United States
Environmental Protection
Agency

Effluent Guidelines Division
WH-552
Washington DC 20460

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Water

EPA

Development Document for Effluent Limitations Guidelines and Standards for the Organic Chemicals and Plastics and Synthetic Fibers

Proposed

Point Source Category
Volume III (BAT)

DEVELOPMENT DOCUMENT

for

PROPOSED EFFLUENT LIMITATIONS GUIDELINES AND NEW SOURCE PERFORMANCE STANDARDS

FOR THE

ORGANIC CHEMICALS AND PLASTICS AND SYNTHETIC FIBERS INDUSTRY

VOLUME III (BAT)

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NOTICE MAR 31 1983

On February 28, 1983, EPA proposed effluent limitations guidelines and standards for the organic chemicals and plastics and synthetic fibers (OCPSF) point source category. The Federal Register notice of this proposal was printed on March 21, 1983 (48 FR 11828 to 11867).

Information received by the Agency after proposal indicates that the total OCPSF industry estimated annual discharges of toxic pollutants are too high. The Agency will be reevaluating these estimates when additional information becomes available prior to promulgation of a final regulation. In the interim, the Agency advises that there should be no reliance on the annual total toxic pollutant discharge estimates presented in the Federal Register notice, the February 1983 OCPSF Development Document, and February 10, 1983 OCPSF Regulatory Impact Analysis.

Environmental Protection Agency

VOLUME III (BAT)

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APPENDIX A

SAMPLE 308 QUESTIONNAIRES

308 BPT QUESTIONNAIRE

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF WATER AND
HAZARDOUS MATERIALS

As you may be aware, we are in the process of reconsidering and re-issuing regulations with respect to water pollutants discharged as a result of the manufacture of organic chemicals and plastics and synthetics. The earlier regulations were issued as 40 C.F.R. Parts 414 and 416, respectively.

The reconsideration of organic chemicals is a result of a joint stipulation filed in the Fourth Circuit Court of Appeals entered into on behalf of companies in the industry and the Environmental Protection Agency. The stipulation requires the Agency to obtain new and more reliable data and requires the industry to cooperate in the gathering and furnishing of data necessary to formulate regulations for the organic chemicals manufacturing point source category.

The reconsideration of plastics and synthetics results from the remand by the Fourth Circuit Court of Appeals of the EPA regulations promulgated on April 5, 1974. The Court ordered the EPA to restudy areas where the record was ruled to be inadequate.

To complete these reconsiderations, the Agency is collecting additional information on the production processes, raw waste loads, treatment methods and costs, and effluent quality associated with the manufacture of these materials. The Environmental Protection Agency is now soliciting your cooperation in obtaining the necessary information.

According to our records, your Corporation produces one or more of the products on the attached list(s) (Part I) which were covered by the referenced regulations. For the plastics and synthetics category, plants that solely purchase polymer, resin or fiber to manufacture finished plastic components should return the enclosed forms unanswered and state this reason. Plants that manufacture plastic components in addition to the polymer, resin or fiber should list the manufactured components as "other products" in the appropriate section and complete the forms.

The information requested shall be provided for each plant of your firm in the format of the attached portfolios. This will allow the Agency to correlate and make available to interested parties the results of the data gathered. If our records are incorrect and you do not feel that the requested information is related to your plant (i.e., you no longer manufacture the products listed or do not produce them at this site), please inform us as soon as possible. In order to expedite the process we have sent a copy of this letter to those individuals and plants of your firm as noted on the attached list.

The information requested in this letter and the enclosed data collection portfolio is sought pursuant to Section 308 of the Federal Water Pollution Control Act Amendments of 1972. That section authorizes this Agency, whenever required for developing any effluent limitation, or other limitation, prohibition, or effluent standard, pretreatment standard, or standard of performance under this Act, to require the owner or operator of any point source to establish and maintain such records, make such reports, install, use and maintain such monitoring equipment or methods (including where appropriate, biological monitoring methods), sample such effluents (in accordance with such methods, at such locations, at such intervals, and in such manner as the Administrator shall prescribe), and provide such other information as the Agency may reasonably require, and to have access to and copy any records, inspect any monitoring equipment and sample any effluents.

Information requested pursuant to Section 308 may not be withheld from EPA on the ground that it is considered to be confidential or proprietary. Section 308(b), however, does accord protection to trade secrets. Accordingly, please indicate clearly on your response any information which you consider to be confidential or to constitute a trade secret, so tnat the Agency may take appropriate protective measures. Any information not so identified in your response will not be accorded this protection by the Agency. Effluent data cannot be protected as trade secrets. Any data may be disclosed to officers, employees, or authorized representatives of the United States concerned with carrying out the Act or when relevant in any proceeding under the Act.

For your convenience, a data collection portfolio has been enclosed with this letter. This form is divided into several parts. Those parts that are applicable to your operations should be filled out and returned to the Agency as soon as possible but in no event later than sixty days after receipt of the letter.

The parts contained in the data collection portfolio are as follows:

Part I. General Information
Part II. Water Use, Reuse and Discharge
Part III. Treatment Technology

Please answer all items. Also, please provide a separate set of responses for each plant. The purpose of this request is to gather all available, pertinent information and is not designed to create an undue burden of sampling requirements on your plant personnel. If a question is not applicable to a particular facility, indicate by writing "Not Applicable". If an item is not known, indicate unknown and include an explanation of the reason for not knowing such information. If an item seems ambiguous, complete as best as possible and state your assumptions in clarifying the apparent ambiguity. Also, submit copies of the summary

data sheets compiled or used in completing the tables in this form.

The Agency will review the information submitted and may, at a later date, require site visits and additional sampling in order to complete the data base.

Thank you in advance for the cooperation of your company. The Environmental Protection Agency is committed to promulgating effluent regulations which are in accordance with the Federal Water Pollution Control Act and which are reasonable. The Agency has found that only with complete cooperation of all parties concerned can thoughtful and fair regulations be published. I am confident that we can anticipate your assistance in carrying out that goal.

Should you have any questions regarding this request, please do not hesitate to contact Lamar Miller with respect to organic chemicals at (202) 426-2582 or Michael Kosakowski at (202) 426-4617 with respect to plastics and synthetics.

Sincerely yours,

Robert B. Schaffer
Director
Effluent Guidelines Division (WH 552)

Enclosures

ORGANIC CHEMICALS OR PLASTICS AND SYNTHETICS (Identify Category)

PART I GENERAL INFORMATION

To be returned within 60 days of receipt to:

Robert B. Schaffer, Director
Effluent Guidelines Division
U.S. EPA (WH-552)
Washington, D. C. 20460

1. Name of Corporation

_____

2. Address of Corporation Headquarters

Street: _____

City: _____

State: _____ Zip Code _____

3. Name of Plant

_____

4. Address of Plant

Street: _____

City: _____

State: _____ Zip Code _____

5. Name(s) of corporation personnel to be contacted for information pertaining to this data collection portfolio.

Name	Title	(Area Code) Telephone

6. Plant NPDES Permit Number(s) _____

Date of expiration _____

If no permit, application number _____

Date of application _____

Corporation_____
Plant_____
City_____ State_____

7. Products produced at this plant site.

a. Indicate which of the products in list 1 (Plastics and Synthetics-page 3) or list 2 (Organic Chemicals-page 4) that you produce at this site and the production rate during the period January 1, 1975 to September 30, 1976. If there is more than one process type for a given product, identify and list each separately. The average daily production while operating should match with the waste water data tables in Part II.

Product	Process	Design Capacity lbs/day	Avg. Daily Production While Operating lbs/day	Year Process Installed

Attach additional pages, if necessary.

LIST 1

PLASTICS & SYNTHETICS

ABS/SAN
Acrylic Resins
Alkyds and Unsaturated Polyester Resins
Cellulose Acetate Fiber/Resin
Cellulose Derivatives
Cellulose Nitrate
Cellophane
Epoxy Resins
Ethylene-Vinyl Acetate Copolymers
Fluorocarbon Polymers
Melamine Resins
Nylon Resins/Fiber
Phenolic Resins
Polyamides
Polyester Resin/Fiber
Polyethylene
Polypropylene Resin/Fiber
Polystyrene
Polyurethane Resins
Polyvinyl Acetate
Polyvinyl Chloride
Rayon
Silicones
rea Resins

LIST 2

ORGANIC CHEMICALS

Acetaldehyde
Acetic Acid
Acetone
Acetylene
Acrylates (includes acrylic acid, methacrylic acid, and esters)
Acrylonitrile
Adiponitrile
Aniline
Benzene
Benzoic Acid
Bisphenol A
Caprolactam
Chloromethanes (Methyl Chloride, Dichloromethyl, Chloroform, and Carbon tetrachloride)
Citronellol
Coal Tar
Cumene
Cyclohexane
Dimethyl Terephthalate
Diphynylamine
Ethyl Acetate
Ethyl Benzene
Ethylene
Ethylene Dichloride
Ethylene Glycol
Ethylene Oxide
Formaldehyde
Hexamethylenediamine
Isobutylene
Isopropanol
Maleic Anhdride
Methanol
Meta-Xylene
Methyl Amines (including mono, di, and tri methyl amine)
Methyl Ethyl Ketone
Methyl Salicylate
Ortho-Nitroaniline
Ortho-Xylene
OxoChemicals
Para-Aminophenol
Para-Cresol
Para-Nitroaniline
Para-Xylene
Phenol
Phthalic Anhydride
Plasticizers (esters of phthalic acid)
Propylene
Sec-butyl-alcohol
Styrene
Tannic Acid
Terephthalic Acid
Tetraethyl Lead
Toluene
Vinyl Acetate
Vinyl Chloride

Corporation_____
Plant_____
City_____ State_____

D. Indicate which of the products in list 3 (Organic Chemicals - page 6) that you produce at this site and the production rate during the period January 1, 1975 to September 30, 1976. If there is more than one process type for a given product, identify and list separately. The average daily production while operating should match with the waste water data tables in Part II.

Product	Process	Design Capacity lbs/day	Avg. Daily Production While Operating lbs/day	Year Process Installed

Attach additional pages, if necessary.

LIST 3

ORGANIC CHEMICALS

Proplyene Oxide
Acetic Anhydride
Ethyl Alcohol, Synthetic
Adipic Acid
Cyclohexanone
Nitrobenzene
Tetrachloroethylene (Perchloroethylene)
Propylene Glycol (1,2-Propanediol)
Diethylene Glycol
Trichloroethylene
N-Butyl Alcohols (N-Propylcarbinol)
Dichlorodifluoromethane
Ethanolamines, Total
Trichlorofluoromethane
4,4-Isopropylidenediphenol (Bisphenol A)
2-Methoxyethanol (Ethylene Glycol Monomethyl Ether)
2-Aminoethanol (Monoethanolamine)
Cresols, Total
Epoxidized Esters, Total
2,2-Iminodiethanol (Diethanolamine)
Triethylene Glycol
Pentaerythritol
Hexamethylenetetramine, Tech.
Ortho-Dichlorobenzene
a-Chlorotoluene (Benzylchloride)
Fumaric Acid
Dipropylene Glycol
Glutamic Acid, Monosodium Salt
Choline Chloride (All Grades)
Para-Nitrophenol and Sodium Salt
Pentachlorophenol (PCP)
Propionic Acid
Xylenesulfonic Acid, Sodium Salt
Aspirin
Acetic Acid Salts, Total
Methyl Bromide
Dodecyl Mercaptans
Salicylic Acid
Benzoic Acid Salts: Sodium Benzoate, tech. and U.S.P.
5-Nitro-Ortho-Toluenesulfonic Acid (SO3H-1)
Benzyl Alcohol
Benzoyl Peroxide
Castor Oil, Ethoxylated
2-Ethylhexanoic Acid (a-Ethylcaproic Acid)
2-Dimethylaminoethanol

Corporation_____
Plant_____
City_____ State_____

c. List below all other products (not appearing on lists 1, 2 or 3) manufactured at this same site that account for at least one percent of the plant's total production. Minor products may be grouped in this listing if the products are similar in nature and made by a similar process. The products should be listed individually with a total production indicated for the group in all instances where grouping is used to report.

Product	Process	Design Capacity lbs/day	Avg. Daily Production While Operating lbs/day

Corporation_____
Plant_____
City_____ State_____

d. List below products not in items 7a, b and c if they account for an inordinate pollution load either in terms of pounds discharged per 1,000 pounds of production (RWL) or difficult treatment problems.

8. For each product indicated in response to Questions 7a and 7b of Part I, attach a process flow diagram which identifies the unit operations involved in each product manufacturing process and all sources and quantities of waste waters from the process operations. Show recycle loops for both process water and non-contact cooling water and specify the blowdown control systems. Indicate raw materials used and contact and non-contact water entering each operation. Identify pollution control devices associated with the process that have wastewater streams. Use consistent units throughout; for example, gallons per hour or pounds per hour. Supplement the diagram with a narrative description for clarity or completeness where necessary.

   The respondent may use process flow diagrams from EPA Development Documents if representative of the process. The process diagrams should be modified to include all requested information.

   On each process flow diagram, clearly state whether the process operational mode is batch, continuous or other. If the answer is "other" the operational mode should be specified. If the process is batch or semi-continuous, describe the length of cycle and frequency.

9. Describe major process modifications made (to each process described in response to Question 8) since January 1, 1972 that significantly affect either the volume of flow, or the amount of waste water pollutants per unit of production originating from that process. Explain the purpose behind each of these modifications. Give your best estimate as to the technological age of each process installation as it now exists.

Corporation_____
Plant_____
City_____ State_____

Give an analysis of the effect of making the modification, i.e., describe the load and flow prior to the modification and after the modification. Do you have future modifications for in-plant control of waste water pollutants scheduled, if so, on which processes? Specifically highlight any process changes that would not be made except for pollution control. Include all such changes in the process flow diagram of Item 8 using a separate block wherever feasible.

Corporation_____
Plant_____
City_____ State_____

RGANIC CHEMICALS OR PLASTICS AND SYNTHETICS (Identify Category)

ART II WATER USE, RE-USE, AND DISCHARGE

be returned within 60 days of receipt to:

Robert Schaffer, Director
Effluent Guidelines Division
U.S. EPA (WH-552)
Washington, D. C. 20460

. Water Use, Total Plant Needs During the Period January 1, 1975 to September 30, 1976

List below for your plant the sources and quantities of water used and describe the disposition of waste waters. If a time period of less than January 1, 1975 to September 30, 1976 is used, state the reason that the values used are representative of that period.

. Water Source:

			Time Period of Calculation
Municipal	_____ mgd	(average value)	_____
Surface	_____ mgd		_____
Ground	_____ mgd		_____
Other (specify)	____ mgd		_____

B. Uses:

Non-contact cooling	_____mgd	_____
Direct process contact (as diluent, solvent, carrier, reactant, by-product, cooling, etc.)	_____mgd	_____
Indirect process contact (pumps, seals, etc.)	_____mgd	_____
Non-contact ancillary uses (boilers, utilities, etc.)	_____mgd	_____
Maintenance, equipment cleaning and work area washdown	_____mgd	_____
Air pollution control	_____mgd	_____
Sanitary and potable	_____mgd	_____
Other (specify)	_____mgd	_____

Corporation_____
Plant_____
City_____ State_____

C. Source of waste water flows:

Non-contact cooling	_____mgd	_____
Direct process contact	_____mgd	_____
Indirect process contact	_____mgd	_____
Non-contact ancillary uses	_____mgd	_____
Maintenance, equipment cleaning and work area washdown	_____mgd	_____
Air pollution control	_____mgd	_____
Sanitary/Potable water	_____mgd	_____
Storm water (collected in treatment system)	_____mgd	_____
Other (specify)	_____mgd	_____

D. Process or Process Contaminated Waste Water Discharged To:
(Do not include cooling water, boiler blowdown, etc.)

Surface water or storm sewer		
Treated	_____mgd	_____
Untreated	_____mgd	_____
Municipal Sewage Treatment Plant	_____mgd	_____
Deep well	_____mgd	_____
Other (Specify and describe briefly)	_____mgd	_____

2. Quality of Water Discharged:

For the period January 1, 1975 to September 30, 1976, summarize your influent, effluent and raw waste loads in Tables A, B, C, D and E. If data for individual waste streams are not available, information for combined waste streams should be furnished which represents the greatest degree of detail available. The tables are located at the end of this section.

Instructions for Completing Tables A, B, C, D and E:

For Tables A, B, C, D and E, use the following definitions and notes. The period covered should correspond with that used for Part I question 7 to calculate average daily production.

Flow - Do not include rainfall runoff, unless it is collected in the treatment system. If collected, estimate the percent of total flow which is attributed to this source.

Average day - Should represent the average of the data period covered.

Significant parameters - Those potential pollutants not specifically listed, but which are introduced into the waste streams as a result of materials used, product produced, process used and for which you have test data.

Corporation_____
Plant_____
City_____ State_____

Identify all data which results from abnormal operating or other conditions.

If use of a different time period (a portion of the time period January 1, 1975 to September 15, 1976) results in more adequate representation of the pollution loads, you may do so if the time period is not less than six months. You should specify the time period and explain why that period is more representative.

Table A - Complete Table A for the combined influent to each treatment facility.

Table B - Complete Table B for each untreated waste discharge point (to surface waters, deep wells, land application, etc.)

Table C - Complete Table C for the treated effluent from each treatment facility. Not applicable to plants that have not yet installed waste treatment facilities. This section is not restricted by type of treatment.

Table D - Complete Table D for the process wastewaters from each of the product/process lines identified in Part I, items 7a and 7b. Do not include non-contact cooling waters but do include all contact cooling waters. If measured values are not known or not available, supply the best estimate available and specify the basis for the estimate. The production basis should be the same as the average daily production while operating that was given in Part I.

Table E - Complete Table E for the plant intake water.

3. Attach the water analysis data summary sheets showing the daily water analyses that were used to compute Tables A through E, e.g. monthly summary tables. Also include any data for the period January 1, 1975 to September 30, 1976 that was omitted in Tables A through E as not being representative.

4. The method of sample collection for the data supplied in response to Question 2, Tables A, B, C, D and E, should be specified (e.g., daily grab sample, 8 hour flow composited, 24 hour continuous, etc.).

5. Indicate all parameters listed in Part II, tables A through E, which were not measured by EPA approved methods.

_____
_____
_____
_____

TABLE A

WASTE LOADS TO TREATMENT FACILITIES

Corporation _____

Plant _____

City _____ State _____

Treatment Facility Name _____

Treatment Facility Description _____

_____

Wastewater Source(s) _____

Time Period Represented _____

Parameter	Daily Minimum	Daily Long Term Average	Daily Maximum	Calendar Monthly Averages Minimum	Calendar Monthly Averages Maximum	Remarks
Flow (MGD)						
pH (pH units)						
Temperature (°C) - Wastewater						
Temperature (°C) - Ambient Air						
BOD_5 (lbs/day)						
COD (lbs/day)						
TOC (lbs/day)						
TSS (lbs/day)						
TDS (lbs/day)						
NH_3 as N (lbs/day)						
TKN as N (lbs/day)						
Phenol (lbs/day)						
Significant Metals (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
Others (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						

TABLE B

UNTREATED PROCESS WATER LOAD DISCHARGED

Corporation _____

Plant _____

City _____ State _____

Discharge Point _____

NPDES Discharge No. _____

Wastewater Source(s) _____

Time Period Represented _____

Parameter	Daily Minimum	Daily Long Term Average	Daily Maximum	Calendar Monthly Averages Minimum	Calendar Monthly Averages Maximum	Remarks
Flow (MGD)						
pH (pH units)						
Temperature (°C) - Wastewater						
Temperature (°C) - Ambient Air						
BOD_5 (lbs/day)						
COD (lbs/day)						
TOC (lbs/day)						
TSS (lbs/day)						
TDS (lbs/day)						
NH_3 as N (lbs/day)						
TKN as N (lbs/day)						
Phenol (lbs/day)						
Significant Metals (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
Others (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						

TABLE C

TREATED PROCESS WASTE LOADS DISCHARGED

Corporation _____

Plant _____

City _____ State _____

Treatment Facility _____

Treatment Facility Description _____

_____

Discharge Point _____

NPDES Discharge No. _____

Do you post-chlorinate this effluent? __ Yes __ No If yes, do you chlorinate __ (A) Full-Time __ (B) Part-Time

Time Period Represented _____

Parameter	Daily Minimum	Daily Long Term Average	Daily Maximum	Calendar Monthly Averages Minimum	Calendar Monthly Averages Maximum	Remarks
Flow (MGD)						
pH (pH units)						
Temperature (°C) - Wastewater						
Temperature (°C) - Ambient Air						
BOD_5 (lbs/day)						
COD (lbs/day)						
TOC (lbs/day)						
TSS (lbs/day)						
NH_3 as N (lbs/day)						
TKN as N (lbs/day)						
Phenol (lbs/day)						
Significant Metals (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
Others (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						

TABLE D

PRODUCT/PROCESS LINES RAW WASTE LOADS

Corporation _____

Plant _____

City _____ State _____

Product _____

Process _____

Time Period Represented _____

Parameter	Daily Minimum	Daily Long Term Average	Daily Maximum	Calendar Monthly Averages Minimum	Calendar Monthly Averages Maximum	Remarks
Flow (gal/1,000 lbs)*						
pH (pH units)						
Temperature (°C) - Wastewater						
Temperature (°C) - Ambient Air						
BOD_5 (lbs/1,000 lbs)**						
COD (lbs/1,000 lbs)						
TOC (lbs/1,000 lbs)						
TSS (lbs/1,000 lbs)						
TDS (lbs/1,000 lbs)						
NH_3 as N (lbs/1,000 lbs)						
TKN as N (lbs/1,000 lbs)						
Phenol (lbs/1,000 lbs)						
Significant Metals (Identify)						
_____ (lbs/1,000 lbs)						
_____ (lbs/1,000 lbs)						
_____ (lbs/1,000 lbs)						
_____ (lbs/1,000 lbs)						
_____ (lbs/1,000 lbs)						
Others (Identify)						
_____ (lbs/1,000 lbs)						
_____ (lbs/1,000 lbs)						
_____ (lbs/1,000 lbs)						
_____ (lbs/1,000 lbs)						

*Indicates gallons discharged (per 1000 pounds of production)

**Indicates pounds discharged (per 1000 pounds of production)

TABLE I

PLANT INTAKE WATER

Corporation _____

Plant _____

City _____ State _____

Product _____

Process _____

Time Period Represented _____

Parameter	Daily Minimum	Daily Long Term Average	Daily Maximum	Calendar Monthly Averages Minimum	Calendar Monthly Averages Maximum	Remarks
Flow (MGD)						
pH (pH units)						
Temperature (°C) - Wastewater						
Temperature (°C) - Ambient Air						
BOD_5 (lbs/day)						
COD (lbs/day)						
TOC (lbs/day)						
TSS (lbs/day)						
TDS (lbs/day)						
NH_3 as N (lbs/day)						
TKN as N (lbs/day)						
Phenol (lbs/day)						
ignificant Metals (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
Others (Identify)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						
_____ (lbs/day)						

Corporation_____
Plant_____
City_____ State_____

6. Has the seed used in the BOD_5 test been acclimated to the waste waters that have been tested?

____ Yes ____ No

If yes, what is the source of the seed?

A ____ sewage treatment plant

B ____ plant treatment facility

C ____ laboratory acclimation

D ____ other explain _____

Corporation_____
Plant_____
City_____ State_____

ORGANIC CHEMICALS OR PLASTICS AND SYNTHETICS (Identify Category)

PART III TREATMENT TECHNOLOGY

To be returned within 60 days of receipt to:

Robert B. Schaffer, Director
Effluent Guidelines Division
U.S. EPA (WH-552)
Washington, D. C. 20460

A. Do you have a treatment system(s) at this plant?

Yes____ No____

If yes, complete the following and attach a separate flow sheet for each distinct treatment facility indicating waste streams treated, unit sizes of treatment equipment, detention times, recycle rates, effluent concentration or design criteria and other pertinent engineering information for operation of the treatment facility. Include treatment of storm runoff, where applicable.

