexachlorocyclohexane. BHC is really a misnomer for this aliphatic compound and should not be

confused with similar aromatic compounds. In 1942, BHC was introduced as a broad spectrum insecticide that took upon special importance since it could kill insects that developed a resistance to DDT. Subsequently, it was found that the insecticidal activity was due primarily to the gamma isomer, now commonly called Lindane. Formulations changed to exclusive use of this isomer and by 1978, the use of BHC was dropped in the U.S. and replaced by Lindane (Sittig, 1980).

Although the terms BHC and Lindane are often used interchangeably, information presented here will emphasize the gamma isomer and it should be recognized that the other isomers show similar but not identical qualities.

Human overexposure to BHC has occurred by accidental ingestion, occupational exposure, and its use as a medical treatment. In each case, the primary illness reflected neurological effects such as seizures, muscle spasms, overall weakness, and grand mal convulsions. Other symptoms of overexposure included liver and kidney damage, pancreatitis, muscle necrosis, vomiting, and headache. In the most severe cases, convulsions were followed by coma, then death. Chronic exposure has been linked to aplastic anemia and other blood disorders. It is important to point out that γ -BHC induced illness may result from exposure via the inhalation, dermal or oral route. It is also important to remember that human exposure to γ -BHC has been approved for medicinal purposes in vaporizers, and in creams, lotions, and shampoos (Gosselin, et al., 1984). Improper use of these materials resulting in exaggerated exposures has contributed to the adverse effects previously discussed.

The mechanism by which BHC are insecticidal is through toxicity to the nervous system (Sittig, 1980). These effects are seen in acute animal studies. The oral LD₅₀ for γ -BHC is 90 mg/kg (rats), 50 mg/kg (rabbits), and 127 mg/kg (guinea pigs). Dermal LD₅₀ values vary greatly depending upon the vehicle used in testing. As a 1% solution in vanishing cream, the dermal LD₅₀ (rabbits) was reported as 50 mg/kg while γ -BHC in the dry powder form had a rabbit dermal LD₅₀ of \approx 4000 mg/kg (Clayton and Clayton, 1981). The various isomers of BHC vary greatly in toxicity. The alpha, beta, and delta isomers have a low degree of acute toxicity but are retained longer than the gamma isomer. γ -BHC has a much greater acute toxicity but has a lower degree of cumulative toxicity since it is retained in the body for a shorter period of time (Sittig, 1980).

Chronic animal studies of γ -BHC indicate that this compound is stored in the fat of the body but does not biomagnify. Within 3 weeks of cessation of exposure, it disappears from the fat (Sittig, 1980). Chronic animal studies indicate

that several BHC isomers including gamma are carcinogenic when administered in the diet of mice. Liver tumors were produced. Studies in rats and dogs were considered to be inadequate for making a carcinogenicity determination as were human data (IARC, 1979). Based on the mouse liver tumor information, the National Toxicology Program has included BHC on its list of carcinogens.

Some reproductive data are available. Exposure levels of 0.5 mg/kg/day resulted in disrupted estrus cycles, inhibited fertility, delayed embryonic development, and reduced viability of the fetuses in rats. This effect was not seen at lower exposure levels. Beagles given 75 mg/kg and 15 mg/kg during gestation had an increased number of stillborn pups. In another study, no teratogenic event was associated with exposures up to 20 mg/kg in rats. Except for a suggestion of chromosomal damage, no substantial studies resulted in a concern of mutagenicity resulting from BHC exposure.

The Permissible Exposure Level in the workplace is 0.5 mg/m³. This is a 5 day, 8 hour exposure value. The World Health Organization has set the acceptable daily intake at 1 µg/kg/day. The U.S. EPA states that it believes that exposure levels to carcinogens ought to be zero, however, they have considered setting interim risk levels for increased cancer risk.

For γ-BHC, the values for increased cancer risk of 10⁻⁵, 10⁻⁶, and 10⁻⁷ are 186 ng/l, 18.6 ng/l, and 1.86 ng/l respectively. These values assume ingestion of 2 liters of water per day and consumption of 6.5 g of fish daily (USEPA, 1980).

CHLORINATED BENZENES

Chlorinated benzenes consist of any benzene compound which contains one or more chlorine atoms. They range from the monochlorinated material to the hexachlorinated one. The dichloro, trichloro, and tetrachloro species can exist in various isomeric forms. There are several generalizations which can be made about these compounds which help understand their environmental qualities. In general, they are used as chemical intermediates for the synthesis of other compounds and can be used as pesticides. They may bioaccumulate in the environment with their potential for bioaccumulation increasing with chlorine content. As their chlorine content increases, their potential for biomagnification also increase, while their potential for biodegradation decreases (Sittig, 1980). Volatility and adsorbability to organic sediments also increase with the number of chlorine atoms (USEPA, 1981). Acute toxicity generally decreases as the number of chlorine atoms

increase. Most chlorinated benzenes are irritating to the skin, eyes, lungs, and mucous membranes (Sittig, 1980).

Animal studies and human experience indicate that all isomers attack the liver, kidney, and nervous system as target organs for toxic effects. Often chlorobenzenes affect the reproductive systems (Clayton and Clayton, 1981). Several representatives of the chlorinated benzene family will be discussed in the following pages.

Dichlorobenzenes have similar toxicities. Kidney, liver, lung, and the blood forming organs appear to be the primary target organs for toxicity based on human exposure information. They also may irritate skin, eyes and mucous membranes (Clayton and Clayton, 1981). An NTP bioassay for carcinogenicity on the ortho isomer concluded that there was no evidence of carcinogenicity. The available mutagenicity data also indicate that this effect is of little concern. The usual route of exposure is inhalation or dermal contact, however, occasionally ingestion of the para isomer has been reported (Clayton and Clayton, 1981). Trichlorobenzenes are produced in relatively small amounts. As with dichlorobenzenes, these isomers have similar toxicities. Human experience indicates that the target organs include lung, blood forming organs, and skin. Based on animal study results, it may be concluded that the kidney, liver, adrenals, and nervous system are potential target organs. These compounds also may cause dermal, eye and mucous membrane irritation. One chronic study for carcinogenicity was negative; however, these results are not conclusive enough to give an indication one way or the other regarding potential carcinogenicity. Mutagenicity and teratogenicity testing results have been negative.

Although acute toxicity in animals has not been shown to be a major concern, (rat oral LD₅₀ 3500 - 10,000 mg/kg), human overexposure to hexachlorobenzene has resulted in a condition called porphyria cutanea tarda, an illness in which porphyrin metabolism is disturbed. This condition does not appear to be an occupational problem even among workers producing hexachlorobenzene (IARC, 1979). Major outbreaks of this illness have been reported when humans ingest seeds treated with hexachlorobenzene. One outbreak involved 5,000 people. The illness was characterized by skin lesions, usually appearing in areas exposed to sunlight. Often the lesions became ulcerated and crusted. Other clinical symptoms included excessive hair growth and hyperpigmentation, corneal opacity, and liver damage. Even 20 years later, a few of the exposed individuals suffer from the hexachlorobenzene exposure. Young children appeared to be most sensitive to the effects of hexachlorobenzene and there were fatalities among this group (Clayton and Clayton, 1981).

Animal toxicity studies appear to support human observations with regard to hexachlorobenzene induced toxicity. Like humans, long term animal exposure to hexachlorobenzene resulted in porphyrin metabolism changes, liver toxicity, and parent to offspring transfer of hexachlorobenzene via mothers' milk. Animal studies also indicated there were effects on the kidney, nervous system, and reproductive process (IARC, 1979). Data are insufficient to assess the mutagenic potential of hexachlorobenzene.

Several chronic animal studies indicate that hexachlorobenzene is an animal carcinogen. Chronic oral exposure to hexachlorobenzene of rats, hamsters and mice have resulted in increased tumor incidence. Sites where incidents are elevated include liver, thyroid, and blood vessels (NTP, 1983).

Chlorobenzenes are listed by several organizations concerned with human health effects of these compounds. Recommendations for occupational air levels of exposure to several chlorobenzenes are as follows: chlorobenzene - 75 ppm; o-dichlorobenzene - 50 ppm; p-dichlorobenzene - 75 ppm; and 1,2,4 trichlorobenzene - 5 ppm (ACGIH, 1984). These values are for 8 hours/day, 5 days/week exposure to airborne concentrations of these materials. The U.S. EPA has set the following criteria for chlorobenzenes in drinking water: monochlorobenzene 20 $\mu\text{g/l}$; dichlorobenzenes 230 $\mu\text{g/ml}$; trichlorobenzene 13 $\mu\text{g/l}$; tetrachlorobenzene 17 $\mu\text{g/l}$; and pentachlorobenzene 0.5 $\mu\text{g/l}$ (USEPA, 1980). The mono and trichlorobenzene values are based upon odor and taste, the others on toxicity. The U.S. EPA states that they feel that exposure levels for carcinogens ought to be zero; however, EPA has set levels of incremental risk for carcinogen exposure. For hexachlorobenzene, incremental risks of cancer of 10^{-5} , 10^{-6} , and 10^{-7} are 7.2 ng/l , 0.72 ng/l and 0.072 ng/l respectively (USEPA, 1981). These values assume ingestion of 2 liters of water per day and consumption of 6.5 g of fish daily. The World Health Organization has set a conditional daily intake for this compound at 0.5 $\mu\text{g/kg/day}$ in foods (Clayton and Clayton, 1981).