Indicate the process lines for which any portion of the waste water flow is diverted to separate treatment, pretreatment or disposal (e.g. deep well, solvent recovery, incineration, etc.). Which portions are so diverted and which portions are combined for joint treatment?

For each treatment facility complete the following:

Name of Facility _____

Source(s) of Waste Water _____

_____

_____

		Year	Cost (1976 dollars)
1	a. Original installation (Battery limits of treatment plant only)	_____	_____
	b. Other costs (include collection system, piping, pumping, etc.)	_____	_____
2	Estimated replacement cost	_____	_____
3	Estimated total capital expenditure for this facility to date	_____	_____
	Annual cost of operation and maintenance (exclude depreciation and debt service cost)	_____	_____

5 List major modifications or additions since original installation and

Corporation _____
Plant _____
City _____ State _____

state the purpose of the modification or addition.

Modification-Addition	Treatment Facility	Year	Cost (1976 Dollars)	Purp

6 List scheduled modifications or additions and estimated date of comple and state the purpose of the modification or addition.

Modification-Addition	Treatment Facility	Year	Cost (1976 Dollars)	Purp

7 Is nutrient addition practiced:

____ Yes ____ No

8 How many employees (equivalent man-years/year) are primarily engaged as operators of the waste water treatment facility? (exclude maintenan

_____

How many employees (equivalent man-years/year) are engaged as support personnel for the waste water treatment facility?

_____

9 Is an operator always present?

____ Yes ____ No

10 Quantity of wastewater treatment facility solid wastes disposed of at present (dry basis)

_____ lbs/day

Corporation_____
Plant_____
City_____ _____ State_____

11 Moisture content of waste solids disposed of at present
_____% moisture

12 Present disposition of solids

_____

_____

_____

13 Estimated annual cost of solids handling and disposal (1976 dollars)
_____$/ton dry basis

14 Planned future disposition of solids:

_____

_____

_____

15 What are the total annual energy requirements for the treatment facility?

Electrical _____Kwhr
Other (e.g. Heat) _____Btu

16 For discharges of industrial wastes to municipal treatment plants are there local pretreatment regulations applying to you?
Yes ____ No ____.

If yes, reference those regulations and attach a copy.

Corporation_____
Plant_____
City_____ State_____

J. Carbon Sorption Technology.

	Yes	No
Have you determined carbon sorption isotherms on your waste waters?		
Have carbon sorption isotherms been determined for waste waters from your plant(s) by a person(s) other than company personnel?	___	___
Have you or anyone else evaluated carbon columns on waste waters from this plant?	___	___
Do you have carbon sorption data from your plant(s) on:		
raw wastes	___	___
biologically treated wastes	___	___
individual process lines	___	___
combined process lines	___	___
pilot plant studies	___	___
contractor evaluations	___	___
cost evaluations	___	___
plant scale evaluations	___	___
operational units	___	___

or each question above which was answered affirmatively, ive a brief description of the data (source and types of wastes, eriod of time covered, plant involved, extent of data base and ontact personnel suggested) in the space below.

Corporation_____
Plant_____
City_____ State_____

C. Filtration

Have you done filtration studies on your waste waters (sand, multi-media, etc.) beyond what was described in Section A, Part III? ____ Yes ____ No

If yes, give a brief description of the data (source and types of wastes, period of time covered, process stream involved, extent of data base and contact personnel suggested) in the space below.

D. Biological Treatment

Have biological treatability studies been conducted on your wastewaters beyond what was described in Section A, Part III? ____ Yes ____ No

If yes, give a brief description of the data and results (source and types of wastes treated, duration of the study, extent of data base, conclusions of study, and contact personnel suggested) in the space below

E. Have other treatability studies, beyond what was described in Section A, Part III, employing treatment processes such as sedimentation, neutralization, hydrolysis, precipitation, oxidation/reduction, ion exchange, phenol recovery, etc., been run on any of the process wastewater streams from the plant? ____ Yes ____ No

If yes, list on a separate sheet those product/process streams from which such treatability studies were conducted. Identify the sheet as response to III-E.

308 BAT QUESTIONNAIRE

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

Dear Sir:

As you may be aware, we are in the process of reconsidering and reissuing regulations with respect to water pollutants discharged as a result of the manufacture of organic chemicals and plastics and synthetics. The earlier regulations were issued as 40 C.F.R. Parts 414 and 416, respectively.

The reconsideration of organic chemicals is a result of a joint stipulation filed in the Fourth Circuit Court of Appeals entered into on behalf of companies in the industry and the Environmental Protection Agency. The stipulation requires the Agency to obtain new and more reliable data and requires the industry to cooperate in the gathering and furnishing of data necessary to formulate regulations for the organic chemicals manufacturing point source category.

The reconsideration of plastics and synthetics results from the remand by the Fourth Circuit Court of Appeals of the EPA regulations promulgated on April 5, 1974. The Court ordered the EPA to restudy areas where the record was ruled to be inadequate.

To implement these reconsiderations, the Agency collected additional information on the production processes, raw waste loads, treatment methods and costs and effluent quality associated with the manufacture of these materials in a data portfolio mailed to your company on October 18, 1976. The Environmental Protection Agency is again soliciting your cooperation in obtaining information to supplement those data previously requested. This portfolio seeks information not requested in the prior portfolio, particularly with regard to the presence or absence of the priority pollutants.

According to our records, your Corporation produces one or more of the products on the attached list(s) (Part I) which were covered by the referenced regulations.

The information requested shall be provided for each plant of your firm in the format of the attached portfolios. This will allow the Agency to correlate and make available to interested parties the results of the data gathered. If our records are incorrect and you do not feel that the requested information is related to your plant (i.e., you no longer manufacture the products listed or do not produce them at this site), please inform us as soon as possible. If you have supplied EPA previously with the information requested, you need not do so again, however, please indicate to whom you submitted the data.

The purpose of this request is to gather all available, pertinent information and is not designed to create an undue burden on your plant personnel. Please return the portfolio to the Agency as soon as possible, but in no event later than sixty days after receipt of the letter.

For your convenience, the form is divided into three parts, with descriptive headings and instructions for completing the portfolio. Please answer all items in each part of the portfolio. If a question is not applicable to a particular facility, indicate by writing "Not Applicable". If an item is not known, indicate unknown and include an explanation of the reason for not knowing such information. If an item seems ambiguous, complete as best as possible and state your assumptions in clarifying the apparent ambiguity. Also, submit copies of the summary data sheets compiled or used in completing the tables in this form.

The Agency will review the information submitted and may, at a later date, require site visits and additional sampling in order to complete the data base.

Addenda A and B attached are a part of this letter. They provide you with information regarding the legal authority for requiring the completion of the portfolio and your options for requesting that certain information be held confidential.

Thank you in advance for the cooperation of your company. The Environmental Protection Agency is committed to promulgating effluent regulations which are in accordance with the Federal Water Pollution Control Act and which are reasonable. The Agency has found that only with complete cooperation of all parties concerned can thoughtful and fair regulations be published. I am confident that we can anticipate your assistance in carrying out that goal.

Should you have any questions regarding this request, please do not hesitate to contact Paul Fahrenthold with respect to organic chemicals at (202) 426-2497 or Michael Kosakowski at (202) 426-2497 with respect to plastics and synthetics.

Sincerely yours,

Robert B. Schaffer

Robert B. Schaffer, Director
Effluent Guidelines Division (WH-552)

Enclosures

ADDENDUM A: AUTHORITY

This request for information is made under authority provided by Section 308 of the Federal Water Pollution Control Act, 33 U.S.C. §1318. Section 308 provides that; "Whenever required to carry out the objective of this Act, including but not limited to . . . developing or assisting in the development of any effluent limitation . . . pretreatment standard, or standard of performance under this Act" the Administrator may require the owner or operator of any point source to establish and maintain records, make reports, install, use and maintain monitoring equipment, sample effluent and provide "such other information as he may reasonably require." In addition, the Administrator or his authorized representative, upon presentation of credentials, has right of entry to any premises where an effluent source is located or where records which must be maintained are located and may at reasonable times have access to and copy such records, inspect monitoring equipment and sample effluents.

ADDENDUM B: CONFIDENTIALITY

Information may not be withheld from the Administrator or his authorized representative because it is confidential. However, when requested to do so the Administrator is required to consider information to be confidential and to treat it accordingly if disclosure would divulge methods or processes entitled to protection as trade secrets. EPA regulations concerning confidentiality of business information are contained in 40 CFR Part 2, Subpart B, 41 Fed. Reg. 36902-36924 (September 1, 1976). These regulations provide that a business may, if it desires, assert a business confidentiality claim covering part or all of the information furnished to EPA. The manner of asserting such claims is specified in 40 CFR §2.203(b). Information covered by such a claim will be treated by the Agency in accordance with the procedures set forth in the Subpart B regulations. In the event that a request is made for release of information covered by a claim of confidentiality or the Agency otherwise decides to make a determination whether or not such information is entitled to confidential treatment, notice will be provided to the business which furnished the information. No information will be disclosed by EPA as to which a claim of confidentiality has been made except to the extent and in accordance with 40 CFR Part 2, Subpart B. However, if no claim of confidentiality is made when information is furnished to EPA the information may be made available to the public without notice to the business.

Effluent data (as defined in 40 CFR §2.302(a)(2)) may not be considered by EPA as confidential. In addition, any information may be disclosed to other officers, employees or authorized representatives of the United States concerned with carrying out the Federal Water Pollution Control Act or when relevant in any proceeding under this Act.

Instructions for Completing the Attached Questionnaire

Part I

1. Questions 1 through 6: You may have completed these items in response to our previous industry survey. If these items have changed please bring them up to date. In addition, indicate in Item 5 the location of the persons familiar with your response to the questions.

2. Question 7: Lists 1, 2 and 3 on Pages 8, 9 and 10 represent the group of products for which a limited data base exists. In order to insure the continued usefulness of the existing data base, current production data for existing and new plants producing chemicals on Lists 1, 2 and 3 is necessary. The previous questionnaire required the reporting of all "minor" products (defined as being greater than one percent of total production). You are now requested to report the production or use volumes of all chemicals on List 4, if they were not previously reported as greater than one percent of total production, regardless of the production or usage rate.

Part II

1. Tables A through E: Place in the columns of the tables labeled "Analytical Results - Concentration ranges - parts per billion" the number of analytical results which have been obtained, in the range indicated at the top of the column. The column labeled "Method" should be filled with the analytical methodology used for the analyses. (For example: gas chromatography, UV spectrophotometry, IR spectrophotometry, NMR, or wet chemistry.) It is not essential to itemize each analysis. If the number of analyses is greater than 100, estimate as accurately as possible the actual number of analyses. Specify the concentration units used in completing the table - define all abbreviations used.

Part III

For the purposes of Part III of the questionnaire the concept of a "set" of effluent data will be very helpful. A "set" of effluent data contains the following items:

(1) a list of all activated sludge plants located at a facility with the product/process wastewater lines discharging to that facility clearly listed;

(2) a summary of long-term data in the format as requested in Tables A and C of this part (the last two pages of Part III): and

(3) a short summary of specific wastewater parameters which are essential in determining the effectiveness of the biological treatment plant at your facility.

The questions in this part are designed to supplement data previously supplied by requesting all or parts of Items 1, 2 and 3 above. The questions will provide a means of effective performance evaluation of the existing wastewater treatment plants.

1. Question 2: If you feel that recent production levels or wastewater treatment plant data are more representative than data included in your previous submission you may submit a new set of data.

2. Question 3: In Part A, clarification is being requested to enable the Agency to identify specific product/process wastewater lines entering treatment facilities. Part B requests summer/winter performance data from the plant to determine the effectiveness of the biological treatment system in place. The evaluation of treatment effectiveness is necessary to establish the effect of operating conditions on the cost of waste treatment.

3. Question 4: For new plants or modified plants, a full set of information is requested.

Example tables are attached for reference.

EXAMPLE TABLE A,B,C,D or E.

		CONCENTRATION RANGES				
PRIORITY POLLUTANT	UNITS	<10	10-100	100-1000	>1000	ANALYTICAL METHOD
BENZENE	PPM	2	25	20	1	GC-Flame Detector
ETHYLENE DICHLORIDE	PPM	3	2	6	2	GC-EC Detector
CHROMIUM	PPM	0	5	6	2	Atomic Absorption
CHROMIUM	PPB	6	1	0	0	Wet Chemical - SULFATE

NOTE: For Units, use standard wastewater abbreviations, such as:

parts per million = ppm
parts per billion = ppb

Line 1 indicates 48 analytical results in the ranges shown.

EXAMPLE RESPONSE TO QUESTION 7d

	Product/ Process Line	Description of the Process Modification or In-Process Control	Intended Objective of the Modification or Control System
1a	Aromatics Aklylation	Steam stripper installed on raw waste line to treatment plant.	remove benzene, toluene, xylene, ethylbenzene to ppm level.

b Quantify the results of the use of the Modification or PC System

Parameter	Effluent Parameter Value+ Before Modification	Effluent Parameter Values+ After Modification
Benzene	40 ppm, 25 gpm	5 ppm, 20 gpm
Toluene	60 ppm, 25 gpm	2 ppm, 20 gpm
Xylene	10 ppm, 25 gpm	10 ppm, 20 gpm

Repeat the above table for each modification

2a			

	Parameter	Effluent Parameter Value+ Before Modification	Effluent Parameter Values+ After Modification
b			

+Flow and concentration if possible

308 LETTER QUESTIONNAIRE FOR THE ORGANIC CHEMICALS AND PLASTICS AND SYNTHETICS MANUFACTURING POINT SOURCE CATEGORIES

PART I GENERAL INFORMATION AND PRODUCT-PROCESS INFORMATION

To be returned within 60 days from date of receipt to:

Robert B. Schaffer, Director
ATTN: P. D. Fahrenthold
Effluent Guidelines Division
U.S. EPA (WH-552)
Washington, D. C. 20460

1. Name of Corporation

_____

2. Address of Corporation Headquarters

Street: _____

City: _____

State: _____ Zip Code: _____

3. Name of Plant

_____

4. Address of Plant

Street: _____

City: _____

State: _____ Zip Code: _____

5. Name(s) of personnel to be contacted for information pertaining to this data collection portfolio

Name and Title	Telephone	Location (plant or Corp.

Corporation_____
Plant_____
City_____State_____

6. Plant NPDES Permit Number_____

Date of Expiration_____

If No Permit, Application Number_____

Date of Application_____

7. This question consists of four parts, some or all of which may apply to your facility. The parts of the question are labeled i through iv, corresponding to questions 7a, 7b, 7c and 7d. Where questions relate to Lists 1, 2, or 3 an update of the previously submitted portfolio (October 18, 1976) is intended The heading beside the question and the Instructions provide more detail regarding the intent of the questions.

Has your plant (since October 18, 1976)

i. Added new production processes for chemicals on Lists 1, 2, 3, or does it use or produce either as a product, by-product or intermediate any chemical on List 4.

ii. Changed design production capacity or average daily production by means of debottlenecking, removal/replacement of process equipment, etc.

iii. Discontinued production process(es) that is(are) on Lists 1, 2 or 3 (see Part I, Pages 7, 8 and 9).

iv. Installed any new process modifications or in-plant controls which affect raw waste characteristics.

☐ None Apply. Proceed directly to Part II.

☐ Some or all Apply. Continue through Part I.

☐ The data submitted in response to the October 18, 1976, request substantially represents current plant operations. Proceed to Question 1, Part II.

Corporation _____
Plant _____
City _____ State _____

7a. If your facility has added the production of new processes for chemicals on Lists 1, 2 or 3, please complete the table below using the units indicated.

Product	Process	Design Capacity (lbs/day)	Avg. Daily Production While Operating (lbs/day)	Date Process Installed (Year, Month)

Attach additional pages, if necessary.

Corporation_____
Plant_____
City_____State_____

7a. (Continued)
Do you produce as a product or intermediate, consume, package or use* as a diluent, solvent, raw material (feedstock) or intermediate in any manner other than in laboratory research or analytical programs any of the 129 priority pollutants (see Part II, Pages 2 and 3) which were not reported in the previous questionnaire dated October 18, 1976? If so, please list these below.

Product	Process	Design Capacity (lbs/day)	Avg. Daily Production While Operating (lbs/day)	Date Process Installed (Year, Mont

Attach additional pages, if necessary.

*the word "use" in this context excludes uses or production of less than 1000 lbs pe year in any research, pilot or laboratory operation.

Corporation _____
Plant _____
City _____ State _____

7b. If your plant has changed design production rate or average daily production rate through process modifications which involve debottlenecking of certain unit operations by the modification or replacement of equipment, or expansion or other similar projects, please complete the table below using the units indicated for products on Lists 1, 2, 3 and 4.

Product	Process	Design Capacity (lbs/day)	Avg. Daily Production While Operating (lbs/day)	Date Process Changed (Year, Month)

Attach additional pages, if necessary.

Corporation _____
Plant _____
City _____ State _____

7c. If your plant has discontinued, since October 18, 1976, the production of any product on List 1, 2 or 3, please complete the table below using the units indicated.

At your discretion, you may indicate the reasons for the discontuance. For example, was the process line technologically out of date and too costly to update; were applicable environmental controls prohibitive; etc.

Product	Process	Date Discontinued (Year, month)	Reason for Discontinuan

Corporation_____
Plant_____
City_____State_____

7d. If you have installed any process modifications (reactor design, distillation column operating conditions, etc.) or in-plant controls, (steam stripping, solvent extraction, etc.) since October 18, 1976, which either significantly affected or were designed to reduce the raw waste loads (flow or composition) of wastewater discharged to either a treatment facility or directly to surface waters, please complete the following table.*

Product/Process line	Description of the Process Modification or in-Process Control	Intended Objective of the Modification or Control System e.g. remove volatile organics.
1a		

b. Quantify the results of the use of the modification or process control system.

Parameter	Effluent Parameter Values+ Before Modification	Effluent Parameter Values+ After Modification

Repeat the above table for each modification

2a		

b. Parameter	Effluent Parameter Values+ Before Modification	Effluent Paramters Values+ After Modification

+ Wastewater flow and pollutant concentration if possible.

*Attach additional tables, if necessary (photocopy this page).
*Attach flow diagrams or drawings, if appropriate.

LIST 1

PLASTICS & SYNTHETICS

ABS/SAN
Acrylic Resins
Alkyds and Unsaturated Polyester Resins
Cellulose Acetate Fiber/Resin
Cellulose Derivatives
Cellulose Nitrate
Cellophane
Epoxy Resins
Ethylene-Vinyl Acetate Copolymers
Fluorocarbon Polymers
Melamine Resins
Nylon Resins/Fiber
Phenolic Resins
Polyamides
Polyester Resin/Fiber
Polyethylene
Polypropylene Resin/Fiber
Polystyrene
Polyurethane Resins
Polyvinyl Acetate
Polyvinyl Chloride
Rayon
Silicones
Urea Resins

LIST 2

ORGANIC CHEMICALS

Acetaldehyde
Acetic acid
Acetone
Acetylene
Acrylates (includes acrylic acid, methacrylic acid, and esters)
Acrylonitrile
Adiponitrile
Aniline
Benzene
Benzoic acid
Bisphenol A
Caprolactam
Chloromethanes (Methyl Chloride, Methylene Chloride, Chloroform, and Carbon tetrachloride)
Citronellol
Coal Tar
Cumene
Cyclohexane
Dimethyl terephthalate
Diphenylamine
Ethyl acetate
Ethyl benzene
Ethylene
Ethylene dichloride
Ethylene glycol
Ethylene oxide
Formaldehyde
Hexamethylenediamine
Isobutylene
Isopropanol
Maleic Anhydride
Methanol
meta-Xylene
Methyl amines (including mono- di- and tri-methyl amine)
Methyl ethyl ketone
Methyl salicylate
ortho-Nitroaniline
ortho-Xylene
Oxo-Chemicals
para-Aminophenol
para-Cresol
para-Nitroaniline
para-Xylene
Phenol
Phthalic anhydride
Plasticizers (esters of phthalic acid)
Propylene
sec-Butyl-alcohol
Styrene
Tannic acid
Terephthalic acid
Tetraethyl lead
Toluene
Vinyl acetate
Vinyl chloride

LIST 3

ORGANIC CHEMICALS

Acetic acid salts, Total
Acetic anhydride
α-Chlorotoluene (Benzyl chloride)
Adipic acid
2-Aminoethanol (Monoethanolamine)
Aspirin
Benzyl alcohol
Benzoyl peroxide
Benzoic acid salts: Sodium Benzoate, technical and U.S.P. grades
Castor Oil, Ethoxylated
Choline chloride (All Grades)
Cresols, Total
Cyclohexanone
Dichlorodifluoromethane
Diethylene glycol
2-Dimethylaminoethanol
Dipropylene glycol
Dodecyl mercaptans
Ethanolamines, Total
Ethyl alcohol, Synthetic
2-Ethylhexanoic acid (α-Ethylcaproic Acid)
Epoxidized esters, Total
Fumaric acid
Glutamic acid, Monosodium salt
Hexamethylenetetramine, Technical grade
2,2-Iminodiethanol (Diethanolamine)
4,4-Isopropylindenediphenol (Bisphenol A)
2-Methoxyethanol (Ethylene Glycol Monomethyl Ether)
Methyl bromide
N-Butyl Alcohols (N-Propyl carbinol)
Nitrobenzene
5-Nitro-ortho-toluenesulfonic acid (SO3H-1)
ortho-Dichlorobenzene
para-Nitrophenol and Sodium salt
Pentachlorophenol (PCP)
Pentaerythritol
Propionic acid
Propylene glycol (1,2-Propanediol)
Propylene oxide
Salicylic acid
Tetrachloroethylene (Perchloroethylene)
Trichloroethylene
Trichlorofluoromethane
Triethylene glycol
Xylenesulfonic acid, Sodium Salt

Corporation_____
Plant_____
City_____State_____

PART II

INTRODUCTION

The objective of this part is to obtain information related to the analysis of wastewaters, and the detection, quantification and treatment of the 129 priority pollutants named on List 4. The logic flow of the questions in this section is as follows:

(1) Identify which wastewater streams have been analyzed for the 129 priority pollutants.

(2) Report the analytical results for those streams where compounds on List 4 have been detected.

(3) Describe all efforts of any scale directed toward the removal of one or more compounds on List 4 by a wastewater treatment or in-process control system since October 1972.

---

1. The identification of compounds on List 4 has been categorized into three areas as follows: (please check the appropriate box)

☐ a. No analyses have been conducted for any of the compounds on List 4. Please go to Question 4 of Part II.

☐ b. Wastewaters have been analyzed for some of the compounds on List 4. Please go to Question 2 of Part II.

☐ c. Wastewaters have been analyzed for some of the compounds on List 4 (e.g., metals, etc.) and part of the data was reported in the previous Section 308 questionnaire (October 18, 1976). Those priority pollutants previously reported are as follows:

_____ _____ _____

_____ _____ _____

_____ _____ _____

Please go to Question 2 of Part II and report on those not included in the previous questionnaire. If there are no additional List 4 pollutants to those listed above, please go to Question 4 of Part II.