Since chlorobenzene compounds are irritating, toxic to the liver, and in general may adsorb to organic matter, care should be taken to avoid dermal contact with them. Many of these compounds are quite volatile and potential exposure may occur if sediments onto which they are entrapped are disturbed. Consideration ought to be given to respiratory protection if this is a possibility.

TOLUENE

Toluene is a flammable and volatile aromatic hydrocarbon used as a solvent in the chemical, rubber and pharmaceutical industries. The general population may be exposed to toluene in perfumes, paints, cigarette smoke and inks (Clayton and Clayton, 1981). Toluene is found in finished municipal water at levels up to 19 mg/l (Sittig, 1981).

This compound may be toxic to fresh water species at concentrations as low as 17.5 ppm. Bioaccumulation is not an important consideration. Toluene is similar to benzene in toxicity except that it shows none of the hematopoietic effects. Previously toxicity attributed to toluene was actually caused by high benzene levels of contamination. Toluene is readily absorbed via the ingestion and inhalation route. It may also enter the body through the skin. Although toluene may be exhaled, its major route of excretion is through rapid oxidation to benzoic acid, conjugation with glycine and excretion in the urine as hippuric acid. Excretion as hippuric acid is proportional to exposure within reasonable limits.

Acute toluene overexposure may cause irritation of the eyes, skin and respiratory tract. It also may cause dizziness, fatigue, confusion, headache, impaired coordination, nausea, muscular weakness and numbness. Very high exposure may result in visual disturbances, unconsciousness, reversible kidney and liver damage or death in humans. Chronic human toluene exposure reportedly can cause cardiac, liver, kidney and brain damage. Prolonged contact with toluene defats the skin resulting in cracking and drying.

Acute animal toxicity tests indicate that toluene is not acutely toxic - oral LD₅₀ (rat) 1 g/kg, dermal LD₅₀ (rabbit) 10 g/kg and inhalation LC₅₀ (rat) 8000 ppm. It is a dermal and eye irritant. Subchronic animal studies by both the inhalation (up to 500 ppm) and oral routes (up to 390 mg/hq/day) produced no significant pathology (Clayton and Clayton, 1981).

Chronic rat inhalation studies at exposure levels up to 300 ppm for 24 months and general skin painting studies in mice did not indicate any carcinogenic potential for this compound. Gene mutation studies in bacteria gave no indication that toluene is a potential mutagen. Chromosomal anomalies have been seen in some studies of toluene exposure. However, other studies do not substantiate the observations. Animal studies indicate that at high exposure levels, embryotoxic effects as well as maternal toxicity could result from toluene exposure. One report of potential teratogenicity in mice was not substantiated by three other studies (USEPA, 1983).

The OSHA occupational exposure limit value is 200 ppm in the air. This value is for an 8 hour, 5 day/week exposure to airborne concentrations of this compound. The National Institute of Occupational Safety and Health recommends that the 8 hour work place limit be set at 100 ppm and a 10 minute ceiling value of 200 ppm be established. The current 200 ppm regulation value is established based upon the lack of irritating and narcotic effects seen in workers exposed to levels below this figure.

The EPA has established an ambient water concentration of 14.3 mg/l as a safe level for protection of human health from the toxicity of toluene. This value is calculated on the assumption that exposure will occur via both drinking water and ingestion of aquatic organisms living in the same contaminated water (USEPA, 1980).