LIST 4

129 PRIORITY POLLUTANTS

Compound Name	Compound Name
acenaphthene	4-chlorophenyl phenyl ether
acrolein	4-bromophenyl phenyl ether
acrylonitrile	bis(2-chloroisopropyl) ether
benzene	bis(2-chloroethoxy) methane
benzidine	methylene chloride (dichloromethane)
carbon tetrachloride (tetrachloromethane)	methyl chloride (chloromethane)
chlorobenzene	methyl bromide (bromomethane)
1,2,4-trichlorobenzene	bromoform (tribromomethane)
hexachlorobenzene	dichlorobromomethane
1,2-dichloroethane	trichlorofluoromethane
1,1,1-trichloroethane	dichlorodifluoromethane
hexachloroethane	chlorodibromomethane
1,1-dichloroethane	hexachlorobutadiene
1,1,2-trichloroethane	hexachlorocyclopentadiene
1,1,2,2-tetrachloroethane	isophorone
chloroethane	naphthalene
bis(chloromethyl) ether	nitrobenzene
bis(2-chloroethyl) ether	2-nitrophenol
2-chloroethyl vinyl ether (mixed)	4-nitrophenol
2-chloronaphthalene	2,4-dinitrophenol
2,4,6-trichlorophenol	4,6-dinitro-o-cresol
chloroform (trichloromethane)	N-nitrosodimethylamine
2-chlorophenol	N-nitrosodiphenylamine
1,2-dichlorobenzene	pentachlorophenol
1,3-dichlorobenzene	phenol
1,4-dichlorobenzene	bis(2-ethylhexyl) phthalate
3,3'-dichlorobenzidine	butyl benzyl phthalate
1,1-dichloroethylene	di-n-butyl phthalate
1,2-trans-dichloroethylene	di-n-octyl phthalate
2,4-dichlorophenol	diethyl phthalate
1,2-dichloropropane	dimethyl phthalate
1,2-dichloropropylene (1,3-dichloropropene)	benzo(α)anthracene (1,2-benzanthracene)
2,4-dimethylphenol	benzo (α) pyrene (3,4-benzopyrene)
2,4-dinitrotoluene	3,4-benzofluoranthene
2,6-dinitrotoluene	benzo(k)fluoranthane (11,12-benzofluoranthene)
1,2-diphenylhydrazine	chrysene
ethylbenzene	acenaphthylene
fluoranthene	anthracene
	benzo(ghi)perylene (1,12-benzoperylene)

LIST 4

129 PRIORITY POLLUTANTS
(Continued)

Compound Name	Compound Name
fluorene	Antimony (Total)
phenanthrene	Arsenic (Total)
dibenzo (a,h) anthracene (1,2,5,6-dibenzanthracene)	Asbestos (Fibrous)
indeno (1,2,3-cd)pyrene (2,3-o-phenylenepyrene)	Beryllium (Total)
pyrene	Cadmium (Total)
tetrachloroethylene	Chromium (Total)
toluene	Copper (Total)
trichloroethylene	Cyanide (Total)
vinyl chloride (chloroethylene)	Lead (Total)
aldrin	Mercury (Total)
dieldrin	Nickel (Total)
chlordane (technical mixture & metabolites)	Selenium (Total)
4,4'-DDT	Silver (Total)
4,4'-DDE (p,p'-DDX)	Thallium (Total)
4,4'-DDD (p,p'-TDE)	Zinc (Total)
a-endosulfan-Alpha	2,3,7,8- tetrachlorodibenzo-p-dioxin (TCDD)
b-endosulfan-Beta	
endosulfan sulfate	
endrin	
endrin aldehyde	
heptachlor	
heptachlor epoxide	
a-BHC-Alpha	
b-BHC-Beta	
r-BHC (lindane)-Gamma	
g-BHC-Delta	
PCB-1242 (Arochlor 1242)	
PCB-1254 (Arochlor 1254)	
PCB-1221 (Arochlor 1221)	
PCB-1232 (Arochlor 1232)	
PCB-1248 (Arochlor 1248)	
PCB-1260 (Arochlor 1260)	
PCB-1016 (Arochlor 1016)	
Toxaphene	

Corporation_____
Plant_____
City_____State_____

2. Complete Tables A through E on the following pages with the results of analyses of wastewaters from processes at the plant site. Include in the tables the names of priority pollutants detected and quantified by the analyses of wastewaters, at the locations described in Tables A through E.

- Table A: Complete this table with the influent raw waste loadings to either on-site treatment or pre-treatment facilities.

- Table B: Complete this table for those process wastewaters which are discharged without treatment to surface waters or to municipal treatment (POTW). Do not include storm waters or non-contact cooling waters.

- Table C: Complete this table for treatment plant effluents discharged to surface waters or pre-treatment facility effluents prior to discharge to a POTW.

- Table D: Complete this table for individual product or process waste streams where priority pollutants have been identified as a constituent. Express values obtained for the priority pollutan in terms of unit raw waste loading, e.g. (lb priority pollutan 1000 lb product).

- Table E: Complete this table for priority pollutants found in the plant intake raw water supply. Its purpose is to establish appropria background levels.

PLEASE READ THESE NOTES BEFORE COMPLETING THE TABLES:

- Please identify all treatment or pre-treatment plants for which Tables A through E apply by a specific designation—especially if more than one facility exists. Use the same name as on the previous 308 letter respons if possible.

- Tables A and C should be influent and effluent of the same facility and identified by the same name.

- Place the number of analytical results which fall within the concentratio ranges shown in the appropriate columns of each table. Please use appropriate concentration ranges such as parts per million, part per billio

Corporation_____
Plant_____
City_____State_____

TABLE A

COMBINED RAW WASTE TO TREATMENT FACILITIES

Complete this table for each treatment or pre-treatment facility at the plant site. Report the results of analyses performed on the influent wastewater to either combined on-site treatment or a pre-treatment facility, for each facility (photocopy the blank page, if necessary).

Treatment/Pretreatment Facility Name_____

Treatment Unit Processes included in the Treatment/Pretreatment Facility

_____

Identify the Product/Process lines which generated the wastewaters for which the analytical results below apply._____

_____

Results presented in the table below were obtained from analyses made

☐ Prior to January 1, 1973 ☐ After January 1, 1973, approximate date_____

Process Wastewater Flow (million Gal/Day) _____ min. _____ avg. _____ max.

		CONCENTRATION RANGES				
Priority Pollutant	UNITS	<10	10-100	100-1000	>1000	Analytical Method

Corporation _____
Plant _____
City _____ State _____

TABLE B

UNTREATED PROCESS WASTEWATER DISCHARGED TO SURFACE WATER OR TO MUNICIPAL TREATMENT

Complete this table with the results of analyses performed on each undiluted process wastewater stream discharged without treatment to surface waters or to municipal treatment. (Photocopy the blank page, if necessary.)

Product/Process producing wastewater _____
or
Wastewater Source _____

Discharge Point _____

Time Period Represented by the Results _____

Results presented in the table below were obtained from analyses made

☐ Prior to January 1, 1973 ☐ After January 1, 1973, approximate date _____

Process Wastewater Flow (million GAL/DAY) _____ min. _____ avg. _____ max.

PRIORITY POLLUTANTS	UNITS	CONCENTRATION RANGES				ANALYTICAL METHOD
		<10	10-100	100-1000	>1000	

Corporation_____
Plant_____
City_____State_____

TABLE C

PROCESS WASTEWATER DISCHARGED FROM FINAL TREATMENT OR PRETREATMENT FACILITIES

Complete this table with the results of analyses performed on the effluent from each wastewater treatment plant or pretreatment plant. (Photocopy the blank page, if necessary.)

Treatment/Pretreatment Facility Name_____

Discharge Point of the Treatment/Pretreatment facility:_____

_____

Results presented in the table below were obtained from analyses made

☐ Prior to January 1, 1973 ☐ After January 1, 1973, approximate date_____

Process Wastewater Flow (million GAL/DAY) _____ min. _____ avg. _____ max.

PRIORITY POLLUTANTS	UNITS	CONCENTRATION RANGES				ANALYTICAL METHODS
		<10	10-100	100-1000	>1000	

Corporation _____
Plant _____
City _____ State _____

TABLE D

PRODUCT/PROCESS LINES RAW WASTE LOADS

Complete this table with the results of analyses on each individual product or process wastewater stream.

Product/Process producing wastewater _____

Or Wastewater Source _____

Results presented in the table below were obtained from analyses made

☐ Prior to January 1, 1973 ☐ After January 1, 1973, approximate date _____

Process Wastewater Flow (million GAL/DAY) _____ min. _____ avg. _____ max.

Does the process wastewater flow include contributions from non-contact wastewater such as cooling tower blowdown, boiler blowdown, etc? If it does, report the percentage as follows: _____ min. _____ avg. _____ max.

		CONCENTRATION RANGES				
PRIORITY POLLUTANTS	UNITS	<10	10-100	100-1000	>1000	ANALYTICAL METHOD

Corporation_____
Plant_____
City_____State_____

TABLE E

PLANT INTAKE WATER

Complete this table with the results of analyses on the raw water intake to the facility.

Results presented in the table below were obtained from analyses made

☐ Prior to January 1, 1973 ☐ After January 1, 1973, approximate date_____

Intake Water Flow (million GAL/DAY) _____ min. _____ avg. _____ max.

PRIORITY POLLUTANTS	UNITS	CONCENTRATION RANGES				ANALYTICAL METHOD
		<10	10-100	100-1000	>1000	

Corporation_____
Plant_____
City_____State_____

3. Describe the sampling and analytical techniques used for priority pollutants.

   . Was/is EPA protocol used for the sampling?_____

   . Was/is the EPA protocol used for the analysis?_____

   . Describe other techniques by pollutant parameter. (attach additional material if clarification of the technique is desirable).

Pollutant	Technique

4. If you have not analyzed for all of the 129 priority pollutants in various waters and wastewaters in your plant, please answer the following questions for those pollutants for which analyses have not been made.

   A. Do you have reason to believe or would you suspect that any of the 129 priority pollutants are present in your plant's raw wastewater or treatment plant effluent as a result of your manufacturing operations or as a result of the presence of your facility at your site? (Do not list suspected presence in intake waters as a source of priority pollutants in answering this question).

      ☐ No - Go to Question 6.

      ☐ Yes - Continue below.

   B. If the answer to 4A is yes, please list the priority pollutants you would suspect to be present and the suspected product source.

Pollutant	Suspected Source

Corporation _____
Plant _____
City _____ State _____

Questions 5 through 8 ask for information on the treatment and research on the treatment of the List 4 priority pollutants. If the information requested was supplied in the previous Section 308 questionnaire, in all cases, please name the priority pollutant and state that the information was already submitted.

---

5. Have treatment facilities (end-of-pipe or in-plant control) been installed specifically for removal of any of the priority pollutants (List 4)?

   ☐ No - Go to Question 7.

   ☐ Yes - Continue below.

   If the answer is yes, please list the treatment unit process or processes, the pollutant(s) removed and whether installed in-plant or end-of-pipe.

   - If in-plant, list the product on which installed.
   - Attach data, flow sheets and drawings as appropriate to define the design criteria and process effectiveness (e.g., removal efficiency, etc.). If this was supplied in the previous questionnaire, please state so after listing the pollutant(s).

Treatment Unit Process(es)	Pollutants removed	Indicate end-of-pipe or specific product on which installed

6. Do you have any data on the removal of specific priority pollutants by existing end-of-pipe treatment facilities or in-process pollutant control processes which were originally designed for removal of conventional pollutant parameters? (e.g., BOD_5, COD, NH, TSS, etc.)

   ☐ No

   ☐ Yes

   If answer is yes, please indicate the treatment process, the priority pollutant(s) studied, the design criteria, and the removal efficiency by the process. Attach flow sheets or other data as appropriate to define.

Corporation _____
Plant _____
City _____ State _____

7. Have you conducted research and/or bench-scale or pilot-scale programs for studying the treatability or removal of one or more of the 129 priority pollutants exclusive of heavy metals?

☐ No

☐ Yes

If the answer is yes, please describe the studies conducted, the priority pollutants examined, and the results of the studies. (attach extra pages if necessary, or provide a copy of the study to complete your answer).

_____

_____

_____

_____

_____

_____

8. Do you have data on the adsorption capacity of activated carbon for specific process wastewaters resulting from the production of any of the compounds presented in Lists 1, 2, 3 or 4.

☐ No

☐ Yes

If yes, list the product/process effluents, the type of carbon tested (granules or powdered) and the adsorption capacity.

Product/Process	Activated Carbon Tested	Adsorption Capacity* (grams adsorbed/ gram carbon)

*Specify adsorbate basis (e.g., COD, TOC, phenol, butadiene, PVA) and concentration ranges evaluated.

Corporation_____
Plant_____
City_____State_____

PART III - TREATMENT INFORMATION

PLEASE READ THE INSTRUCTIONS FURNISHED PRIOR TO PROCEEDING TO THIS PART.

1. Even though you may not now be discharging to a municipal sewer system, is there an adequate municipal trunk sewer close by to which you could discharge?

   ☐ No

   ☐ Yes - Distance from production facilities _____ ft.

   ☐ Already discharging to a municipal system

2. If the recent data from your plant production or wastewater treatment plant operation is, in your opinion, more representative than that submitted in the previous questionnaire, you may submit new data (since October 18, 1976) on Tables A and C attached, being careful to:

   A. Include the beginning and end dates of the period covered.

   B. Show average daily production for each product corresponding to the time period that treatment plant and raw waste parameters are reported in Tables A and C. You may use any of the tables in Part I, Question 7 to report production data.

   C. If raw waste is discharged without treatment, describe discharge point (e.g., municipal sewer, river, etc.).

   D. Indicate where (location) the samples were taken which generated the analytical data presented in Tables A and C.

   E. Continue responding to the questions in this part.

3. Does your plant operate an activated sludge process for treatment of either a single process wastewater effluent or a combination of two or more product/process wastewater effluents?

   ☐ No - It is not necessary to complete the remainder of Part III

   ☐ Yes - (1) If you reported data from your plant in the previous portfolio, dated October 18, 1976, please continue below with Parts A & B.
   (2) If you commenced operation or modified an activated sludge plant since October 18, 1976, and did not report on it or its operation, please go to Question 4.

   Certain areas of the previous question portfolio dated October 18, 1976, were not adequate to provide the necessary information for an effective performance evaluation of the existing wastewater treatment plants. The performance evaluation is essential in determining the cost of waste treatment as a functi of plant operating criteria, external factors such as temperature, etc.

Corporation _____
Plant _____
City _____ State _____

Parts A and B request supplemental or clarifying information resulting from gaps in the previous submittals. Complete this page for each Activated Sludge System on this site

A. If the answer to Question 3 is yes, please list each activated sludge facility operated at your plant and list the product/process effluents included in the influent to the activated sludge process.

Activated Sludge Plant Name	Type of Activated Sludge System (Contact Stabilization, Conventional, etc.)
_____	_____

Product/Process lines discharging to this treatment plant.

_____

_____

Activated Sludge Plant Name	Type of Activated Sludge System (Contact Stabilization, Conventional, etc.)
_____	_____

Product/Process lines discharging to this treatment plant.

_____

_____

Activated Sludge Plant Name	Type of Activated Sludge System (Contact Stabilization, Conventional, etc.)
_____	_____

Product/Process lines discharging to this treatment plant.

_____

_____

Corporation_____
Plant_____
City_____State_____

B. List the following daily average values. Please select a three-month operating period representing typical summer conditions, and if climate changes are significant, select a second three-month period representing winter conditions. Unless otherwise noted below, production levels reported in your October 18, 1976, questionnaire will be used as representative of the data below.

Activated Sludge Plant Name or Designation (Complete for each plant)

		Summer	Winter	
1.	Influent total BOD_5 concentration	_____	_____	mg/l
2.	Effluent total BOD_5 concentration	_____	_____	mg/l
3.	Influent soluble BOD_5 concentration	_____	_____	mg/l
4.	Effluent soluble BOD_5 concentration	_____	_____	mg/l
5.	Influent TSS concentration	_____	_____	mg/l
6.	Effluent TSS concentration	_____	_____	mg/l
7.	Mixed liquor suspended solids concentration maintained in the aeration tank	_____	_____	mg/l
8.	Mixed liquor volatile suspended solids concentration maintained in the aeration tank	_____	_____	mg/l
9.	Temperature of mixed liquor	_____	_____	°C
10.	Detention time maintained in the aeration tank	_____	_____	hours
11.	F/M ratio	_____	_____	
12.	Sludge production (excess biological sludge)	_____	_____	lbs/day
13.	Total oxygen (air) supplied	_____	_____	lbs/day
14.	Is activated carbon added to the activated sludge system?	_____	_____	

Corporation_____
Plant_____
City_____State_____

4. Have you modified, installed or added any wastewater treatment facilities since submission of the October 18, 1976, portfolio.

☐ No - If no, the balance of the portfolio does not apply.

☐ Yes - continue to the next paragraph

A. If the answer to the above questions is yes, please complete the following items, if information is available, for <u>each</u> new or modified wastewater treatment facility.

1. Please describe the modified or new facilities.

2. State the purpose of the change/new installation.

3. Give the month/year of the change/new installation.

4. State the capital costs of the change/new installation.

5. State the operating costs for the system changed or added.

6. Give the new operational parameters. (If the new unit operation is activated sludge, the operational parameters should be listed below.) Attach a diagram illustrating the process as it currently exists.

7. Please complete the attached Table A (treatment plant influent raw waste load) and Table C (treatment plant effluent characteristics) for the new or modified facility.

8. Please complete Sections B and C for each new or modified activated sludge plant.

Corporation _____
Plant _____
City _____ State _____

B. Please list each activated sludge facility operated at your plant and list the product/process effluents included in the influent to the activated sludge process.

Activated Sludge Plant Name	Type of Activated Sludge System (Contact Stabilization, Conventional, etc.)

Product/Process lines discharged to the activated sludge plant

_____

_____

Activated Sludge Plant Name	Type of Activated Sludge System (Contact Stabilization, Conventional, etc.)

Product/Process lines discharged to the activated sludge plant

_____

_____

Activated Sludge Plant Name	Type of Activated Sludge System (Contact Stabilization, Conventional, etc.)

Product/Process lines discharged to the activated sludge plant

_____

_____

Activated Sludge Plant Name	Type of Activated Sludge System (Contact Stabilization, Conventional, etc.)

Product/Process lines discharged to the activated sludge plant

_____

_____

Corporation _____
Plant _____
City _____ State _____

C. Complete this question for each activated sludge plant indicated in Part A above. List the following daily average values. Please select a three-month operating period representing typical summer conditions, and if climate changes are significant, select a second three-month period representing winter conditions.

Activated Sludge Plant Name or Designation _____
(Complete for each plant) _____

		Summer	Winter	
1.	Influent total BOD_5 concentration	_____	_____	mg/l
2.	Effluent total BOD_5 concentration	_____	_____	mg/l
3.	Influent soluble BOD_5 concentration	_____	_____	mg/l
4.	Effluent soluble BOD_5 concentration	_____	_____	mg/l
5.	Influent TSS concentration	_____	_____	mg/l
6.	Effluent TSS concentration	_____	_____	mg/l
7.	Mixed liquor suspended solids concentration maintained in the aeration tank	_____	_____	mg/l
8.	Mixed liquor volatile suspended solids concentration maintained in the aeration tank	_____	_____	mg/l
9.	Temperature of mixed liquor	_____	_____	°C
10.	Detention time maintained in the aeration tank	_____	_____	hours
11.	F/M ratio	_____	_____	
12.	Sludge production (excess biological sludge)	_____	_____	lbs/day
13.	Total oxygen (air) supplied	_____	_____	lbs/day
14.	Is activated carbon added to the activated sludge system?	_____	_____	

TABLE A

WASTE LOADS TO TREATMENT FACILITIES

Corporation _____

Plant _____

City _____ State _____

Treatment Facility Name _____

Treatment Facility Description _____

_____

Wastewater Source(s) _____

Time Period Represented _____

Sample location (eg. combined raw waste sump, etc.) _____

Parameter	Daily Long Term Average Minimum	Daily Long Term Average Maximum	Calendar Monthly Averages Minimum	Calendar Monthly Averages Maximum	Remarks
Flow (MGD)					
pH (pH units)					
Temperature (°C) - Wastewater					
Temperature (°C) - Ambient Air					
BOD_5 (lbs/day)					
COD (lbs/day)					
TOC (lbs/day)					
TSS (lbs/day)					
TDS (lbs/day)					
NH_3 as N (lbs/day)					
TKN as N (lbs/day)					
Phenol (lbs/day)					
Significant Metals (Identify)					
_____ (lbs/day)					
_____ (lbs/day)					
_____ (lbs/day)					
_____ (lbs/day)					
_____ (lbs/day)					
Others (Identify)					
_____ (lbs/day)					
_____ (lbs/day)					
_____ (lbs/day)					
_____ (lbs/day)					

TABLE C

TREATED PROCESS WASTE LOADS DISCHARGED

Corporation_____

Plant_____

City_____ State_____

Treatment Facility_____

Treatment Facility Description_____

_____

Discharge Point_____

NPDES Discharge No._____

Sample location (final discharge line, etc.)

_____

Do you post-chlorinate this effluent? ___Yes ___No

If yes, do you chlorinate ___(A) Full-Time ___(B) Part-Time

Time Period Represented_____

Parameter	Daily Minimum	Daily Long Term Average	Daily Maximum	Calendar Monthly Averages Minimum	Calendar Monthly Averages Maximum	Remarks
Flow (MGD)						
pH (pH units)						
Temperature (°C) - Wastewater						
Temperature (°C) - Ambient Air						
BOD_5 (lbs/day)						
COD (lbs/day)						
TOC (lbs/day)						
TSS (lbs/day)						
TDS (lbs/day)						
NH_3 as N (lbs/day)						
TKN as N (lbs/day)						
Phenol (lbs/day)						
Significant Metals (Identify)						
_____(lbs/day)						
_____(lbs/day)						
_____(lbs/day)						
_____(lbs/day)						
_____(lbs/day)						
Others (Identify)						
_____(lbs/day)						
_____(lbs/day)						
_____(lbs/day)						
_____(lbs/day)						

APPENDIX B

CHEMICAL PRIORITY LIST FOR SCREENING AND VERIFICATION SAMPLING PROGRAMS

APPENDIX B

CHEMICAL PRIORITY LIST FOR SCREENING AND VERIFICATION SAMPLING PROGRAMS

In September, 1977, the Organic Chemicals Manufacturing Industry Working Group approved a priority list containing seven categories of products manufactured by the industry. This Appendix lists the chemicals in each of the top five priorities. The last two priority lists are omitted because of their length.

The seven levels of priorities are as follows:

Priority 1: Chemicals manufactured in excess of 5 million pounds per year (top 100 production items) that are on the list of priority pollutants. This list contains 25 products.

Priority 2: Chemicals derived from priority pollutants that are identified in an ORD survey (Radian report) and are manufactured in excess of 5 million pounds per year. This list contains 19 products.

Priority 3: The organic chemicals on the list of priority pollutants, not including Priority 1 above and not including pesticides. This list contains 67 products.

Priority 4: Chemicals derived from priority pollutants but that are manufactured at less than 5 million pounds per year. This list contains 146 products.

Priority 5: All other organic chemicals manufactured in excess of 5 million pounds per year. This list contains 81 products.

Priority 6: Organic, non-pesticide entries on the TOSCA list that are not in Priorities 1 through 5 above. This list contains 325 products.

Priority 7: The remainder of the 20,000 commercial industrial chemicals.

CHEMICALS IN PRIORITY LEVELS 1 THROUGH 5

Priority 1

Acrylonitrile
Benzene
Bis(2-ethylhexyl) Phthalate
Butyl Benzyl Phthalate
Carbon Tetrachloride
Chloroform
1,4-Dichlorobenzene
Diethyl Phthalate
Dimethyl Phthalate
Di-n-butyl Phthalate
Di-n-octyl Phthalate
Ethyl Benzene
Methyl Bromide
Methyl Chloride
Dichlorodifluoromethane
Nitrobenzene
4-Nitrophenol
Pentachlorophenol
Phenol
Tetrachloroethylene
Methylene Chloride
Toluene
Trichloroethylene
Trichlorofluoromethane
Vinyl Chloride

Priority 2

Acetone
Adipic Acid
Aniline
Benzoic Acid
Benzoid Acid Salts, Sodium Benzoate
Benzyl Alcohol
Benzyl Chloride
Bisphenol A
Cumene
Cyclohexane
Cyclohexanone
Cyclohexanone/Cyclohexanol (AK oil)
Diisopropyl Benzene
Diphenylamine
Fumaric Acid
Maleic Anhydride
Methyl Ethyl Ketone (2-butanone)
Phthalic Anhydride
Styrene

Priority 3

Acenaphthene
Acenaphthylene
Acrolein
Anthracene
1,2-Benzanthracene
Benzidine
Benzo(a)pyrene (3,4-benzopyrene)
3,4-Benzofluoranthene
11,12-Benzofluoranthene
1,12-Benzoperylene
Bis(chloromethyl) Ether
Bis(2-chloroethyl) Ether
Bis(2-chloroethoxy) Methane
Bis(2-chloroisopropyl) Ether
Bromoform (tribromomethane)
4-Bromophenyl Phenyl Ether
Chlorobenzene
Chloroethane
2-Chloroethyl Vinyl Ether (mixed)
2-Chloronaphthalene
2-Chlorophenol
m-Chlorophenol
4-Chlorophenol
4-Chlorophenyl Phenyl Ether
Chlorodibromomethane
Chrysene
1,2,5,6-Dibenzanthracene
1,2-Dichlorobenzene
1,3-Dichlorobenzene
3,3-Dichlorobenzene
Dichlorobromomethane
1,1-Dichloroethane
1,2-Dichloroethane
1,1-Dichloroethylene
1,2-trans-dichloroethylene
2,4-Dichlorophenol
1,2-Dichloropropane
1,3-Dichloropropylene (1,3-dichloropropene)
2,4-Dimethylphenol
4,6-Dinitro-o-cresol
2,4-Dinitrophenol
1,2-Diphenylhydrazine
Fluoranthene
Fluorene
Hexachlorobenzene
Hexachlorobutadiene
Hexachlorocyclopentadiene
Hexachloroethane
Indeno(1,2,3-C,D)pyrene
Isophorone
Napthalene
2-Nitrophenol

3-Nitrophenol
4-Nitrophenol
N-Nitrosodimethylamine
N-Nitrosodiphenylamine-n-propylamine
Parachlorometa Cresol
Phenanthrene
Pyrene
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)
1,1,2,2-Tetrachloroethane
1,2,4-Trichlorobenzene
1,1,1-Trichloroethane
1,1,2-Trichloroethane
2,4,6-Trichlorophenol
2,4,5-Trichlorophenol
N-nitrosodiphenylamine

Priority 4

Acetanilide
Acetphenone
Acetone Cyanohydrin
Acrylamide
Alkylnaphthalenes
Alkyl (C_8, C_9) phenols
Allyl Alcohol
m-Aminobenzoic Acid (Anthranilic Acid)
o-Aminobenzoic Acid
p-Aminobenzoic Acid
Aminoethylenthanolamine
Aniline Hydrochloride
m-Anisidine
o-Anisidine
p-Anisidine
Anisole
Anthraquinone
Benzaldehyde
Benzamide
Benzoquinone
Benzenedisulfonic Acid
Benzenesulfonic Acid
Benzil
Benzilic Acid
Benzoin
Benzonitrile
Benzophenone
Benzotrichloride
Benzyl Chloride
Benzylamine
Benzyl Dichloride
Bromobenzene
Bromonaphthalenes
Chloranil
m-Chloroanaline
o-Chloroanaline
p-Chloroanaline
o-Chlorobenzaldehyde
p-Chlorobenzaldehyde
o-Chlorobenzoic Acid
p-Chlorobenzoic Acid
m-Chlorobenzyl Chloride
o-Chlorobenzyl Chloride
p-Chlorobenzyl Chloride
Chloronaphthalenes
m-Chloronitrobenzene
o-Chloronitrobenzene
p-Chloronitrobenzene
m-Chlorotoluene
o-Chlorotoluene
p-Chlorotoluene
Cyclohexanol
Cyclohexene

Cyclohexylamine
Decahydronaphthalenes
Diacetone Alcohol
2,7-Diaminobenzoic Acid
3,5-Diaminobenzoic Acid
2,4-Dichloroanaline
3,4-Dichloroanaline
m-Dichlorobenzene
o-Dichlorobenzene
p-Dichlorobenzene
Dichlorohydrin
Dicyclohexylamine
Diketene
n,n-Dimethylanaline
2,3-Dimethylanaline
2,4-Dimethylanaline
2,5-Dimethylanaline
2,6-Dimethylanaline
3,4-Dimethylanaline
Dimethyl Sulfide
Dimethyl Sulfoxide
2,4-Dinitrotoluene
2,6-Dinitrotoluene
Dinitrotoluenes (mixed 2,4/2,6)
Diphenyl
Diphenyl Sulfoxide
p-Dodecylphenol
Ethylenediamine
Ethyl Orthoformate
Glyceraldehyde
Glycerin
Glycerol
Hexalene Glycol
Hydroquinone
Maleic Acid
Malic Acid
1-Malic Acid
+ and - Malic Acid
Mesityl Oxide
Methacrylic Acid
n-Butyl methacrylate
Methyl methacrylate
2-Methylaniline
4-Methylaniline
3-Methylaniline
n-Methylaniline
Methylcyclohexane
Methylcyclohexanol
Methylcyclohexanone
Methyl Isobutyl Carbinol
Alpha-Naphthalene Sulfonic Acid, Sodium Salt, & Formaldehyde Condensate
Beta-Naphthalene Sulfonic Acid, Sodium Salt, & Formaldehyde Condensate
Alpha-Naphthol

Beta-Naphthol
o-Nitroanisole
p-Nitroanisole
m-Nitrobenzoic Acid
o-Nitrobenzoic Acid
p-Nitrobenzoic Acid
Nitrophenol
Nitrotoluene
Nonylphenol
Octylphenol
p-Phenetidines
Phenosulphonic Acid, Ammonium Salt, Sodium Salt, Zinc Salt, and Formaldehyde Condensate
m-Phenylinediamine
o-Phenylinediamine
p-Phenylinediamine
Phthalimide
Phthalimide, potassium salt
Phtholonitrile
Piperazine
Resorcinol
Sodium Phenate
Succinic Acid
Sulfanilic Acid
Tetrachlorophthalic Anhydride
1,2,3,4-Tetrahydronaphthalene
Tetrahydrophthalic Anhydride
Tetramethylethylenediamine
Toluene-2,4-diamine
Toluene diamines (2,4/2,6)
p-Toluenesulfonamide
Toluenesulfonic Acid
p-Toluenesulfonyl Chloride
Trichloroaniline
1,1,2-Trichloro-1,2,-trifluorethane
Vinylidine Chloride
Xylenes (mixed)
2,4-Xylenol
Xylidine
m,p-Xylenes

Priority 5

Acetaldehyde
Acetic Acid
Acetylene
Acrylic Acid
Adiponitrile
Amyl Acetate
Amyl Alcohols
Caprolactam
Citronellol
Dimethyl Terephthalate
Ethyl Acetate
Ethyl Amines
Ethylene
Ethylene Glycol
2-Ethylhexyl acrylate
Ethylene Oxide
Formaldehyde
Hexamethylenediamine
Isobutylene
Isopropanol
Linear alcohol ethoxylates
Methanol
M-Xylene
Mono-Methyl Amines
di-Methyl Amines
tri-Methyl Amines
Methyl Salicylate
Nylon salt
o-Xylene
n-Butanol
n-Propanol
p-Aminophenol
o-Aminophenol
p-Cresol
p-Nitroaniline
o-Nitroaniline
p-Xylene
p-Nitrophenol and Sodium Salt
Propylene (propene)
Sec-butyl-alcohol
n-Butyl acrylate
Butylenes
Tannic Acid
Terephthalic Acid
Tetraethyl Lead
Vinyl Acetate
Acetic Acid Salts, Total
Acetic Anhydride
Aspirin
Benzoyl Peroxide
Castol Oil, Ethoxylated

Choline Chloride (all grades)
Cresols, total
Cresote
Diethylene Glycol
Dipropylene Glycol
Diphenylisodecyl phosphate
n-Dodecyl Mercaptans
Tert-Dodecyl Mercaptans
Ethyl acrylate
Ethyl Alcohol, Synthetic
Glutamic Acid, Monosodium Salt
Hexamethylenetetramine, Tech.
Pentaerythritol
Proprionaldehyde
Propionic Acid
Propylene Glycol (1,2-Propanediol)
Propylene Oxide
Salicyclic Acid
Triethylene Glycol
Xylenesulfonic Acid, Sodium Salt
2-Aminoethanol (monothanolamine)
2-Dimethylaminoethanol
2-Ethylhexanoic Acid (a-Ethylcaproic Acid)
2-Methoxyethanol (Monomethyl Ether)
2,2-Iminodiethanol (Diethanolamine)
5-Nitro-o-Toluenesulfonic Acid (SO3H-1)
n-Propanol
iso-Butyraldehyde
n-Butyraldehyde

APPENDIX C

ANALYTICAL METHODS DEVELOPMENT AND REVIEW OF DATA

I. INTRODUCTION

A. General

Perhaps no other aspect of pollution control is more fundamental than the definition of the pollutants to be controlled. An important early step in developing regulations limiting the discharge of pollutants to the environment and in designing a treatment system to meet such limitations is the characterization of the pollutant load by sampling and analysis. After the treatment system has been installed, the discharge must be regularly monitored to evaluate the effectiveness of the treatment system and compliance with the discharge limitations.

In gathering data to develop and support regulations, the reliability of the results is more important than the specific analytical methodology employed to characterize the wastewater. Where available, "standard" methods should be used to eliminate the inconvenience and expense of establishing a non-standard method suitable to the specific wastewater. However, the notion that data of acceptable quality is inherently associated with the use of a "standard" method is incorrect. If performed improperly, however, either a standard or a non-standard method can yield faulty data. (Taylor 1981).

Programs to assure the reliability of analytical results should focus on the quality of the results, not the analytical techniques employed. If the accuracy and precision data accompanying a reported number meet the criteria chosen, the analytical techniques are acceptable. (Amore 1979).

The data produced by all analytical techniques reflect the variations in human and equipment performance that are inherent to the analyses. The critical questions are: What are the precision and accuracy of a reported value and are these acceptable for the application use of the data? These questions are answered by emphasizing quality assurance in laboratory operations and minimizing measurement errors to produce results appropriate to how the data is to be used.

B. Quality Assurance/Quality Control

Quality assurance/quality control (QA/QC) includes all of the laboratory activities necessary to determine the precision (repeatability) and accuracy (relationship to the true value) of an analytical measurement. The accuracy of the measurement is determined by adding (spiking) a known amount of analyte (the pollutant of interest) to a wastewater sample. Recovery (the ratio of the amount of spike detected to the amount that had been added) depends on the matrix (the other pollutants and chemicals in the wastewater) and the analytical technique, and may be used to adjust the observed value to obtain the "true" value (observed value ÷ recovery = true value).

An analytical method that fails to detect an organic compound spiked into pure water (i.e., zero recovery) is inappropriate. When that method has been modified so that substantial (>50%) recovery from pure water is consistent and predictable, and the accuracy and precision have been established, the method is validated. If the accuracy and precision achieved in the pure water is also realized in varying wastewater matrices, the method can be considered

standardized. Accuracy and precision in a wastewater sample usually differs from that achieved in pure water.

Measurements at concentrations near the detection limit for the compound of interest (e.g. less than 10 parts per billion for most organics), create additional problems. The limit of detection for any pollutant is the lowest concentration of that pollutant that is distinguishable from background concentrations with a known degree of confidence. Below this concentration, the pollutant is "not detected". The limit of determination for each pollutant is the concentration at which one can state with a known degree of confidence that the pollutant is present. Between the limit of detection and the limit of determination, the pollutant is "detected but unconfirmed".

Two errors affect any analysis: operator inconsistency and matrix interference. Practice should reduce operator error. Reduction of matrix interferences is more difficult,since it is impossible to anticipate all possible matrix interferences. In metals analysis, most of the interfering compounds are destroyed by acidic high temperature digestion prior to measuring the metals concentration. Digestion prior to the analysis of specific organic compounds however, is not feasible, since it would alter the individual compounds.

Specific methods or, if appropriate, standard methods can be utilized to reduce the interferences from a specific matrix. Regardless of method, the measurements should be validated with an adequate QA/AC program. This approach is in many cases the only practical means of accurately quantifying organic priority pollutants in a variety of wastewater matrices.

C. Wastewater Analysis in the OCPSF Industry

Each product/process employed in manufactured organic chemicals, plastics, or synthetic fibers produces a wastewater containing priority pollutants characteristics of the product/process. Few manufacturing facilities in the Organic Chemicals and Plastics/Synthetic Fibers (OCPSF) industrial category have the same combination of product/processes, so the wastewater generated at a single plant cannot represent the entire industrial category. This diversity creates a wastewater analysis challenge not found in those industrial categories where the product/process mix and the associated priority pollutants are more consistent throughout the industry.

The variability of the wastewater matrices found within the OCPSF industry suggests that a specific analytical method may not produce the same precision and accuracy at all plants. While an off-the-shelf "standard" method may be more convenient to use, it may not be entirely appropriate for every wastewater sample. In a plant manufacturing organic chemicals, for example, the types and amount of pollutants in the process wastewater vary with the product/process operating conditions and with the combination of product/processes that are being operated. To minimize the impact of these variations on the data, an analytical method should be selected that is appropriate for the specific wastewater matrix that is being analyzed. It is also important to ensure that the samples are collected in such a way as to be representative of the wastewater being sampled, and that the integrity of the

samples is protected during short-term storage, transport and preparation for analysis at the receiving laboratory.

D. Available Analytical Methods

At the beginning of the BAT study, EPA had no validated analytical methods for measuring organic priority pollutant concentrations in the OCPSF Industry. Three analytical methodologies were available for measuring individual organic priority pollutants at low concentrations in wastewaters. These methodologies were (1) gas chromatography/mass spectrometry (GC/MS), (2) gas chromatography/conventional detector (GC/CD), and (3) high performance liquid chromatography (HPLC). Conventional detectors include flame ionization (FID), electron capture (EC), photoionization (PI), and Hall electroconductivity. HPLC is recently finding more frequent application, especially in the pesticide industry.

Both GC/MS and GC/CD use gas chromatography to separate the individual compounds extracted from a wastewater sample. The mass spectrometer (MS) is a universal detector that can identify and measure organic compounds without prior programming, whereas conventional detectors (CD) identify a particular compound by comparing its retention time with that of a known compound under the same column conditions.

GC/MS is a broad-spectrum technique--a large number of compounds in a single sample can be identified and measured. It is generally used in conjunction with a solid state microelectronic computer system; the mass spectrometer repeatedly scans the mass spectrum. Because most of the detectable constituents of a sample can be identified from their mass spectra, GC/MS is a very versatile method for determining pollutants in a sample without advance knowledge of what pollutants are there.

GC/CD is a targeted technique; it can recognize and measure only those compounds for which it has been calibrated. The usual method for identifying a GC peak as a specific pollutant is to measure its absolute retention time under strictly controlled operating conditions, or its relative retention time compared to a standard compound under the same conditions.

Sample preparation for either GC/CD of GC/MS can be quite complicated and time-consuming if there are many organic compounds present in the sample matrix. If only a few compounds are present, the sample may be injected directly into the GC. If a large number of compounds are present, however, they must be separated into broad groups by three or more extraction procedures. Complicated sample preparation increases the degree of pollutant loss and sample contamination.

II. ANALYTICAL METHOLODOLOGIES AND QA/QC FOR THE BAT STUDY

The analytical methodologies and QA/AC used in the BAT study are summarized in TABLE C-1 and discussed below.

NOTES TO TABLE C-1

1. The latest EPA EMSL Cincinnati GC/CD and GC/MS methods are also shown, for comparison.

3. Date of first publication of method.

4. Reference document for first release of method.

5. EPA method number and name of analytical technique employed.

6. The portion of sample to be analyzed by the method.

7. Data or specifications for identification of specific compounds.

8. The maximum acceptable number of mass spectrometer scans within which the characteristic mass spectral ions must maximize for the compound to be considered detected.

9. Maximum acceptable discrepancy in GC retention time between a peak and that of the standard for compound identification.

10. The maximum acceptable discrepancy in the ratios of the characteristic mass spectral ions between the standard and those of the sample.

11. The number of characteristic mass spectral ions specified by the method.

12. Information supporting the identification of a compound and the measurement of its concentration.

13. The instrumental method used to calculate compound concentration.

14. Relationship between mass of chemical injected and output signal.

15. The number of data points used for calibration.

16. How often the calibration is to be verified.

17. Criteria and specifications of procedures that support data validity.

18. Specific test which demonstrates instrument performance.

19. Compound(s) used for end-to-end system test.

20. Test which demonstrates mass spectrometer sensitivity and spectrum validity, relative to EMSL standard peak intensity ratios.

21. Standard deviation obtained with multiple analyses of standards (i.e., replicability).

22. Requirements for initial precision evaluation.

NOTES TO TABLE C-1
(continued)

23. Specifications for subsequent precision measurements .

24. Recovery of known masses of standards added to a standard sample (usually reagent water) or to an OCPSF sample.

25. The compounds employed for accuracy measurement.

26. The specification levied by the method.

27. Blanks required to demonstrate freedom from contamination and interferences.

28. Required frequency of analysis of lab blanks.

29. Blanks carried to and from the sampling site.

30. Only major differences between the methods are listed.

31. Gas chromatograph column type.

32. Analyst's flexibility in selecting alternate column.

33. Means by which sample is separated from the water.

34. Analyst's flexibility in removing interferences from sample.

35. Specified ratio of water volume to extract volume.

36. Memo from Telliard to EGD project officers through Bob Schaffer dated May 27, 1977, entitled "Sampling and Analysis Procedures for Screening of Industrial Effluents for Priority Pollutants."

37. Analysis for compounds included in the "volatiles" fraction, containing 30 specific priority pollutants.

38. NS means "no specification given" in method.

39. Employed internal standard, which uses compounds different from the compounds to be measured.

40. Test standard containing all compounds to be analyzed by the method in the particular fraction specified.

41. Decafluorotriphenylphosphine used for calibration.

42. Quality control charts showing deviation of results from true or average value chronologically for each standardization measurement.

NOTES TO TABLE C-1
(continued)

43. Two times the standard deviation of the spike concentration that had been added.

44. AR means as required.

45. One required with each Sample Set (SS).

46. Not applicable; the purge and trap method does not "concentrate" the sample in a liquid phase.

47. The semi-volatiles (acid and base/neutral) fractions. Some protocols also include GC/MS confirmation of a pesticide or PCB found by GC.

48. PCP is pentachlorophenol; Benz is benzidine. These two compounds are employed to test GC column performance for the acid and base/neutral fractions, respectively.

49. Not required by this method.

50. Capillary columns were permitted but were seldom used.

51. Separatory funnel extraction or continuous liquid/liquid extraction.

52. Either internal (see 39) or external (which uses the compound to be measured) standard calibration methods could be used.

53. Required either multi-point calibration or a standard which matched closely the concentration(s) of the compound(s) found in the sample.

54. No precision and accuracy requirement in these methods, but a quality control/quality assurance program containing precision and accuracy requirements was suggested in the Federal Register notice.

55. Surrogate compounds -- compounds which simulate the behavior of the compounds being analyzed.

56. Methods were given in work statements sent by EPA to its contract laboratories.

57. The OCV (Organic Chemicals-Verification Phase) program was directed at testing for a given pollutant or group of pollutants at each plant. Each analyst was allowed to fractionate the sample as seen fit in order to successfully analyze for the pollutant(s).

58. The specifications and requirements set at each EPA contract laboratory for each pollutant reflected the analytical judgment of the analysts and quality assurance personnel.

59. The actual priority pollutant under investigation was tested.

NOTES TO TABLE C-1
(concluded)

60. Analytical precision was determined by analysis of duplicate samples.

61. Each OCV analytical method specifies extraction methods. For the 5-plant study, EPA chose the OCV methods to be used by each contract laboratory. No flexibility in choosing and applying the method (other than that specifically permitted in the method) was allowed.

62. As required and as permitted by the method, based on the judgment of the analyst.

63. This GC/CD method was chosen as typical of the latest 304(h) EMSL Cincinnati methods. Methods 601-613 are all similar.

64. Less than three times the standard deviation obtained by analysis of standards during the sample analysis.

65. Four replicate analyses of standards.

66. Within three standard deviations, combined with analysts' judgment.

67. Cowen, W.F., and J. L. Simons (Catalytic, Inc.) Analytical Methods for the Verification Phase of the Bat Review (for Organics and Plastics and Synthetics Industrial Category). September, 1980.

A. Screening Phases I and II

1. Analytical Methods

As noted in the Federal Register (3 December 1979, p. 69464), when Congress passed the 1977 Clean Water Act, "...section 304(h) analytical methods were not available in many cases....because only on rare occasions had industry monitored or had EPA regulated (priority) pollutants." In the fall of 1977, the only methodology recommended by the EPA was the GC/MS screening protocol, which EPA had not yet validated. The Organic Chemicals Branch (OCB) of EPA's Effluent Guidelines Division adopted this GC/MS analytical protocol for the Screening Phase work. Introduced by the Agency in April of 1977, this protocol was also used extensively by other branches within the Effluent Guidelines Division, by EPA contractors and Regional laboratories, and by private labs.

The analytical methods used during the Screening Phase are described in "Sampling and Analysis Procedures for Screening of Industrial Effluents for Priority Pollutants" (USEPA 1977). Since the purpose of screening was to identify all priority pollutants among a host of other compounds that may be present in a wastewater sample, GC/MS methodology was appropriate because it is not as subject to interferences as other analytical alternatives. The GC/MS screening protocol was intended for the qualitative and semi-quantitative determination of organic priority pollutants during EPA's initial survey of industrial effluents.

The screening protocol's procedure for extracting organic priority pollutants from wastewater samples was either purge and trap, or liquid-liquid extraction. Some compounds may be recovered from the wastewater by either procedure. The efficiency of recovery depends on the vapor pressure (volatility) and water solubility of the compound. When a compound is efficiently recovered by both procedures, the GC conditions determine the procedure of choice. In general, the GC conditions selected for the purge and trap are not suitable for organic priority pollutants that elute from the GC column later than chlorobenzene.

The purge and trap recovery procedure involves purging the wastewater sample with an inert gas (Helium) and trapping the purged organic compounds by adsorption on a resinous substrate (Tenax-silica gel). The trapped organic compounds are subsequently desorbed by heating the trap and directing the desorbate into the GC/MS system. This method detects a group of 29 priority pollutants, which are mostly halogenated C1-C5 hydrocarbons. It was recognized that the two priority pollutants acrolein and acrylonitrile are so water-soluble that they cannot be efficiently recovered by the purge and trap procedure. Direct aqueous injection was recommended for these two compounds, as well as any of the volatiles that may be present at more than one part per million.

For the less volatile organic priority pollutants, a liquid-liquid extraction procedure (Webb 1978) separated them into groups that are selectively extracted at differed pH. Extraction with methylene chloride at pH11 removes basic and neutral compounds. Included in this group are 46 priority pollutants: halogenated aromatics, nitroaromatics, nitrosamines,

polyaromatics (PAH's) and phthalate esters. Extraction with 15 percent methylene chloride in hexane at pH7 removes neutral compounds. Included in this group are 26 priority pollutants: organochlorine pesticides and polychlorinated biphenyls (PCB's). Extraction with methylene chloride at pH2 removes acidic compounds. Included in this group of 11 phenolic priority pollutants: phenol, chlorophenols and nitrophenols.

The solvent extract was dried and filtered by passing it through a short column of sodium sulfate, which had been prewashed with methylene chloride. After evaporating the solvent to concentrate the extract, it was injected into the GC/MS system. Pesticides were to be initially quantified using GC/EC (electron capture detector), since that is a much more sensitive detector than the MS and is specific for compounds containing halogens. Pesticide identity was to be subsequently confirmed using GC/MS, when more than 40 nanograms of the pesticide was injected (EC detector subject to overload).