ARSENIC

Arsenic is an inorganic material found in the earth's crust at an average concentration of 5 ppm (Bowen, 1979). The element is rarely found in pure form, rather it occurs as an oxide, metal arsenical, or in some type of compound with other elements. Many of the forms in the environment are oxides which are of lower solubility. As with many other inorganic materials, solubility and mobility is increased as the pH decreases or increases. The oxygen content of the water also influences the oxidation state and solubility of arsenic. For the most part, arsenic and its compounds strongly adsorb to soils and sediments. Arsenic and its compounds are extremely persistent and do not degrade. Arsenic has had widespread industrial use for hardening metals, insecticides, veterinary products, pharmaceuticals, weed killers, and other agricultural products (Clayton and Clayton, 1981). Arsenicals are acutely toxic to most aquatic organisms with an acute LC₅₀ in the range of 0.8 to 1,400 ppm and chronic limits in the range of 0.9 to 3.0 ppm. Unlike cadmium, arsenic toxicity is not changed by changes in water hardness. Arsenic does bioconcentrate in aquatic organisms, so body levels can be found that are 350 times greater than the surrounding environment (USEPA, 1980). Contaminated fish and shellfish could then act as an arsenic source for humans.

The acute oral toxicity for arsenic trifluoride for rats is in the range of 15 to 24 mg/kg for aqueous solutions compared to 145 to 214 mg/kg of the dry powder (Clayton and Clayton, 1981). The difference probably reflects differences in absorption from the gut. Toxic doses resulted in hemorrhage in the stomach and intestines as well as fatty degeneration and cell necrosis in the liver. Mutagenic and cytogenetic changes have been noted in a variety of animal cells as a result of arsenic exposure, but the consistency

of the response can often be quite variable. Likewise, studies indicate that arsenic is carcinogen and mutagenic in rodents (Department of Labor, 1983).

Acute exposures in humans that result in deaths are rare since these events usually arise from poisonings. The smallest recorded fatal dose was 130 mg, and death after a fatal dose usually takes 24 to 48 hours. Acute symptoms include abdominal pain, inflammation, vomiting, and hemorrhaging. Chronic exposure in humans has been documented through a variety of occupational exposures where dermal, inhalation, and ingestion are all important routes of exposure (Clayton and Clayton, 1981).

Dermal exposure leads to a variety of skin problems. Reddening and formation of various types of skin eruptions are quite common. In certain cases, there may be an increase in skin pigmentation, and increased sweating may occur. In addition to changes in the skin, some degree of percutaneous absorption can occur which could lead to other chronic health problems.

Chronic exposure to inhalation and/or ingestion of arsenic has led to a variety of physical ailments. Inhalation has led to problems with the respiratory system which includes emphysema, rhinitis, perforation of the nasal septum, and other symptoms (Clayton and Clayton, 1981). Drinking arsenic contaminated water resulted in changes in immune function, cardiovascular disorders, peripheral vascular disorders, and nerve degeneration in the peripheral nervous system (IARC, 1980). Human arsenic exposure has also been associated with reproductive effects which includes increased abortions as well as some malformations of offspring, particularly Down's syndrome. Finally, there are a variety of positive reports linking arsenic exposure to human cancer. In particular, ingestion of arsenic appears to lead to increased dermal cancer.

CADMIUM

Cadmium is normally used in electroplating to reduce corrosion of the substrate metal; in paint and pigment manufacturing; and as a stabilizer in plastics manufacturing, electrical equipment, and fungicides.

In the environment as a whole, cadmium is stable and persistent. At a pH of less than 6.5, cadmium exists in soluble forms with a high mobility in an aqueous phase. As the pH increases, the solubility and mobility will both decrease substantially. Cadmium that is associated with a sediment/soil will generally remain bound to the sediment/soil, but some leaching will occur at low pH values. Sediments

and soils will generally act as a sink for environmental cadmium (Sittig, 1980).

The acute animal toxicity of cadmium salts, such as the chloride, are moderately high with LD₅₀'s in the range of 30 to 175 mg/kg. Cadmium concentrates in the kidney, liver, thyroid and pancreas, but the kidney is the most important target. When zinc is present at a ratio of 4:1 with cadmium, it blocks the harmful effects of cadmium (Clayton and Clayton, 1981). Accumulation of cadmium occurs over time, so that low-level exposures over a long period of time can also lead to toxicity. Toxicity to aquatic animals is in the range of 0.2 to 80 ppb acutely or 0.2 to 50 ppb chronically. Aquatic toxicity is very dependent upon water hardness, and decreases as hardness increase (USEPA, 1980). Fish and other aquatic organisms can bioconcentrate cadmium in their tissue to a level which is 2,000 times greater than their environment; consequently, ingestion of aquatic organisms by humans may pose a significant health threat.

Some work has been done to examine the carcinogenicity of cadmium in animals (Clayton and Clayton, 1981). The studies have found localized cancer at the site of cadmium injection which appeared to be prevented by co-injection with zinc. Oral administration of cadmium has generally been unsuccessful in producing tumors in animals. Likewise, studies examining mutagenesis have also been unsuccessful. Terratogenic effects in test animals are equivocal since both positive and negative results have been obtained. Cadmium appears to cause testicular necrosis, so that male fertility may be reduced from chronic exposure (Clayton and Clayton, 1981).