Metals were determined by flame or flameless atomic adsorption. Total cyanides were analyzed by a colorimetric method after distillation. Total phenols were determined by the 4-aminoantipyrine (4-AAP) colorimetric method, often giving values several orders of magnitude higher than the GC/MS value for simple phenol. The 4-AAP procedure measures most phenols (not para-substituted phenols) as well as various non-phenolic compounds found in industrial wastewaters. The 4-AAP data accompanied metal and cyanide analyses as part of a package of "classical pollutants". Since the 4-AAP test is not compound specific for phenolic priority pollutants, such data had no further use in this study.

2. Quality Assurance/Quality Control

The following discussion refers to the April 1977 revision of the March 1977 Screening protocol (USEPA 1977).

The QA/QC procedures for the volatile fraction of the priority pollutants that are explained next include:

a. Analysis of blanks

b. Daily calibration of the GC/MS system with priority pollutant and internal standards. Daily MS tuning with DFTPP (Decafluorotriphenyl phosphine).

c. Calibration procedure gave recovery-corrected data.

d. GC/MS system testing. Quality control of data precision by replicate analyses of internal standards.

e. No tolerance specifications for compound identification criteria.

A blank is reagent water, i.e., water in which no priority pollutants or interfering compounds can be detected by the analytical method being used. A trip blank septum-sealed in a vial accompanied each shipping container of

samples. The purpose of analyzing a trip blank was to test for contamination during sampling and sample transport. Laboratory blanks were analyzed to demonstrate that the GC/MS system was free of interferences and contamination. Laboratory blanks were to be analyzed each day prior to the first sample and between samples afterward. While practices varied from one laboratory to another, analysis of laboratory blanks between samples was frequently omitted if the previous sample showed a low content of priority pollutants.

The GC/MS system was to be calibrated daily by spiking reagent water at a level of 20 ppb with a "cocktail" of priority pollutants and three internal standards. These compounds were recovered by the purge and trap technique and analyzed by the GC/MS system. From these results, response factors for each priority pollutant could be calculated and used to determine concentration in the subsequent analyses. Since the priority pollutants and internal standards were recovered together from the reagent water during the calibration procedure, concentrations that were subsequently computed from the calibration response factors were recovery corrected values.

The screening protocol also called for the MS to be tuned daily with 20 nanograms of DFTPP. Since the retention time of DFTPP on the GC column used for volatiles was too long to be practical, the tuning requirement was met by replacing the GC column used for volatiles with the one used for base/neutrals. Although not allowed by the screening protocol, some analysts introduced the DFTPP directly into the MS by means of a probe. DFTPP has since been replaced by p-Bromofluorobenzene, so that now the MS tuning compound can be conveniently added with the other standards for the daily calibration and avoid the GC column change necessitated by the DFTPP.

The GC/MS system was to be tested and the precision of the purge and trap-GC/MS procedure was to be routinely determined (frequency unspecified) by spiking reagent water with three internal standards and performing replicate analysis. The three compounds were Bromochloromethane, 2-Bromo-1-chloropropane and 1,4-Dichlorobutane. These compounds are not priority pollutants, but were used because they span the range of GC column retention times of the volatile priority pollutants. Quality control charts were to be constructed showing results as a function of time, or number of analyses performed.

Qualitatively, a weakness of the screening protocol for volatile priority pollutants was the lack of specifications on compound identification criteria. Characteristic masses or mass ranges were tabulated and were the only information afforded for qualitative determinations. No tolerances were given for relative retention time ($\pm$ minutes), or for correspondence with published mass spectral peak height ratios ($\pm$ percent). Most laboratories compensated for this omission by applying the tolerance specifications that were provided for the semi-volatile priority pollutants (see following discussion). If the presence or absence of a compound was in doubt, the laboratories were instructed to report the compound as being present.

The QA/QC procedures for the semi-volatile fractions of the priority pollutants that are explained next include:

a. Analysis of blanks

b. Calibration (frequency unspecified) at two concentrations with priority pollutants and an internal standard (D10 anthracene). Daily MS tuning with DFTPP.

c. Calibration procedure did not give recovery-corrected data.

d. Daily GC/MS system testing with pentachlorophenol (acid GC column), and with benzidine (base/neutral GC column). No quality control of data precision by replicate analysis.

e. Tolerances were specified for compound identification criteria.

A trip blank was to be analyzed with each set of samples. This blank was obtained in the field by pumping reagent water through the sampling pump's system of plastic tubing. For this reason, the trip blank was also known as a "tubing blank." To avoid unnecessary GC/MS analysis of blanks, the screening protocol allowed the extract of the blank to be run on GC/FID, using the GC column appropriate to the acid or base/neutral fraction. If no peaks greater or equal to that of the D10 anthracene internal standard appeared, then a GC/MS analysis of the blank was not required.

The GC/MS system was to be calibrated at unspecified intervals by direct injection of a "cocktail" of priority pollutants and an internal standard (D10 anthracene) at two concentration levels, 10 and 100 ppb. Since the calibration standards were not carried through the extraction procedure, the concentrations subsequently computed from the calibration response factors were not recovery corrected. The screening protocol did not require that the semi-volatile priority pollutant data be corrected for recovery.

The GC/MS system was to be tested each day. To test with the GC column used for the acid fraction (Tenax-GC), 100 nanograms of Pentachlorophenol was to be used. To test with the base/neutral GC column (SP-2250), 40 nanograms of Benzidine was to be used. These compounds were to be injected directly into the respective column. If the compound could not be detected by the GC/MS system, the GC column was to be replaced. There was no requirement to maintain Quality Control charts on the precision of this method, as was required for the purge and trap method.

Relatively rigorous criteria were applied to the identification of semi-volatile priority pollutants. Three conditions were specified:

a. The characteristic ions for the compound must be found to maximize in the same mass spectral scan.

b. The time at which the GC peak occurs must be within a window of ± 1 minute of the retention time of the compound.

c. The ratio of three mass spectral peak heights must agree within ± 20 percent with the relative intensities given for the compound.

If the presence or absence of a compound was in doubt, the laboratories were instructed to report the compound as being present.

B. Verification Phase

1. Background

Although well suited for a qualitative assessment of organic priority pollutants in wastewater samples, the GC/MS method used for the Screening Phase--The Screening Protocol--was not appropriate for the improved quantitation sought in the Verification Phase. Determination of extraction efficiency (percent recovery) and replication to measure precision and accuracy for many disparate waste streams would have made a substantial increase in the number of analyses to be performed. The continued use of the GC/MS method with more rigorous QA/QC would have made the analytical costs prohibitive, and there were not sufficient qualified GC/MS-equipped laboratories available in the fall of 1977 to handle the samples.

OCB obtained data of adequate quality at reasonable cost by substituting conventional detectors for the mass spectrometer and modifying existing "state-of-the-art" GC/CD methods. GC/MS was used to confirm priority pollutant identification, and was routinely reserved for those instances when interferences in the sample matrix so complicated the analysis that the use of GC/CD proved impractical. By using the less expensive and widely available GC/CD methods routinely, and by using the substantially more expensive GC/MS method for 10 to 15 percent of the samples, OCB cost-effectively combined the two methodologies without severely compromising QA/QC. An added benefit of this approach was that the level of QA/QC employed and the practice of the methods in a variety of matrices demonstrated the applicability of the GC/CD methods for the analysis of effluents from product/processes within OCB's assigned industries.

The 1979 Water Pollution Control Federation literature review (Journal WPCF, Vol. 51, No. 6, pp. 1134-1171) of analytical methods used in research published during 1978 (when Verification began) showed that GC/CD was the methodology most often used for measuring organic priority pollutants. Of 186 published investigations involving a number of organic priority pollutants in a wide range of sample types, 150 utilized GC/CD. At least part of the reason for this dominance was the GC/CD instruments were more widely available than GC/MS at that time.

In June of 1977, EMSL published a preliminary collection of GC/CD methods selected from a computerized literature search by the Denver National Enforcement Investigation Center (NEIC) of EPA. A year later EMSL let contracts on a program to validate some of these methods. Although not yet

validated, these methods were proposed in the Federal Register (3 December 1979, p. 69532 ff). Having already begun to use GC/CD methodology during Verification for reasons previously discussed, OCB continued its methods development program concurrent with the Verification Phase. The chronologies and milestones of the two independent GC/CD method development and validation efforts are presented in FIGURE C-1. EMSL's contracting laboratories began delivery drafts of validated GC/CD methods six to nine months after OCB had completed it own verification program. EMSL's validated methods will be promulgated by EPA in 1983 as the 600 series.

Dr. C. A. Hammer of Envirodyne Engineers compiled an initial list of proposed methods for OCB's Verification program from the EMSL selections and the NEIC bibliography. The literature search had focused on methods suitable for groups of priority pollutants that would have similar responses to extraction procedures, chromatographic conditions, and detectors. A method appropriate to each of these was assembled in a loose-leaf manual and made available to both OCB's contractor laboratories and Verification plants.

OCB's objective was to develop site-specific (in many cases matrix-specific) GC/CD methods by analyzing actual industry wastewater samples. Often sampling product/processes effluents with high matrix interference potential, OCB's experimental approach used spiked and duplicate sample analyses to define the validity of each measurement.

2. Analytical Methods

Before the initial plant visit, a list of priority pollutants to be verified was compiled using the analytical results of the Screening Phase (see Section V of Volume II) and predictions from the product/processes known to be operating at the plant. The list and a package of appropriate methods selected from the then current GC/CD methods manual were sent to the plant well in advance of the initial visit to allow time for their review. During the initial visit, a grab sample of wastewater was taken from each location to be sampled. The grab samples were used by the EPA contract laboratory to tune the proposed analytical method to the specific wastewater matrix at each sampling location. Extraction, cleanup, and GC conditions were modified as necessary during three weeks of method evaluation. At the end of that time (approximately one week before the actual Verification sampling was to commence), the method variation found to be most appropriate in each sample matrix was specified. EPA discouraged the contract laboratory from subsequently modifying the method significantly, and informed the plant what methods would be used.

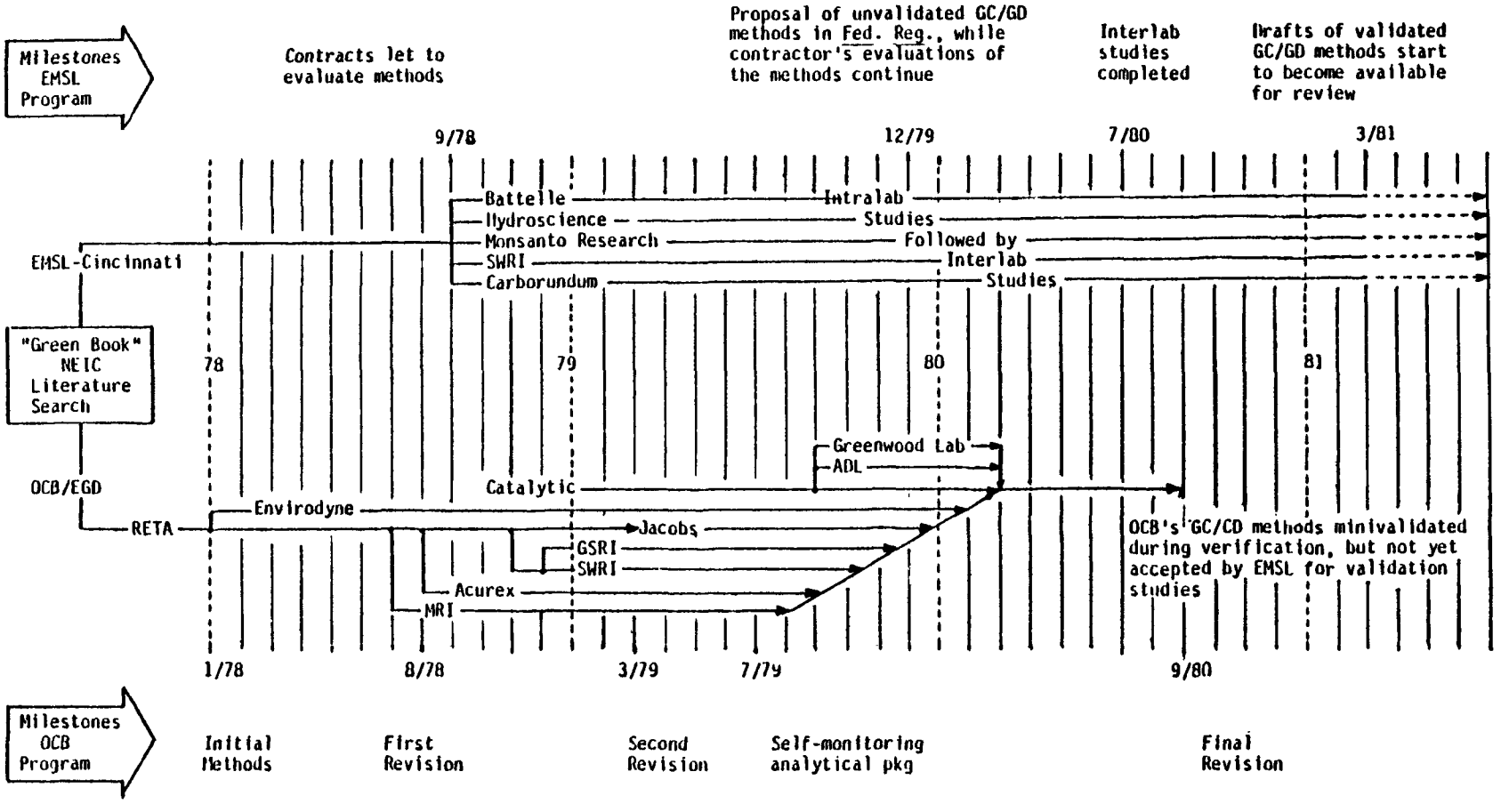
The purpose of discouraging further change in the methods was primarily to offer each plant an opportunity to replicate OCB's Verification sampling and analysis. The benefits anticipated from replication were:

(a) A doubling of the number of data points.

(b) A chance for the plant to evaluate the analytical methods for utility and cost-effectiveness, particularly in comparison to GC/MS; and

FIGURE C-1

Comparative Chronologies of OCB and EMSL Method Validation Programs



(c) An interlaboratory comparison of OCB and plant analytical results would be compiled.

Many plants elected to do no replication or to limit their replicative participation to samples of the final effluent, or of the combined untreated wastewater. For self-verifying plants, the plant performed the principal analysis and OCB's contractor performed the replicate analysis. A potentially important contribution to the methods development effort was reduced by the limited plant participation.

Seven contracting laboratories eventually participated in OCB's Verification methods development: Envirodyne, Midwest Research Institute, Southwest Research Institute, Gulf South Research Institute, Jacobs Engineering Group (PJB Labs), Acurex Corporation, and A.D. Little. Beginning in January 1979, the key analysts from each of these laboratories met approximately every two months to discuss developments and update methods. As methods were matched with the matrices at each plant, variations (usually a change in GC conditions or column) were forwarded to all team members and documented in the methods manual. This continuing update of successful modifications avoided redundant effort and cost-effectively helped resolve new matrix problems simultaneously encountered by members of the team.

Verification methods for metals and cyanides were the same as those used during the Screening phase. For organic compounds, however, the verification methods developed were designed to isolate, concentrate, and quantify one or more compounds from each of the following groups of organic priority pollutants:

(a) Pesticides, PCBs, and phthalates
(b) Phenols
(c) Volatile organics
(d) Halogenated volatile organics
(e) Polynuclear aromatic hydrocarbons
(f) Nitrosamines
(g) Acrolein and acrylonitrile
(h) Chlorobenzenes
(i) Haloethers
(j) Chlorinated hydrocarbons

TABLE C-2 lists the analytical methods used during Verification. Details of each analytical procedure, including precision and accuracy data, are presented in the September 1980, report by W. F. Cowen and J. L. Simons of Catalytic, Inc., entitled "Analytical Methods for the Verification Phase of the BAT Review", under EPA Contract No. 68-01-5011. Analytical methods were varied as required by the sample matrix and each variation was assigned a number. Any method used in the program was identified by a procedure code number, a variation number, and the laboratory that was responsible for its initial use. Several of the procedure codes include methods that were later submitted for validation to EMSL contract laboratories.

Dr. W. F. Cowen of Catalytic, Inc. continually summarized and studied the precision and recovery data to determine which methods gave the most consistent results despite a variety of wastewater matrices. Supplementary

TABLE C-2

ANALYTICALS METHODS USED DURING THE VERIFICATION PHASE

ANALYTICAL PROCEDURE	CODE NO.
Direct Aqueous Injection Procedure for GC Analysis of Acrolein and Acrylonitrile	1*
Method for Benzidine and Its Salts in Wastewater	2
Method for Organochlorine Pesticides and Phthalate Esters in Industrial Effluents	3*
Total Cyanide	4
A-26 Resin/GC-FID Method for Phenols	5
Analysis of Nitrosamines	6
Microextraction Method for Organic Compounds in Industrial Effluents	7*
Purge-and-Trap Procedures for Analysis of Volatile Organic Compounds in Effluents	8*
Method for Polychlorinated Biphenyls (PCB's) in Industrial Effluents	9
Analysis of Arsenic and Selenium in Industrial Effluents by Flameless Atomic Adsorption Spectrophotometry and Hydride Generation	10*
Analysis of Silver, Antimony, and Thallium in Industrial Effluents by Flameless Atomic Adsorption Spectrophotometry	11*
Analysis of Beryllium, Cadmium, Chromium, Copper, Nickel, Lead and Zinc in Industrial Effluents by Flame or Flameless Atomic Adsorption Spectrophotometry	12*
Mercury in Water (Manual Cold Vapor - Atomic Adsorption Technique)	13*
Pentane Extraction of Organics in Wastewaters for GC Analysis	14*

TABLE C-2 (Concluded)

ANALYTICAL PROCEDURE	CODE NO.
Acid Extraction Procedure for Phenols	15
Analysis of Nitroaromatics, Isophorone, and Chlorobenzene	16
Analysis of Polynuclear Aromatic Hydrocarbons in Industrial Wastewater	17
Procedure for Vapor Equilibration (Headspace) Analysis	18
Procedure for Determination of Phenolic Compounds by Solvent Extraction	19
Procedure for the Determination of Neutral and Basic Compounds by Solvent Extraction	20
Procedure for Volatile Aromatic Hydrocarbons by Solvent Extraction	21

NOTE: Code numbers 1 through 18 are the procedures initially compiled for OCB by Envirodyne from EMSL and NEIC information. Procedures 19 through 21 are modifications of procedure Number 7 (Microextraction) for specific groups of organic compounds. Code numbers with an asterisk(*) are those 13 routinely used by OCB's contractor laboratories.

laboratory studies at A.D. Little and Greenwood Labs further developed those methods that showed promise of general applicability. Of the methods originally compiled, some were never practiced because the classes of priority pollutants (e.g., pesticides, PCBs) requiring these methods were not encountered. From the initial compilation of 18 methods used for analyzing organic priority pollutants, 13 were routinely used by OCB's contractor laboratories.

OCB's choice of GC/CD methodology during the Verification phase in preference to the GC/MS Screening protocol resulted in the following advantages:

(a) GC/CD quantification was simplified, through compound spiking to generate recovery data. GC/MS support was used in a number of instances for pollutant identification.

(b) For the typical case of monitoring 10 to 20 compounds by two to three methods, the cost using conventional detectors was less than that using the mass spectrometer. According to information presented by W.L. Budde and J. W. Eichelberger of EPA-EMSL in "Analytical Chemistry", Vol. 51, No. 6, May 1979, p. 567A, for 10 compounds, the cost using conventional detectors is about one-third that using mass spectrometry; for 20 compounds, GC/CD costs about 40% of what GC/MS costs.

(c) The time required for a given analysis was reduced. At the inception of the program, GC/MS laboratories were delivering analytical results three to six months after receipt of the samples. The GC/CD methodology reduced this delivery time to one month.

(d) The wider availability of GC/CD instrumentation and qualified analysts/technicians at the time facilitated the analysis of the large number of samples from the many product/processes studied.

A disadvantage anticipated for GC/CD was that it would require more clean-up of the solvent extract in order to separate subclasses of priority pollutants from each other, as well as from other organic compounds in the sample matrix. In each of the wastewater samples that were examined, no more than 20 priority pollutants were detected; generally 10 to 20 were detected. Chromatographic resolution problems were less than expected, because those subclasses of priority pollutants that are difficult to separate from one another were rarely present in the same sample.

The traditional exhaustive solvent extraction/evaporative concentration methods used at the beginning of the program were gradually replaced by simplified and more expeditious extraction procedures, the most important of which was microextraction. Volatile organic compounds were traditionally extracted exhaustively (continuously or in multiple steps) with excess solvent, coextracting much of the organic matrix. Subclasses of priority pollutants then had to be separated from interfering organic

components by sample cleanup before concentration by evaporation. For the simpler and faster microextraction, a small aliquot of the sample was extracted in one step with an even smaller amount of solvent (100:1). Partitioning of many priority pollutants into the extraction solvent (aided by salting out) left most of the GC-interfering organic compounds in the sample. With the right choice of GC column conditions, it was frequently unnecessary to cleanup the extract to separate interfering organics for a satisfactory GC analysis. Because the extract was already concentrated, evaporative concentration of the extraction solvent was unnecessary. This shortened analysis time significantly and eliminated the potential for alteration or loss of sample components during evaporation.

Later in the program another innovation was implemented: static head-space analyses of volatile organic compounds. This technique's advantages for measuring volatile organics are analogous to the advantages of microextraction for measuring extractable organics. Its original purpose was to prevent the loss of volatile compounds in cases where it was necessary to open the septum-sealed vial for transfer to a purge and trap apparatus, or for compositing.

Another change from traditional methodology was the use of liquid crystal and capillary GC columns for separation of polycyclic aromatic hydrocarbons.

Analysts were allowed to use any method that they considered applicable to a particular sample, as long as it did not require routine use of the GC/MS. The analysts were, however, required to execute QA/QC procedures adequate to validate the method used.

3. Quality Assurance/Quality Control

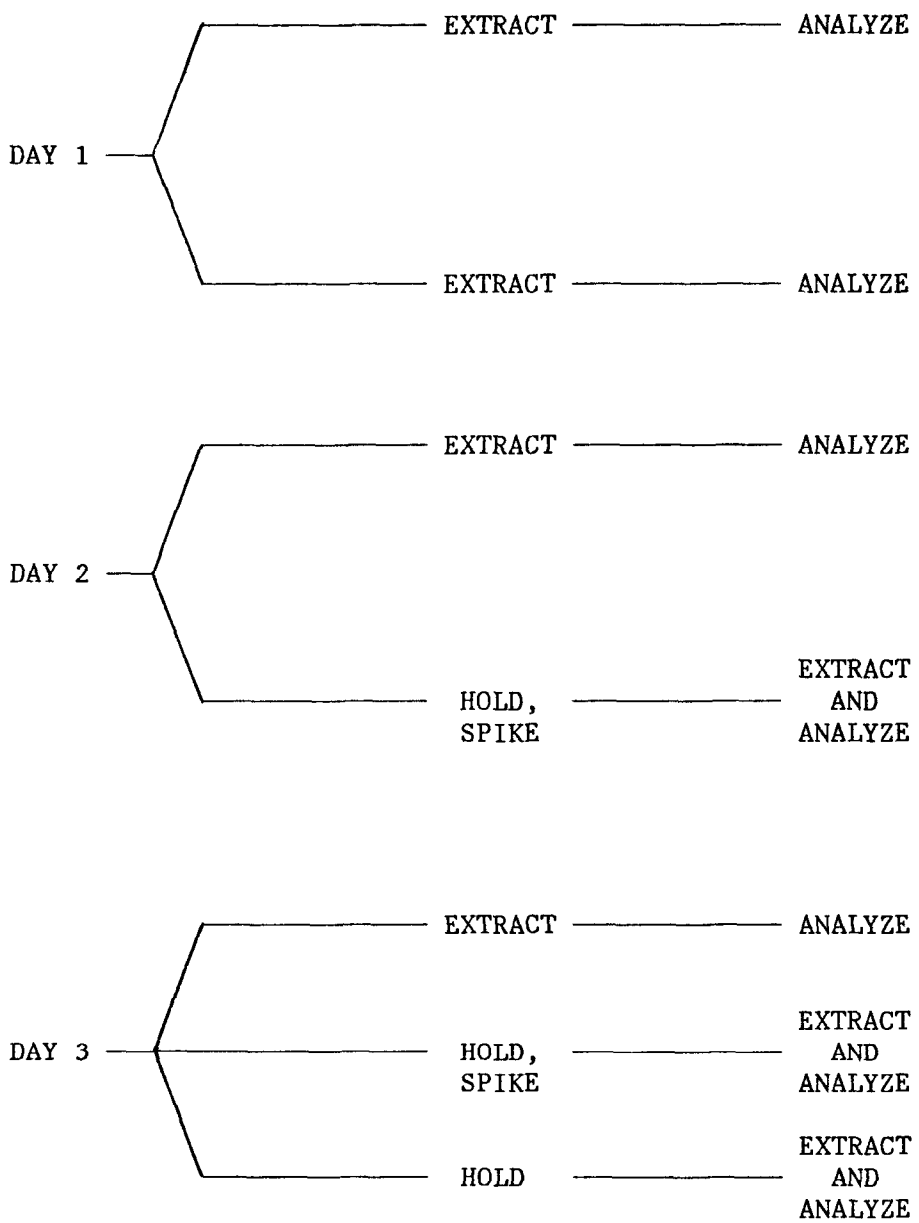
The QA/QC program for verification evolved in three stages. In 1978 (Stage 1), the first six plants were verified with a QA/QC program consisting of a blind spike on the Day 2 composite sample. In the next set of five plants (Stage 2), spikes were added at 2 to 5 times the concentrations detected in the grab samples taken at the pre-sampling meeting. In addition, duplicates of 10 percent of the Day 2 and Day 3 samples were analyzed. When plant personnel collected and analyzed the samples (self-verification), an OCB contract laboratory collected split samples and analyzed them, spiking samples collected on Day 1 of the verification exercise to a concentration double that detected in the unspiked sample. Stage 3 was the program indicated in FIGURE C-2, in which a designed set of spike and duplicate samples were taken. An unspiked, stored control sample (taken on Day 3) was required only in cases of prolonged storage (greater than 48 hours) before spiking. This control was rarely required.

Before adding the spike, it was necessary to wait for the results of the unspiked sample analyses, so that the spike level could be made appropriate for accurate calculation of the recovery of the added spike. Recovery was calculated as:

$$\% \text{ Recovery} = 100 \times \frac{[\text{Detected in Spiked Sample}] - [\text{Detected in Unspike Sample}]}{[\text{Added as Spike}]} \times$$

FIGURE C-2

VERIFICATION QUALITY ASSURANCE/QUALITY CONTROL PROGRAM



where [] denotes concentration.

The formula assumes that the volume of the added spike was small compared to the sample volume. Otherwise, corrections must be made to account for the added volume.

During the Verification program, the recommended concentrations to add for spiking were:

(a) Twenty times the instrument-response detection limit, if the sample concentration was less than 10 times the instrument-detection limit; or

(b) Two times sample concentration, if the sample concentration was at least 10 times the instrument-detection limit.

Ten percent of the samples analyzed for metals and cyanide were spiked, and duplicate analyses were performed on 10 percent.

The purpose of determining spike recoveries on two of the three samples collected at each site during verification was to calculate the efficiency of solvent extraction procedures. Measured concentration may be adjusted for spike recovery. For example, if only 50 percent of the phenol that had been added was detected, the unspiked sample concentration of phenol detected was assumed to be 50 percent of the correct concentration. Correction of the measured concentration may be made by the following equation:

$$\text{Adjusted Concentration} = 100 \times \frac{\text{Measured Concentration}}{\text{Percent Recovery of Spike}}$$

In the case of metals and cyanides, which were not extracted by liquid/liquid partitioning, no adjustments were made to the raw data, although spike recovery data was reported by some of the analytical laboratories. Recovery efficiency and consistency were useful to the analysts during verification in judging whether or not an extraction procedure was appropriate to the sample matrix.

It should be noted that the proposed effluent limitations are based on measured concentrations that were reported by individual laboratories. These concentrations were not further adjusted mathematically for recovery of spike, as indicated above. Exceptions to this are concentration values measured by a system that was calibrated by adding the internal standard directly to the wastewater sample (matrix). When calibration response factors were determined by this procedure, concentrations measured were automatically recovery-corrected.

C. CMA Five-Plant Study

Samples were analyzed for a selected group of priority organic pollutants that were characteristic of each plant, together with certain conventional and nonconventional pollutants. All organic priority pollutants included in this study were not analyzed at all five plants. Analyses were not run for pesticides, polychlorinated biphenyls, or metals.

EPA's contract laboratories analyzed all influent and effluent samples for selected organic priority pollutants using GC/MS or GC/CD procedures (44 FR 69464 et. seq., December 3, 1979, or variations acceptable to the EPA Effluent Guidelines Division). For example, one EPA laboratory used GC coupled with flame ionization detection (GC/FID). Approximately 25 percent of the influent and effluent samples collected at each participating plant were analyzed by the CMA contractor using the GC/MS procedures cited above. The variations in the analytical procedures used by the EPA contract laboratories and the CMA laboratory during this study are summarized in Appendix A of the April 1982 Engineering-Science report entitled "CMA/EPA Five-Plant Study," as are the sampling protocols for each of the five plants.

Each participant provided daily analyses of the convention/nonconventional pollutants in their influent and effluent wastewaters, using the methods found in "Methods for Chemical Analysis of Water and Wastes," EPA 600/4-79-020, March 1979. Additionally, four of the participants analyzed from 25 to 100 percent of the samples collected by EPA for the same organic priority pollutants that were evaluated by the Agency. At a minimum, those analyses included duplication of the CMA contractor's analyses.

III. REVIEW OF DATA FROM SAMPLING STUDIES

A. Introduction

In June, 1982, the EPA requested the Environmental Engineering Committee of its Science Advisory Board (SAB) to review portions of the Contractor's Engineering Report on the Analysis of the Organic Chemicals and Plastics/Synthetic Fibers Industries. Such reviews help EPA realize its goal of developing regulations based on data obtained by credible scientific methods. The SAB was asked to address three major issues in its review of the analytical methods and data used in developing the proposed BAT effluent limits.

1. The adequacy of the overall experimental plan, the analytical methods used and the application of those methods.

2. The quality of the data presented, particularly with respect to whether compounds were adequately identified and whether accuracy and precision were determined.

3. The adequacy of the data for drawing reasonable conclusions from which defensible effluent guidelines could be developed.

At the time of the request, the EPA had not yet completed its summary of the Verification data upon which the proposed BAT effluent limits are based. The SAB was, therefore, unable to address issues (b) and (c). The material available for SAB's review included:

1. A description of the technical approach that was used by EPA and its contract laboratories in developing analytical methods during Verification (see Part I of this Appendix).

2. The analytical methods with multiple variations that were developed and carefully documented during Verification, accompanied by a summary of the recoveries obtained with these methods in a wide variety of OCPSF wastewater matrices.

3. The QA/QC methodology that had been employed during Verification to establish the precision and accuracy of the analytical results.

The SAB criticized the dominant use of GC/CD methodology during verification on grounds that compound identification had not been adequately confirmed. The SAB also criticized the QA/QC methodology as being insufficient. Subsequent to the SAB criticisms, the OCPSF Industry also criticized the verification analytical methodology on similar grounds. At SAB meetings, however, representatives of the OCPSF Industry have stated that during Verification the EPA:

1. Used state-of-the-art GC/CD methods.

2. Used more QA/QC than the OCPSF Industry could have afforded.

In response to these criticisms, EPA has conducted an extensive review of all organic priority pollutant analytical data that it has used in support of the proposed regulations. A similar review for the metal priority pollutant data was unnecessary, since neither the SAB nor the OCPSF Industry were critical of that analytical methodology. The review will be completed prior to promulgation of a final rule. All data not meeting the standards of quality described in the following sections are being deleted from that database being used to develop the regulations.

In the following sections, the method by which the analytical data was reviewed and its quality assessed is described for the three data collection programs:

1. Screening Phases I and II
2. Verification
3. CMA Five-Plant Study

B. Screening Phases I and II

1. Description of the Review

Section II.1 of this Appendix described the analytical methods employed in Phase I and II Screening. Screening data was from one-day composited samples of both treated and untreated wastewaters from over 143 OCPSF manufacturing plants. These studies were the first comprehensive analysis of OCPSF Industry wastewaters and EPA's first large scale field use of GC/MS analytical procedures. At that time, the Agency did not have an existing database on priority pollutants for the OCPSF Industry, nor had a predictive scheme been worked out to show the relationship between product/process chemistry and the occurrence of priority pollutants. Thus, there was nothing available with which the screening results could be compared. Since QA/QC was not extensive in the screening protocol, there was

no systematic means of detecting problems with the analytical procedures with which the data was being acquired.

2. Use of Phase I and II Data

Screening data from a plant, plus additional priority pollutants that were suggested by the raw materials and process chemistry of the products being manufactured at the plant, were used during the verification program to develop a presumptive list of priority pollutants that were to be verified at that plant. These considerations were also part of the priority pollutant selection criteria for plants in the EPA/CMA Five-Plant Study.

The OCPSF plants that were selected for screening during Phases I and II, represent a broad coverage of the product classes listed under the corresponding SIC Codes for these two industrial categories. Thus a compilation of the priority pollutants that were identified during screening of the combined untreated contact process wastewater of all of these plants constitutes a universe of priority pollutants that characteristically occurs within the industry.

The screening data were also used in a multivariate statistical analysis to confirm subcategories. This application of the screening data recognizes the semi-quantitative nature of the data (see Appendix F, "Subcategorization Multivariate Analysis").

C. Verification Phase

1. Background

The Environmental Engineering Committee of EPA's Science Advisory Board (SAB) reviewed the technical approach to wastewater analysis for priority pollutants that had been used during Verification, but not the raw data. The "Report of Meeting in Chapel Hill, N.C. June 24-25, 1982" included in those minutes the findings of the SAB's analytical consultants. This review is still underway and will be completed before publication of the final rule.

2. 1982 Data Review by Original Contract Laboratories

EPA employed six contract laboratories to analyze samples from 29 direct discharge plants in the 1978 to 1980 Verification Study. SAB's major concern was that all the laboratories had used GC/CD (gas chromatography with conventional detectors) methods, which SAB felt needed confirmation by GC/MS (gas chromatography with mass spectroscopy detection) and more extensive QA/AC. The results from three plants were not reviewed extensively because all GC/CD data from the EPA contract laboratories that analyzed samples from these plants had been confirmed by GC/MS during Verification. At two of these three plants, samples had been split and analyzed by both a laboratory under contract to the plant and the plant laboratory. The plant laboratory data agreed with its contract laboratory data within the limits considered normal for these analyses, which obviated a need for extensive review of the data from these plants.

The remaining four EPA contract laboratories performed analyses for samples from the remaining 26 plants. The analysts that had been responsible for the Verification analytical work at the four laboratories met in October, 1982, to adopt a uniform approach to validating the GC/CD data and to attempt to locate all data that might be of some use in confirming the Verification GC/CD data.

From October, 1982, to March, 1983, all of the former EPA contract laboratories and principal analytical chemists from the Verification program cooperated in a review of the data, using the review procedure that had been developed at the October meeting. The analysts examined laboratory notebooks, chromatograms, mass spectral tapes and other information that documented the methods that had been used during Verification. EXHIBIT C-1 (at the end of the Appendix) is a copy of the form used for this review. Every data point obtained by the four laboratories has been or will be reviewed. Pending completion of the review, the data bank for these proposed effluent limitations, which was frozen in December, 1982, has been made available in summary format as part of the public record.

3. Results of the Review

(a) GC/MS Confirmation of GC/CD Data. The presence of many priority pollutants that had been detected by GC/CD during the analysis of Verification Samples was confirmed by GC/MS, or had been confirmed by GC/MS in a preliminary grab sample. These confirmations were encoded into the December, 1982, BAT data summary. While EPA limited the use of GC/MS by its contract laboratories during Verification, the laboratories eventually applied GC/MS confirmation to more than 10 percent of the total samples that were analyzed.

(b) GC/CD Data Qualitation. The criteria EPA applied during its recent review of Verification GC data to eliminate questionable data points were more rigorous than those that were proposed in the 600-series methods (Federal Register, Vol. 44, No. 1233, pp. 69464 to 69552, December 3, 1979), and are nearly identical to published revisions of the 600-series GC/CD methods (EMSL, Cincinnati, EPA-600/4-82-057, July 1982). The only major difference is the criterion for retention time agreement between standard and sample. The 1979 600-series methods do not specify the maximum retention time discrepancy that is acceptable for the identification of a specific compound. The EMSL's July, 1982 revision of these methods proposes "three times the standard deviation of a retention time for a compound"..."based upon measurements of actual retention time variations of standards over the course of a day", and "... the experience of the analyst should weigh heavily in the interpretation of chromatograms". For confirmation of Verification results, EPA's review contractor measured the retention time difference between GC charts with a millimeter scale (aided by overlaying the charts on a light table at some laboratories), or by integrator. EPA's contractors thus applied 1982 criteria to their qualitative review of the GC/CD Verification data.

(c) GC/CD Data Quantitation. Quantitative measurements at EPA's contract laboratories employed multi-point calibration over the working range of the detection system for compounds with non-linear responses, and single-point calibration for those compounds with a linear response. These

calibration procedures are the same as those proposed by EPA in 1979 (Federal Register, Vol. 44, No. 1233, pp. 69464 to 69552, December 3, 1979) and in the July 1982 EMSL methods (EPA-600/4-82-057).

(d) Interlaboratory Comparisons. During Verification at several plants, duplicate samples were analyzed by the plant's laboratory, by a commercial laboratory under contract to the plant, or by the EPA contract laboratory. The data from these analyses offers both qualitative and quantitative comparisons between labs using the same methodology, or where one laboratory used GC/CD while the other laboratory used GC/MS. Such comparisons can be made, when all of the information garnered from the review has been encoded.

4. Effect of the Review on the OCPSF Industry Database

Data for organic priority pollutants that were collected from six plants during Verification have already been deleted from the statistical analysis to determine BAT effluent limitations. These plants were sampled early in the Verification program, when blind spiking was used in the QA/QC procedure. In those instances in which an inappropriate spiking level was used, the data would certainly be quantitatively unreliable and may often be qualitatively suspicious. Since this data was of variable quality, a decision was made to exclude all of it from the statistical analysis.

Since the review will provide GC/MS confirmation for many GC/CD data points, it is expected to enhance the overall quality of the database and justify retainage of most of those influent-effluent data pairs with a significant difference in concentration. In general, the review of the Verification data focused on influent-effluent data pairs where the influent concentration was greater than about 30 ppb.

D. CMA Five-Plant Study

1. Description of the Review

Section V of Volume II describes the Five-Plant Study. Late in 1982, EPA staff and contractors reviewed the data by comparing results from analyses of the same samples at (1) EPA contract laboratories, (2) CMA contract laboratories, and (3) the plant laboratory or plant contract laboratory. Errors in transcription and encoding and in application of GC analyses were found and corrected. This section gives details of this review.

2. Transcribing and Encoding Errors

In the Five-Plant Study, laboratories used GC/MS or GC/CD analytical methods. The data were transcribed from instrument readouts to data sheets and, in some cases, from data sheets to typewritten report forms. EPA then encoded the data from the typewritten forms and data sheets into its Five-Plant computer database. The Agency's 1982 review of printouts from this database revealed occasional disparities between the printout and results that had been reported by laboratories. Typical errors were the transposition of results from treated and untreated effluent streams, typographical errors, and encoding errors. Copies of printouts with suspect values noted were sent to

the original EPA contract laboratories and to the original participating CMA laboratories for confirmation. Confirmed transcription and encoding errors were corrected. In a few instances, laboratories corrected the GC/MS results after re-examining the original analytical data recorded on magnetic tape. The laboratory originally responsible for each data point made the final decision on correcting that data point.

3. Errors from Improper Application of GC/CD Methods

When the Five-Plant Study began, EPA sent samples to an EPA contractor laboratory for analysis using GC/MS methods. From these results, EPA determined which GC/CD methods its contractor laboratory should employ for monitoring the treated and untreated wastewaters at three of the five plants for the 30-day period. In contrast to the flexibility afforded EPA contractor laboratories during the Verification Phase, EPA contractor laboratories in the Five-Plant study were not permitted to modify the selected GC/CD methods or employ alternate methods, if an interference was suspected. EPA now admits this approach was faulty. The contractor laboratories should have been permitted the same flexibility in response to GC/CD interferences, or only GC/MS methods should have been employed.

For each plant, a technical contractor's review compared GC/CD data from EPA's contract laboratory with GC/MS results from the CMA plant laboratory, or CMA contract laboratory. These data showed significant disparities. EPA was then faced with determining which data were acceptable. Two approaches were applied: (1) the GC/CD and GC/MS results were compared statistically; and (2) the technical contractor's review of the GC/CD data evaluated concerns such as the potential interference with GC/CD peaks by non-priority pollutant compounds. The EPA review staff and technical contractor reviewed the chromatograms supporting the GC/CD data at the IFB contractor's facility. In addition, the IFB contractor reviewed the chromatograms and detection limits data and subsequently recommended some of the major changes described next.

The results of the statistical comparison, a paired-sample T-test, were inconclusive because of a shortage of both GC/CD and GC/MS analyses from split samples. EPA's review contractor recommended removal of all GC/CD data from the CMA Five-Plant database because of the disparities with GC/MS results, the impossibility of determining which GC/CD data points were valid, and the failure to use the interference elimination options which had been employed in Verification Phase GC/CD methods.

Following the review contractor's technical recommendation and on its own technical evaluation, EPA decided to delete all GC/CD data from the Five-Plant database. Deletion of GC/CD data reduces the total number of data points by approximately 60 percent. However, GC/MS data exist for all the pollutants detected by GC/CD at all plants. EPA has determined from interlaboratory comparisons that the remaining GC/MS data are adequately precise and accurate for developing the proposed BAT effluent limits. Therefore, all GC/MS data from the Five-Plant database have been retained.

EXHIBIT C-1

ORGANIC CHEMICALS VERIFICATION (OCV) PROGRAM

METHOD VALIDATION FORM LABORATORY: _____

1. APPLICABLE OCV DATA

METHOD _____ PLANT _____ STREAM _____

POLLUTANT _____

2. METHOD SUMMARY

2.1 Extraction: _____

2.2 Column: Length _____ I.D. _____ Plates _____ Est _____

Packing _____

2.3 Detector: _____

3. CONFIRMATORY DATA

3.1 Confirmed by GCMS _____ 2nd Column _____ 2nd Temp _____

Other _____

3.1.1 Qual confirm: Yes ___ No ___

3.1.2 Quant confirm: Yes ___ No ___

GC ___ ug/l GCMS ___ ug/l

3.2 Describe "other" confirmatory technique

_____

_____

3.3 Describe how confirmatory technique was applied:

_____

_____

_____

4. QUALITATIVE DETAILS (not required if results are confirmed both qualitatively and quantitatively by GCMS)

4.1 Retention time window as compared to standard

4.1.1 Absolute ± ___ sec Est ___ Meas ___

4.1.2 Relative ± ___ Est ___ Meas ___ Int Sd _____

4.1.3 Specification applied? Yes ___ No ___ Est ___

4.2 Peak width @ half height

4.2.1 Of standard: ___ mm

4.2.2 Before spike: ___ mm Interference present?

4.2.3 After spike: ___ mm Yes ___ No ___

4.3 Specific detector/interferences

4.3.1 Nature of potential interferences

Responsive _____

Non-responsive _____

4.3.2 Specificity ratio (response to pollutant divided by response to interference: FID = 1.0)

Responsive ___ Est ___ Meas ___

Non-responsive ___ Est ___ Meas ___

4.3.3 Other evidence that only a single compound was measured

_____

4.4 Methodology to remove interferences

_____

_____

5. QUANTITATIVE DETAILS

5.1 Calibration

5.1.1 Int Std ___ Ext Std ___

5.1.2 Number of initial calibration points ___

5.1.3 Frequency of calibration check _____

5.2 Detector Range

5.2.1 Within upper linear limit? Yes ___ No ___

5.2.2 Within calibration limit? Yes ___ No ___

5.2.3 Pollutant level measured: ____ ug/l

5.2.4 Detection limit: ____ ug/l Est ____ Meas ____

5.2.5 Signal-to-noise ratio of pollutant measurement _____

6. STATISTICS

6.1 Replicates

6.1.1 Initial number: ___ % MD ___ Est ___ Meas ___

6.1.2 Ongoing: _____ % RSD ___ Est ___ Meas ___

6.1.3 Control limits: Upper: ___ Lower: ___

6.2 Inter-lab comparisons: % RSD ___ Est ___ Meas ___
List other labs _____

7. COMPARISON WITH 1979 FEDERAL REGISTER 600 SERIES METHODS (if method used was 600 series method, skip this section)

7.1 Nearest 600 series method: _____

7.2 Expected comparison

7.2.1 Detection limit: Yes ___ No ___ Est ___ Meas ___

7.2.2 Linear range: Yes ___ No ___ Est ___ Meas ___

7.2.3 Specificity: Yes ___ No ___ Est ___ Meas ___

7.2.4 Reproducibility: Yes ___ No ___ Est ___ Meas ___

7.3 Was 600 series method available at the time this sample was analyzed? Yes ___ No ___

7.4 Do you feel that the method used produced data comparable to the 600 series method on this sample?
Yes ___ No ___
If no, why not? _____

8. RECONCILIATION WITH PRODUCT/PROCESS

8.1 Is pollutant presence consistent? Yes ___ No ___

8.2 Was pollutant found in raw water or blanks?
Yes ___ No ___

8.3 Evidence that matrix was constant:
_____
_____

9. REGULATORY

I believe the pollutant reported on this form was qualitated and quantitated accurately.
Yes ___ No ___

10. PERFORMANCE EVALUATION: Date _____

11. Other comments

_____

_____

_____

_____

Signature: _____ Date: _____

Print: _____

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APPENDIX D

ACTIVATED CARBON AND STEAM STRIPPING QUESTIONNAIRES

ACTIVATED CARBON QUESTIONNAIRE

Company _____ _____

Location _____

Date _____

U.S. ENVIRONMENTAL PROTECTION AGENCY

ORGANIC CHEMICALS BRANCH

EFFLUENT GUIDELINES DIVISION

SURVEY OF ACTIVATED CARBON WASTE WATER TREATMENT SYSTEMS

The purpose of this survey is to gather data on activated carbon treatment for the removal of priority pollutants from waste water discharges. Of particular interest are the procedure for design of systems and the availability of procedures for predicting the performance of activated carbon on priority pollutants, especially when other adsorbable or nonadsorbable compounds are present.

Relatively simple and short responses to the questions will generally suffice. However, any amplification or additional comments will be appreciated.

Company _____

Location _____

Date _____

PART I: SYSTEM IDENTIFICATION

1. Company_____

2. Location_____

   Person responding or to whom further questions should be sent

   _____

3. Do you have an activated carbon system (ACS) operating on a waste water stream containing one or more of the priority pollutants listed in Table I attached?

   Yes ___ If the answer is yes, please continue with this questionnaire. If more than one installation is involved, copy this questionnaire and complete one set for each installation.

   No ___ If the answer is no, please return the questionnaire; no further data are required.

Company _____

Location _____

Date _____

PART II: SYSTEM DESCRIPTION

1. Please diagram the treatment system using gross blocks for pretreatment and post-treatment (if used) and more detail for the activated carbon system (see examples).