Human exposure has also resulted in kidney and some liver problems as with experimental animals (Clayton and Clayton, 1981). The most important routes of human exposure are ingestion or inhalation of cadmium contaminated dusts, soils, or sediments. Because of the properties of cadmium, vapors are not generated or important for inhalation, and dermal absorption will not be significant either. As with animal studies, the presence of zinc can reduce the adverse health impacts of cadmium. Chronic exposures of cadmium need to be in excess of 250 µg/day to produce adverse health effects in humans. Adverse health effects could include changes in kidney, liver, pancreas, thyroid, and bone function (Sittig, 1980). Common problems arising from exposure are protein excretion in the urine and anemia. Human cancers that may have been caused by cadmium exposure were a result of inhalation of contaminated dusts in foundry operations, not ingestion of particulates, soluble salts, or contaminated food.

Cadmium in the environment is persistent and has wide spread consequences. The material accumulates in aquatic animals, mammals, and humans to produce adverse health effects. The important routes of exposure are ingestion of contaminated materials and inhalation of contaminated particles. For protection to humans, ingestion from all sources should be less than 250 µg/day.

THALLIUM

Thallium is a widely distributed element, comprising approximately 0.003 percent of the earth. It is isolated as a byproduct of production of other metals such as zinc, lead and cadmium. Although used elsewhere in the world as a rodenticide, thallium sulfate use for this purpose stopped in 1972 in the U.S. (Clayton and Clayton, 1981). Low levels of thallium are found in plants and some water. In plants, the levels range up to 10 µg/kg (wet weight). Thallium levels in drinking water seldom exceed 0.3 µg/L. Limited information indicates that significant thallium exposure may occur via smoking. Cigarette smokers excrete twice as much thallium as do non-smokers. It is estimated that daily adult intake of thallium is about 6 µg/day (Sittig, 1980).

Human exposure to thallium has resulted in toxic effects under occupational, clinical and accidental exposure conditions. Usual routes of intoxication are ingestion (accidental or clinical) and dermal or inhalation (occupational). No fatalities have ever been reported as a result of acute industrial exposure. Fatalities caused by thallium usually result from the ingestion of thallium rat poison. Thallium salts also have been used for treatment of syphilis, ringworm and tuberculosis. These uses have produced a clinical picture which gives perspective to its human toxicity. Human toxicity from overexposure includes neurological disorders, gastrointestinal disorders, hair loss, kidney damage, liver damage and death (Clayton and Clayton, 1981). Estimated human oral LD₅₀ values range from 200 to 1,000 mg/kg. Permanent damage after acute exposure to thallium is unusual except for possible hair loss resulting from very heavy exposure. There are no data which indicate that either children or the developing fetus is especially sensitive to toxicity resulting from thallium overexposure (Sittig, 1980).

Animal data for acute toxicity indicate that LD₅₀ values of thallium compounds range from about 10 to 65 mg/kg. These data indicate that this compound is considered very toxic. Subacute and chronic studies indicated that these compounds are relatively toxic at low dietary exposure levels. In rats, dietary levels as low as 15 ppm produce adverse effects. At this dietary level, hair loss is the effect noted. This effect is also seen in humans. At higher

dietary levels, weight loss was observed. Distribution and excretion of thallium was often found to be different in laboratory animals than man. Human autopsy material indicates that thallium deposition was localized in the scalp, kidney, heart, and spleen in that order. In rats, thallium localized in the kidney, followed by the gut, gonads, and pancreas. Similar differences were seen in excretion. In rabbits, about equal percentages of thallium were excreted in the feces as in the urine; in rats, about 50 percent more thallium was excreted in the feces as in the urine, but man, 25 times more thallium was excreted in the urine than in feces (Sittig, 1981).

The U.S. occupational exposure threshold limit value for thallium of 0.1 mg/m^3 is based upon analogy with other heavy metals. Although this value is for 8 hours, 5 days/week exposure to soluble thallium salts, this value is really applicable to all thallium compounds. The EPA water quality criteria value for thallium of $13.0 \text{ } \mu\text{g/L}$. This value assumes ingestion of 2 liters of water and 6.5 grams of fish or shellfish (USEPA, 1980).

WDR99/040

DRAFT

RECEIVED

MAR 11 1985

N.Y.S.D.E.C.