2. Give flow rate of waste water _____lb/hr

   Temperature _____°F

   Composition (at entrance to ACS contact)

3. Effluent composition after ACS contact.

4. AC loadings (by component, if available).

   _____lb/100 lb AC

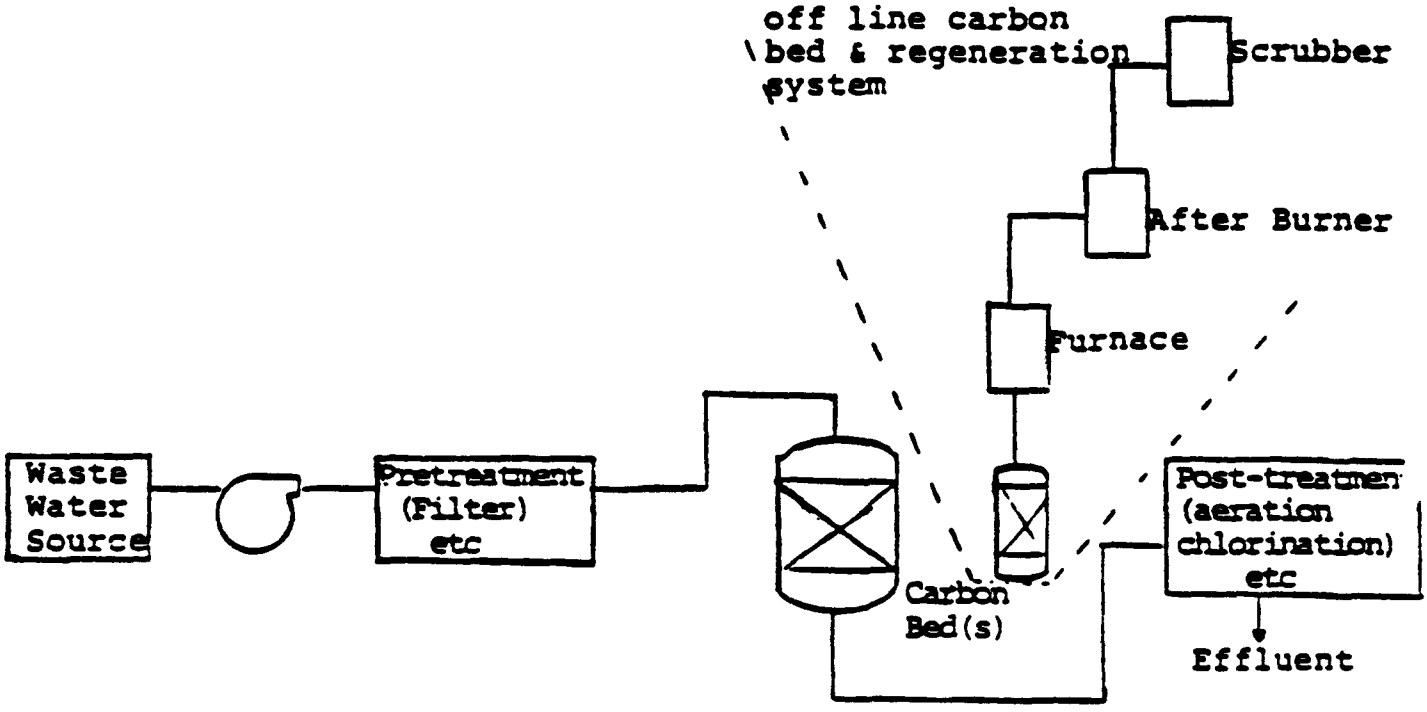
5. Residual on AC after regeneration or reactivation (if used).

Company _____

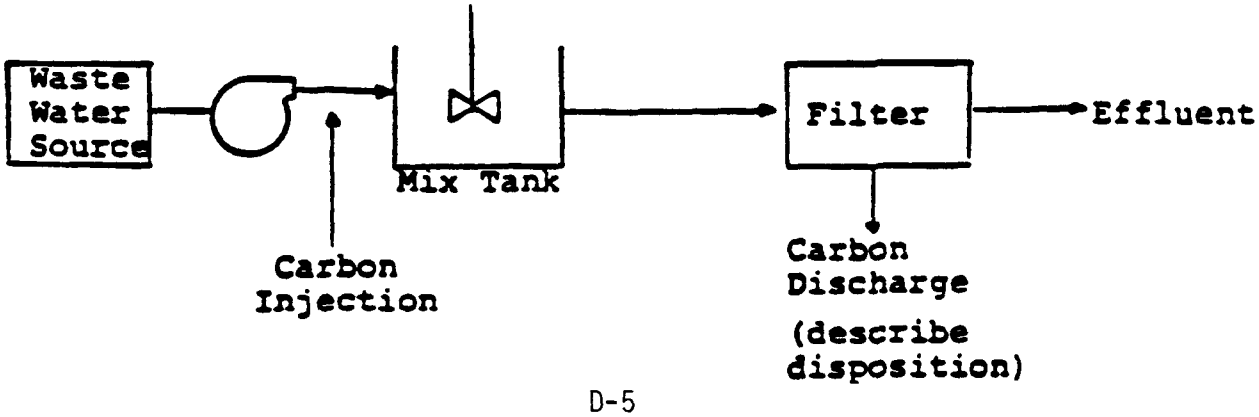
Location _____

Date _____

PART II: EXAMPLE DIAGRAMS



FIXED BED SYSTEM



Company _____

Location _____

Date _____

PART III: DESIGN BASIS

1. Was system bench scale tested or piloted before design?

Yes _____

No _____

2a. If answer to 1 is yes:

Describe tests and note whether individual components, a synthetic mixture, or the authentic plant effluent was used.

2b. If answer to 1 is no:

Describe basis for design of plant unit.

Company _____

Location _____

Date _____

PART IV: COMMENTARY

Based on experience with the unit described, comment on the following:

1. Can AC systems be specified from available design parameters without need for tests?

2. If testing is believed necessary, describe briefly minimum program to assure meeting effluent guidelines.

3. How does the actual performance of the unit described correspond with the predicted performance? If available, give quantitative results for individual components.

4. By hindsight, how would you modify the procedure used for specifying and designing the subject system?

STEAM STRIPPING QUESTIONNAIRE

Form Approved
OMB No. 158-R0160

Company _____

Location _____

Date _____

STEAM STRIPPING

Copy and answer Parts I through IV of this questionnaire for each steam stripper used to reduce the raw waste loading prior to direct discharge or discharge to an end-of-pipe treatment system (whether it be a publicly owned treatment works, a regional industrial treatment system, your own on-site treatment system, or other system such as a nearby refinery's biological treatment system).

PART 1

1. List the names of all process(es) whose waste water discharges constitute a portion of the feed (charge) to the steam stripper. In the event more than one process waste water stream makes up the charge (feed) to the stripper, give the approximate percentage of each stream on a flow or weight rate basis. Please place the percentages of each stream in the table below.

_____

_____

_____

_____

Stripper Feed Source		*Percentage of Feed
1. _____		1. _____
2. _____		2. _____
3. _____		3. _____
Total Feed flow (gpm)	and/or	weight (lb/hr)
_____		_____

* Indicate whether basis is weight (lb/hr) or flow (gpm).

Company _____

Location _____

Date _____

Part II

This part of the survey requests information adequate to assemble a trial material balance around the stripper. Operating data is the preferred source of information requested in this part. Flow or stream composition data based on limited monitoring or calculations (engineering estimates) is required as an alternative.

1. Please attach a process flow diagram of the steam stripper. Kindly number and label all waste streams that are associated with the operation of the stripper such as the charge (feed), reflux, overhead product, decanter water, bottoms, etc. Indicate in your drawing major equipment items such as pumps, heat exchangers, etc. A sample sketch has been provided for reference.

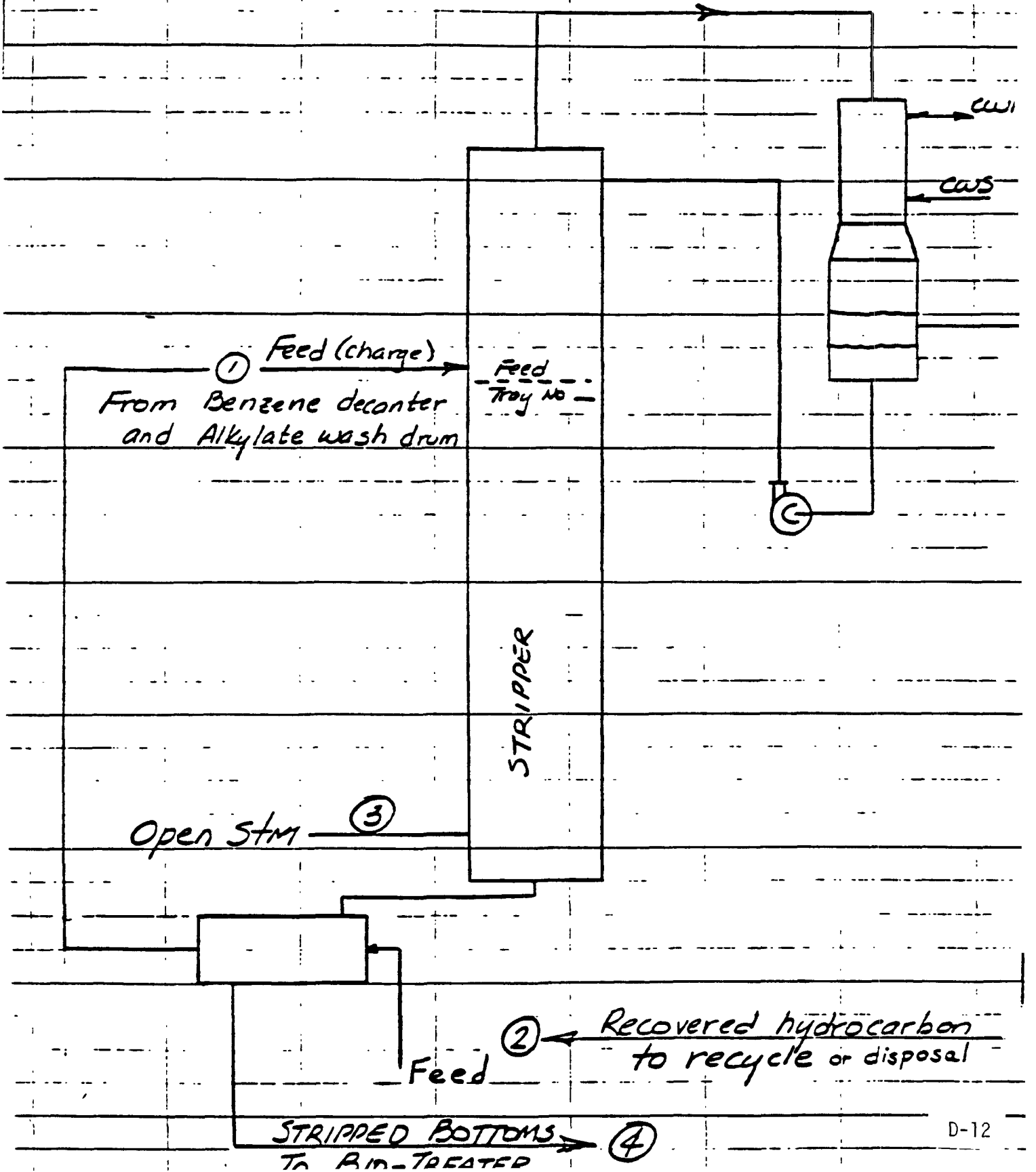
2. Complete the attached Table I with the information requested for each stream numbered in the sketch prepared in (1) above. Note that two sets of information are requested. One set consists of general stream characterization parameters such as flow, temperature, pH, BOD, TOC, etc. with spaces for additional or different characteristics, and organic or inorganic compounds known to be present. The second set labeled "component" refers to priority pollutants identified or indicated to be present in the waste waters associated with the stripper.

Company _____

Location _____

Date _____

Sample Process Flow Diagram
Steam Stripping



Part II

TABLE I

	STREAM NUMBER				
	1	2	3	4	5
FLOW, lb/hr TEMP, °C TOC, mg/l BOD, mg/l pH					
COMPONENT	Concentration ppm				

Company _____

Location _____

Date _____

Complete Table I according to instructions on page 2

Company _____

Location _____

Date _____

Part III

This part of the survey requests information essential to evaluate the operating performance of the stripper and the energy consumption of the operation per unit of pollutant removed from the waste water.

1. Utility Requirements

   A. Steam Requirements

      Pressure _____ psia

      Temperature _____ °F

      Rate _____ lbs/hr

Is open steam used or does the column utilize a reboiler?

_____ Open Steam _____ Reboiler

   B. Cooling Water Use

      Condenser Influent Temperature _____ °F

      Condenser Effluent Temperature _____ °F

      Flow Rate _____ gal/hr.

   C. Other Energy Requirements

      Electricity _____ kwh

      Compressed air _____ scfm

      Inert gas _____ scfm

2. Column Specifics

   (The sketch called for in II(1) above can be expanded upon to provide the following information).

Company _____

Location _____

Date _____

Part III (Continued)

A. Feed Rate _____ lb./hr.

B. Feed Temperature _____ °F

C. Operating Pressure

Top _____ psia

Bottom _____ psia

D. Operating Temperature

Top _____ °F

Bottom _____ °F

E. Column Diameter _____ ft.

F. Number of Theoretical Trays (if known) _____ No.

G. Actual Number of Installed Trays or Packing Height _____ No. or ft. (specify)

H. Type of Trays or Packing _____

I. Tray Spacing _____ ft.

J. Overall Column Height _____ ft.

K. Reflux Ratio* (if any) _____

L. Reflux Rate _____ lb./hr

M. Reflux Temperature _____ °F

N. Bottoms Flow Rate _____ lb./hr.

O. Bottoms Temperature _____ °F

P. Materials of Construction

Trays _____

Packing _____

*Use overhead flow rate as the basis of this value

Company _____

Location _____

Date _____

Part III (Continued)

Column or Vessel_____

3. Specify the ultimate disposition of column overheads (i.e., incineration, returned to process, etc.) for both the aqueous and the organic phases.

_____

_____

_____

_____

4. Specify the method of disposition of column bottoms. (i.e., discharged to biological treatment, discharged directly to surface waters, reused as cooling tower make-up, etc.)

_____

_____

_____

_____

5. Are there any substances present in the influent stream to the steam stripper that interfere with the removal of the pollutant(s) listed in (2) above. (i.e., maximum boiling azeotropes, pH adjustment, foaming, scaling, necessity to equalize flow or feed concentration, etc.). If so, please list and explain the nature of the interferences and any methods devised to minimize or eliminate these interferences. Also explain how successful these methods have been.

_____

_____

_____

_____

Company _____

Location _____

Date _____

Part III (Continued)

---

6. Operating Specifics

A. Is the steam stripper operated in a continuous or batch mode? If batch, explain.

_____

_____

_____

_____

_____

_____

B. Explain the method of treatment of the process wastewater that is normally discharged to the stripper when the steam stripper is down for repair.

_____

_____

_____

_____

_____

_____

Company _____

Location _____

Date _____

Part IV

Your responses to the following questions will be used to determine the desireability of additional follow-up relating to capital and operating costs associated with the steam stripper.

A. Was the steam stripper installed as a new piece of equipment?

Yes _____ No _____

B. When was the steam stripper installed?

_____

C. Do you have detailed cost information (both capital and operating) relating to the steam stripper as a separate unit?

Yes _____ No _____

D. Are you willing to share this cost information with EPA to be used to verify the cost of the installation of steam strippers for the treatment of waste water.

Yes _____ No _____

E. Operating Labor

Direct Operating_____work-days/yr.

Maintenance_____work-days/yr.

Supervisory_____work-days/yr.

F. Do you have V-L equilibrium data which was used to design the stripper from your own experiments, or Henry's Law Constants, or vapor pressure data, or activity coefficient data, or other correlations?

No_____ Yes_____ Identify which one_____

Company _____

Location _____

Date _____

Part IV (Continued)

G. Do you have information regarding the cost impacts of installing the stripper on the following off-site activities? (check either Yes or No, or indicate (a), (b) or (c) as appropriate)

		Yes	No
a.	Steam generation or a major revision in distribution	_____	_____
b.	electrical substation capacity	_____	_____
c.	Instrument air capacity	_____	_____
d.	Valve/Piping/Wiring systems to supply a, b, or c	(a) (b) (c)	(a) (b) (c)

APPENDIX E

TREATABILITY STUDIES

APPENDIX E
TREATABILITY STUDIES*

GENERAL

The following is a summary of treatability studies sponsored by the Organic Chemicals Branch on activated sludge, activated carbon adsorption, steam stripping, and organic resin adsorption processes. The intent of the research was to collect data on biological and physical constants for specific priority pollutants and derive methods for predicting the removal of pollutants in single and multi-component waste streams. These data were intended for use in benchmarking the Agency's computer Model (see Appendix K).

BIOLOGICAL TREATMENT

Introduction

Activated sludge treatment is perhaps the most common treatment technology practiced by industry. To determine which pollutants are effectively removed by this technology in real systems, the Organic Chemicals Branch developed a pollutant-based treatability model for activated sludge treatment. Beyond the evaluation and application of existing models of biological processes, data for specific priority pollutants were required to accurately predict their susceptibility to removal, their effects on the biological treatment process, and their fate during the treatment process.

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*From U. S. Environmental Protection Agency. 1981. Contractors Engineering Report Analysis of Organic Chemicals and Plastics/Synthetic Fibers Industries, Effluent Guidelines Division, Contract No. 68-01-6024, Chapter 3.

Biological treatment involves the breakdown and stabilization of organic material by aerobic or anaerobic microorganisms. Organic matter is removed from wastewater by microbial oxidation and cell synthesis, essentially accelerating the natural water purification mechanisms. Bacteria are the primary organisms involved in the transformation of waste constituents ultimately into carbon dioxide, water, cellular building blocks, and energy. Treatment processes available for industrial application include: variations of the activated sludge process, aerated lagoon systems, oxidation or contact stabilization ponds, trickling filters, rotating biological discs, and anaerobic lagoons, or digesters. Historically, the activated sludge process, in which aerobic microorganisms are mixed with the influent wastewater and subsequently removed as a sludge, has had the broadest application to industrial wastes. The toxicity of the waste, the biodegradability of the waste (typically judged by the BOD/COD ratio), and the metabolic rate of the microorganisms (i.e., the effective removal rate) all influence process efficiency.

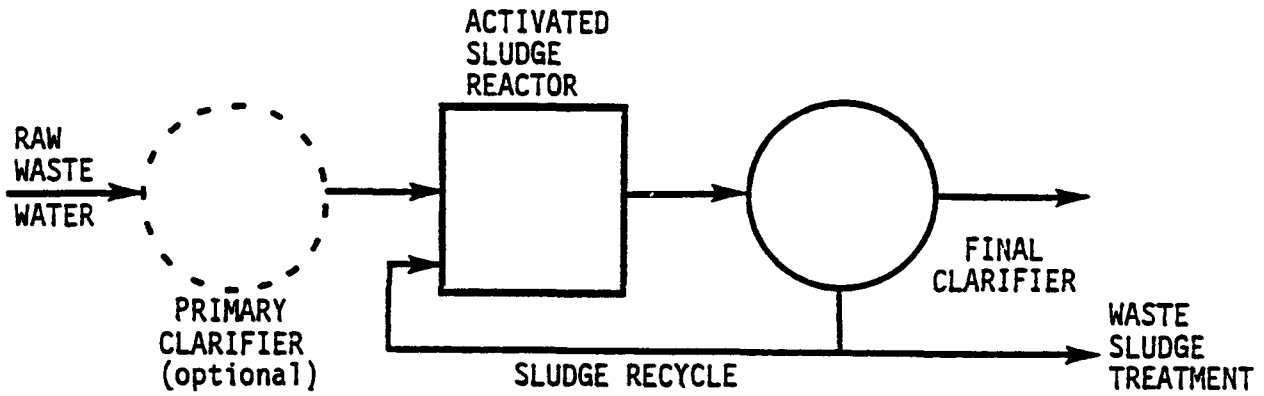
Basic environmental conditions (i.e., proper microbial growth conditions) must be met for microbial metabolism and stabilization of the waste organics to occur. These conditions include (1) oxygen availability, (2) near neutral pH, (3) available growth-limiting nutrients, nitrogen and phosphorus, (4) absence of toxic materials, and (5) adequate mixing. Further, biological processes can be designed to operate optimally by properly controlling the following rate-controlling variables: (1) microorganism concentration, (2) bacterial acclimation or adaptation, (3) temperature level, (4) contact duration and mode, and (5) organic feed concentration.

The activated sludge process was chosen for the modeling effort conducted by Catalytic, Inc., because it has proven to be cost-effective in treating relatively low concentrations of organics found in industrial wastes, and can be designed to provide more operational flexibility than other types of biological treatment. A flow diagram for a

typical activated sludge process, in which aerobic microorganisms are mixed with the influent wastewater and subsequently removed as sludge, is shown in Figure E-1.

FIGURE E-1

TYPICAL ACTIVATED SLUDGE PROCESS



SOURCE: Kincannon and Gaudy, no date.

Biokinetic Models

The theoretical approach used in the design of biological treatment systems is to develop mathematical models which depict relationships between parameters that control efficiency of microbial growth and substrate removal. The purpose of these design models is to provide predictive equations consistent with the underlying metabolic principles governing the waste treatment process. The general approach to developing biokinetic models is to write mass balance equations describing the mass rate of change in substrate (i.e., organic pollutants) and in biomass of the microorganisms. The models incorporate various assumptions regarding fundamental relationships governing microbial growth, and factors derived from laboratory bench scale or pilot plant studies.

Various models, or kinetic approaches, are available for use in designing activated sludge processes. The basic formulas for four well-known models--Eckenfelder's, McKinney's, Lawrence and McCarty's, and Gaudy's--are presented in Table E-1. A materials balance for substrate (S) can be derived, as shown in Table E-2 from each equation.

TABLE E-1

KINETIC APPROACHES FOR THE ACTIVATED SLUDGE PROCESS

Design Approach	Basic Formula
Eckenfelder	$V = \frac{(S_i - S_e)\ F}{K_e\ S_e\ X}$
	$V = \frac{S_i (S_i - S_e)\ F}{K_e'\ S_e\ X}$
McKinney	$V = \frac{(S_i - S_e)\ F}{K_m\ S_e}$
Lawrence and McCarty	$V = \frac{\theta_c Y_t (S_i \theta S_e)\ F}{(1 + K_d \theta_c)\ X}$
Gaudy	$V = \frac{Y_t F [S_i - (1+\alpha) S_e] + \alpha X_R F}{k_d} - \frac{(1 + \alpha) F}{k_d}$

Where:

V	=	Volume of the reactor
S_i	=	Influent BOD_5
S_e	=	Effluent BOD_5
X	=	MLSS or MLVSS
F	=	Influent flow rate
X_R	=	Waste sludge SS or VSS
K_e	=	Eckenfelder's 1st order substrate removal rate constant
K_e'	=	Eckenfelder's 2nd order substrate removal rate constant
K_m	=	McKinney's substrate removal rate constant
Y_t	=	True cell yield (all models)

TABLE E-1 (Continued)

k_d = Maintenance energy coefficient (all models)

θ = Sludge retention time (mean cell retention time)

α = Recycle flow rate

SOURCE: Kincannon, 1979.

TABLE E-2

MATERIALS BALANCE FOR SUBSTRATE, S

Balance Model	Mass Rate of Change		Mass Rate due to Inflow		Mass Rate due to Outflow		Mass Rate due to Metabolism
Eckenfelder	$\frac{dS}{dt}.V$	=	$F.S_i$	-	$F.S_e$	-	$K_e X.S_e.V$
McKinney	$\frac{dS}{dt}.V$	=	$F.S_i$	-	$F.S_e$	-	$K_m S_e.V$
Lawrence-McCarty	$\frac{dS}{dt}.V$	=	$F.S_i$	-	$F.S_e$	-	$K.X.\frac{S_e}{K_s+S_e}.V$
Gaudy	$\frac{dS}{dt}.V$	=	$F.S_i+\alpha FS_e$	$-F(1+\alpha)S_e$		-	$\mu_{max}\frac{X}{Y_t}.\frac{S_e}{K_{s+}S_e}.V$

Where:

V	=	Volume of the reactor
S_i	=	Influent BOD_5
S_e	=	Effluent BOD_5
X	=	MLSS or MLVSS
F	=	Influent flow rate
F_w	=	Solids wastage flow rate
K_e	=	Eckenfelder's 1st order substrate removal rate constant
K_m	=	McKinney's substrate removal rate constant
K	=	Maximum substrate utilization rate (Lawrence and McCarty)
K_s	=	Saturation constant (Lawrence and McCarty, Gaudy)
μ_{max}	=	Maximum specific growth rate (Gaudy)
Y_t	=	True cell yield (all models)
α	=	Recycle flow rate

SOURCE: Kincannon, 1979.

Based on an comparison of these four models by Kincannon and Gaudy (no date) and Kincannon (1979), certain relationships are held in common in each of the mass balance equations:

1. The mass rate of change of substrate in the reactor is equal to the rate of change in concentration of substrate (dS/dt) multiplied by the volume of the reactor (V).
2. The rate of change of substrate concentration is increased by the inflow of the substrate.
3. The rate of change of substrate concentration is decreased by the flow of substrate concentration out of the reactor, and by the rate at which substrate is utilized for growth of the microorganisms in the reactor.

Eckenfelder's and McKinney's Models

Eckenfelder's and McKinney's models are developed assuming that the rate of substrate removal is a first order reaction. The models differ in the relationship used to describe the substrate utilization rate. In McKinney's model, this term is dependent only upon the substrate concentration in the reactor (S_e) and a substrate removal rate constant (K_m^1). The rate of substrate utilization in Eckenfelder's model is dependent upon the substrate concentration (S_e), the concentration of biomass (X), and a removal rate constant (K_e).

Lawrence and McCarty Model

The Lawrence and McCarty model is developed according to a second approach to biological modeling--using Monod kinetics, which is based on the Michaelis-Menten hypothesis for rates of enzyme-catalyzed reactions. In Lawrence and McCarty's model, the rate of substrate utilization and removal is related to substrate concentration (S_e), biomass concentration (K_m), and two constants, K and K_s. K is defined as the maximum specific substrate utilization rate, and K_s is termed the saturation constant.

The Lawrence and McCarty model is distinct in that the primary system parameter to control treatment plant design is the mean cell residence time (θ_c), a parameter that is mathe-matically equivalent to the reciprocal of the net microbial growth rate (μ_n). Mathematically, the mean cell residence time is defined as:

$$\theta_c = \frac{VX}{F_W X_R + (F - F_W) X_e}$$

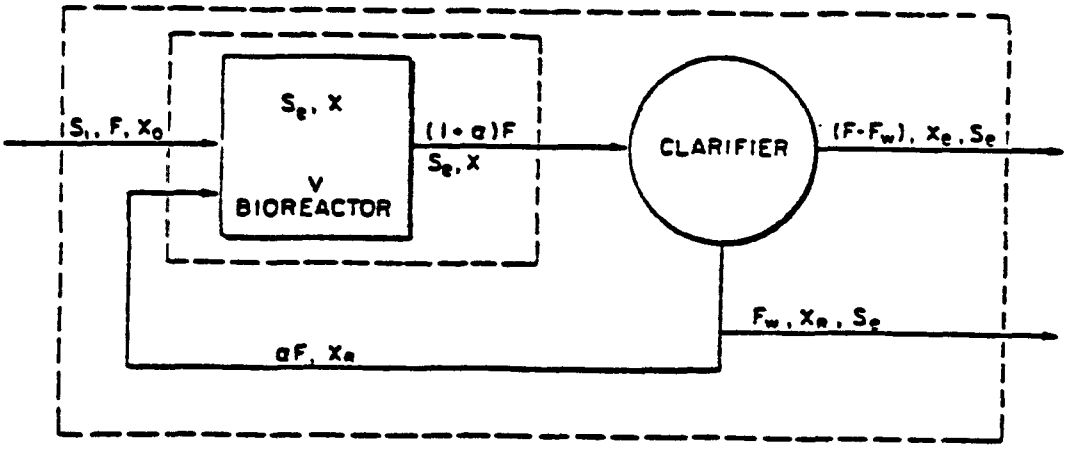
In the above equation, F and F_w represent the influent flow rate and the solids wastage flow rate, respectively; X_e and X_R represent the biological solids concentrations in the aeration tank and in the clarifier effluent, respectively.

Gaudy's Model

Gaudy's model, also based on Monod kinetics, differs from the above three as a result of writing the mass balance around the bioreactor rather than around the whole activated sludge process. This approach separates the biological unit process of biological growth and substrate utilization occurring in the activated sludge tank from the unit operation of physical separation accomplished in the clarifier (see Figure E-2). As a consequence of writing the balance around the bioreactor, the effect of α (a factor related to solids recycling) on the substrate balance is noted. The mass rate of substrate utilization is related to the growth of the biomass and the biomass "constants", i.e., the maximum specific growth rate (μmax), the saturation constant (K_s), and the "true" cell yield (Y_t).

FIGURE E-2

FLOW DIAGRAM, ACTIVATED SLUDGE PROCESS SHOWING NOTATION AND MASS BALANCE ENVELOPES



SOURCE: Kincannon, 1979.

Data Development for Biokinetic Models

One feature common to all of the kinetic approaches described above is the inclusion of biokinetic constants, or K-rates. Reliable biokinetic constants, values determined empirically in wastewater treatability studies, are necessary if the design models are to have an accurate predictive application.

In the Catalytic modeling approach, kinetic formulas are used to calculate removal of organic matter by the activated sludge process. As Catalytic's approach involved the determination of rate coefficients for each product/process waste load, two types of treatability data were required:

1. Reaction rate constants for BOD, and
2. K-rates specific to the pollutant regardless of its product/process source.

The individual process stream data identified during field sampling were used to calculate a weighted-average reaction rate for the summation of the BOD load from the

contributing product/processes. Thus, the activated sludge process design was based on that weighted-average BOD reaction rate (K-rate) that describes the total plant waste. For the development of pollutant-specific K-rates, treatability data requirements were two fold: the biological reaction rate (K-factor), and the lowest attainable concentration.[1]

Based on data obtained from OCB's screening and verification sampling and analysis program, from EPA's 308 questionnaires, and from Catalytic's laboratory test results, treatability factors for individual pollutants were calculated using the following equation:

$$K = \frac{S_o^2 - S_o S_e}{TXS_e}$$

where:

K = Treatability factor for any priority pollutant (day^{-1})

S_o = Influent concentration (mg/l) of the priority pollutant

S_e = Effluent concentration (mg/l) of the priority pollutant

X = Mixed liquor volatile suspended solids (mg/l)

T = Basin detention time (days).

---

[1]For a more complete discussion of the development of the treatability factors used in the Catalytic computer model, see the 1980 Catalytic Report. The treatability data developed by Catalytic during the 1980 analysis of the applicability of activated sludge appear in the Parameter and Treatment Selection file (Catalytic, 1980).

The minimum attainable value for a priority pollutant in the treated effluent was considered to be the lowest value observed in an effluent for which data was considered acceptable. Of the 115 organic priority pollutants, biological treatability factors for 86 pollutants were established in this manner.

For 20 other priority pollutants, the biological treatability factor was estimated by using Eckenfelder's Modified (zero-order) Equation:

$$KS_e = \frac{S_o(S_o - S_e)}{Xt}$$

where:

S_o = Influent concentration, mg/liter

S_e = Effluent concentration, mg/liter

X = Mixed liquor volatile suspended solids concentration (MLVSS), mg/liter

t = Hydraulic retention time, days

K = Reaction rate constant, day^{-1}.

However, there were several areas where information was needed to support the computer modeling effort relative to biological treatment. Specifically, more data were needed on the relative treatability of certain compounds, compound groups, priority pollutants and process raw waste loads. More work was needed relative to finding and supporting the best predictive mathematical process model. As such, a main objective of the Catalytic work was to refine or continue to verify the method of combining kinetic factors of components of a mix to determine the overall treatability of the wastewater. To have run laboratory studies in order to determine biokinetic constants for every possible combination of organic chemicals would have been prohibitively expensive and time consuming. However, the determination of biological constants for wastes containing single compounds was feasible; and from this data constants could be combined to give

a single constant for wastes containing a mix of organic compounds.[1] Grau-type K values as high as 20 day^{-1} have been encountered and values between 2 and 10 day^{-1} have been determined for other chemicals. There is, however, a lower range of K's for which data is lacking. Systems utilizing chemicals with low K values are the most difficult to operate because upsets are easier to provoke and the slowly degradable organic chemicals often present handling problems.

Methodology Development

To address the problem of an inadequate data base, Catalytic, Inc., under contract to Effluent Guidelines Division's Organic Chemical Branch has developed a methodology for bench-scale biological studies to evaluate treatability factors (K-factors) of selected organic compounds. The treatability of various classes of organic chemicals had previously been grouped through the use of literature surveys according to degradability.[2] As such, five general classes of degradability were established, as shown in Table E-3

TABLE E-3

CLASSES OF DEGRADABILITY

CLASS	K RATE	
I.	Highly degradable	20
II.	Easily degradable	10
III.	Moderately degradable	2
IV.	Slowly degradable	0.5
V.	Biostatic or biotoxic	0

[1]The best method of combining K values (e.g., straight average, rate limiting K, weighted average, etc.) has not yet been deter-mined. Presently, the average of component K factors is being used.

[2]Chemicals were classified by existing degradation data or by structural analysis.

Initial work to gain more precise information on specific treatability factors and on combining K-rates was performed by Catalytic from 1976 to 1978 (Catalytic, 1979b). In these initial experimental systems, the chemical or chemicals of interest were used as the sole source of carbon for the bacteria. This type of laboratory operation represents an artificial situation which is not likely to occur in a full scale chemical plant. Also, because of the very specific biological population which develops under these conditions, operational problems were encountered.

Thus, a new laboratory methodology was devised as part of a Phase 2 study to overcome some of these operational problems. The new systems used sludge age as a variable, since this parameter is required to evaluate all the current biological models. Another significant change from the earlier work was that a portion of the feed for each system was made up of a mixture of readily biodegradable organic compounds, in addition to the chemical of interest. This "base mix" was composed of ethylene glycol, ethyl alcohol, glucose, glutamic acid, acetic acid, phenol, and nutrients (ammonium sulfate, phosphoric acid and salts) as required. In addition to stabilizing the bench scale system, the impact of non-biodegradable and/or slightly biodegradable compounds on biokinetic rates could then be evaluated.

The kinetic equation used in the Catalytic system is the Grau model (or Eckenfelder's second order equation). This approach has enjoyed acceptance by industry and allows more flexibility in predicting effluent quality under varying influent concentrations, which typically occur in industrial biological treatment systems, than does a first order equation. Other kinetic models such as those utilizing solids retention time (SRT) as a primary variable (which includes the Lawrence and McCarty models and the Gaudy model), were considered less extensively verified. The Grau equation is shown below:

$$KS_e = \frac{S_o(S_o - S_e)}{Xt}$$

where:

K = kinetic constant

S_o = BOD influent concentration (mg/liter)

S_e = BOD effluent concentration (mg/liter)

X = MLVSS (mg/liter)

t = Aeration time (days)

Gaudy (1980) has pointed out some limitations of the Grau model; it is not a good mechanistic relationship because it combines at least four separate biokinetic constants known to be determining factors in characterizing the behavior of activated sludge into one "constant", and combines separate engineering control parameters which independently affect S_e. Further, operating conditions such as net specific growth rate which can affect the performance of an activated sludge system, are not built into this model for assessing removal of priority pollutants. However, Gaudy also noted that industrial information expressed in terms of the Grau model was more readily available, and that the model would prove useful for estimating effluent guidelines relating to biological treatment for a broad group of combinations of compounds. He has recommended the gathering of kinetic data applicable to the testing of a variety of models in order to verify or modify values used in the Catalytic computer model; such research is presently in progress at Oklahoma State University (see the following section).

To obtain a K value for a specific chemical based on the Grau model, Catalytic ran several bench-scale systems to obtain different effluent BODs. The values of $\frac{S_o(S_o - S_e)}{Xt}$ were plotted versus the effluent BOD values; the slope of the resultant line equaled the K value for the chemical in question.

From Catalytic's Phase 2 experimental work came kinetic information on 15 compounds, tested with "base mix" only and in combination, and run at various loadings. Compounds selected for study represented known raw materials, products and by-products expected in industrial effluents, groups of chemical compounds for which little biotreatability information was available, and priority pollutants where possible. Other primary considerations in selecting compounds for study included solubility, volatility, chemical stability in water, toxicity, carcinogenicity, odor, flammability, chemical compatibility with the other chemicals in a mix, availability, and cost. See Table E-4 for a list of compounds studied according to the Catalytic methodology. Results of this experimental work are present in the Catalytic files (Catalytic, 1979b).

Oklahoma State University Studies

The methodology for determination of biokinetic constants in activated sludge models developed by OCB and their contractor, Catalytic is also being applied by researchers at Oklahoma State University. An EPA-sponsored study by Kincannon is now in progress with objectives consistent with those of the Catalytic study:

1. To determine biokinetic constants for wastewaters containing 24 major organic compounds
2. To determine a method for combining biological constants for evaluating complex waste streams.

Biokinetic constants are being determined for design models developed by Eckenfelder, McKinney, Lawrence and McCarty, and Gaudy. To develop a methodology for combining K-rates, three compounds are run individually and in combination, with pilot plants operated at three different sludge ages for each compound or combination. In addition, specific compounds in the off gases from the pilot plants are being measured to determine the strippability of volatile organic compounds. Preliminary results for four sets of priority pollutants (three pollutants run individually and in combination) have been

TABLE E-4

ORGANIC COMPOUNDS STUDIED IN
CATALYTIC'S PHASE II BENCH-SCALE BIOLOGICAL SYSTEM

N-Butyl Phthalate (nBP) 75%, Base Mix* 25%

O-Nitrophenol (ONP)** 75%, Base Mix 25%

Maleic Acid 75%, Base Mix 25%

Acetonitrile 75%, Base Mix 25%

Acetonitrile 25%, ONP 25%, Maleic Acid 25%, Base Mix 25%

Base Mix (with and without phenol** in the mix)

Butyl Acetate 75%, Base Mix 25%

Methyl Cellulose 75%, Base Mix 25%

Methyl Formate 75%, Base Mix 25%

Melamine 75%, Base Mix 25%

Catechol 75%, Base Mix 25%

Formamide 75%, Base Mix 25%

Ethanol 75%, Base Mix 25%

Ethanol (denatured) 75%, Base Mix 25%

Isophorone ** 75%, Base Mix 25%

Ethylene Dichloride** 75%, Base Mix 25%

2-Naphthol-3,6-disulfonic Acid 75%, Base Mix 25%

1-Phenyl-2 thiourea 75%, Base Mix 25%

Base Mix without Glutamic Acid 100%

Base Mix without Ethanol 100%

Base Mix without Glucose 100%

Methyl Formate 25%, Formamide 25%, Maleic Acid 25%, Base Mix 25%

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*Base mix is composed of ethylene glycol, ethyl alcohol, glucose, acid, acetic acid, and phenol.

**Priority pollutant.

reported in four quarterly reports (Kincannon, 1980a and 1980b, and Kincannon and Stover, 1981a and 1981b). See Table E-5 for the list of compounds studied.

Effect of Priority Pollutants on Biological Treatment

The optimal operation of an activated sludge process is subject to change as a result of a number of environmental conditions, including the presence of compounds toxic to the system's microbial population. These several conditions acting simultaneously on the biological system make it difficult to determine whether a particular chemical, or a combination of toxic compounds, is responsible for an observed malfunction of a waste treatment process. Such a data base is essential to the development of pre-treatment requirements for industrial wastes containing priority pollutants to treatment works employing the activated sludge process.

The effect of 24 priority pollutants on the performance of batch and continuous flow bench-scale activated sludge pilot plants was studied by Gaudy et al. (1979). Additionally, eight of these compounds were studied in continuous flow pilot plants operated at a net specific growth rate (μ_n) of 0.2^{-1} (θ_c = 5 days); four of the eight were also studied in extended aeration pilot plants. See Table 3-9 for a list of those priority pollutants tested. Each test compound was added to the feed at dosage levels that increased from 5 mg/liter to 20 or 25 mg/liter to 50 mg/liter. Following a period of operation at a steady dosage level of 50 mg/liter, the unit was subjected to daily cycling or pulsing of the concentration of test compound (e.g., 25 to 0 to 25 mg/liter).

The following conclusions with respect to the effects of the 24 priority pollutants on the pilot plant performance were drawn after a two-year experimental period. Results are also summarized in Table E-6 . An evaluation of plant performance was based on a comparison of the residual soluble COD and effluent suspended solids in the test unit and the control unit.

TABLE E-5

PRIORITY POLLUTANTS STUDIED
IN OSU'S BENCH-SCALE BIOLOGICAL SYSTEM

Compounds Studied (with Percent Total COD)[1]

Set 1: Individual Feed
1,1,2,2-Tetrachloroethane (TCE) 9%
Nitrobenzene (NB) 33%
2,4-Dichlorophenol (DCP) 21%

Combined Feed
TCE 2%, NB 14%, DCP 75%

Set 2: Individual Feed
Acrolein (Ac) 66%
Acrylonitrile (Acry) 66%
1,2-Dichloropropane (DCP) 2%

Combined Feed
Ac 22%, Acry 22%, DCP 1%

Set 3: Individual Feed
Methylene Chloride (MC) 5%
Benzene (BEN) 67%
Ethyl acetate (EA) 67%

Combined Feed
MC 2%, BEN 22%, EA 32%

Set 4: Individual Feed
1,2-Dichloroethane (DCE) 13%
Phenol (Ph) 85%
1,2-Dichlorobenzene (DCB) 33%

Combined Feed
DEC 3%, Ph 34%, DCB 3%

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[1]The remaining COD was contributed by "base mix," a readily biodegradable synthetic wastewater.

SOURCE: Kincannon, 1980a and 1980b, and Kincannon and Stover, 1981a and 1981b.

TABLE E-6

SUMMARY OF EFFECTS OF PRIORITY POLLUTANTS OSU BENCH-SCALE BIOLOGICAL SYSTEM

COMPOUND[1]	PARAMETER	BATCH UNIT DOSAGE (mg/l)					CONTINUOUS FLOW UNIT: $\mu n = 0.2^{-1}$ DOSAGE (mg/liter)					CONTINUOUS FLOW UNIT: EXTENDED AERATION DOSAGE (mg/liter)				
		0	5	20/25	50	CYCLIC	0	5	20/25	50	CYCLIC	0	5	20/25	50	CYCLIC
PENTACHLOROPHENOL	CODE	O	X	X	X	X										
	SSE															
1,2-DICHLOROETHANE	CODE	O	O	O	O		O	O	O	O						
	SSE						O	O	X	X						
NITROBENZENE	CODE	O	O	O	O	O	O	O	O	O	O					
	SSE						O	O	O	O	X					
TRICHLOROETHYLENE	CODE	O	O	O	O	O	O	O	O	O	X					
	SSE						O	O	O	O	X					
METHYLENE CHLORIDE	CODE	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
	SSE						O	X	X	X	X	O	O	O	O	O
PHENOL	CODE	O	O	O	O	O	O	O	X	X	X	O	X	X	X	X
	SSE						O	X	X	X	X	O	O	O	X	X
2-CHLOROPHENOL	CODE	O	X	X	X	O	O	O	O	O	O	O	O	O	O	O
	SSE						O	O	O	X	X	O	O	O	O	O
4-CHLORO-3-METHYL PHENOL	CODE	O	O	X	X		O	O	O	?		O	O	O	?	
	SSE						O	O	O	O		O	O	O	O	

O = Performance same as control
X = Performance poor compared with control
? = Performance poor in both control and test system
Blank indicates parameter not measured

[1]The following priority pollutants, tested in the batch unit, did not affect performance:

Anthracene	Hexachlorobenzene
Benzene[a]	Hexachloroethane
Bromoform	Naphthalene
Carbon Tetrachloride	2-Nitrophenol
Chlorobenzene	Tetrachloroethane
Chloroform	Tetrachloroethylene
Ethylbenzene	1,1,2-Trichloroethane
Fluorene	Toluene

[a]Also tested in continuous flow unit ($\mu n = 0.2\ day^{-1}$)

SOURCE: Gaudy et al., 1979

1. Of the 24 compounds studied in batch systems, only two compounds (pentachlorophenol and 2-chlorophenol) gave evidence of causing metabolic stress to the system, as judged by comparison of residual soluble COD in the test unit and control unit at a daily feed concentration of 5 mg/l. At higher concentrations (20 to 25 and 50 mg/l), there was evidence of metabolic disturbance for only one additional compound, 4-chloro-3-methylphenol.

2. For the eight compounds tested in the continuous-flow activated sludge pilot plant operated at $\mu_n = 0.2$ days^{-1}, there was no evidence of increased soluble COD in the effluent at the 5 mg/l dose. At this dose, however, there was an increase in suspended solids in the effluent of pilot plants dosed with phenol and methylene chloride. In addition, at higher dosage levels, there was an increase in soluble COD and suspended solids in the effluent for the pilot plant dosed with phenol. For the units dosed with 2-chlorophenol at the 50 mg/l dose, methylene chloride at the 5 mg/l dose and dichloroethane at the 25 mg/l dose, soluble COD in the effluent was not affected but there was an increase in effluent suspended solids concentration. Under alternating concentration levels (daily changes from 25 to 50 to 25 mg/l followed by 0 to 25 to 0 mg/l) there was increased soluble COD and suspended solids in the effluent for the unit dosed with trichloroethylene. For the pilot plants dosed with nitrobenzene, 2-chloro-phenol, methylene chloride and 1,2-dichloroethane, cyclic loading led to increased suspended solids in the effluent.

3. For the four compounds tested in the extended aeration pilot plant, increased soluble COD in the effluent was reported only for the unit dosed with phenol at the 5 mg/l dosage level. There was no increase in effluent suspended solids in any of the four systems at this dosage level. At the higher dose levels (20 and 50 mg/l) and during the period of cyclic loading of the test compound, the units dosed with phenol showed increased soluble COD and suspended solids.

FATE OF PRIORITY POLLUTANTS

Introduction

In developing predictive models for biological treatment of specific organic compounds found in industrial effluents, an important distinction needs to be made between treatability and removability of the specific compounds. Removability describes the change in concentration of the compound between entering and leaving the treatment process. Treatability is a more specific term, relating to the treatment mechanism by which compounds can be evaluated (biodegradation, air stripping of volatile compounds, adsorption on sludge, etc.) and compared to other compounds (Kincannon et al., 1981).

To date, most design models have originated from a simplified mass balance for the substrate, represented as:

CHANGE OF MASS IN REACTOR	=	MASS ENTERING REACTOR	-	MASS LEAVING REACTORIN EFFLUENT	-	MASS CONSUMED BIOLOGICALLY

As adsorption and stripping are not included in this mass balance equation, treatability data derived on this basis may:

1. Incorrectly determine biokinetic constants (and thus inaccurately predict substrate treatability by giving biological processes credit for removal).
2. Leave unrecognized air and solid waste pollution problems, resulting from stripping and adsorption of the various organic compounds.

A more correct substrate mass balance would thus be (Kincannon and Stover, 1981):

CHANGE OF MASS INRE-ACTOR	=	MASS ENTERING REACTOR IN EFFLUENT	-	MASS LEAVING REACTOR	-	MASS STRIPPED	-	MASS ADSORBED ON SLUDGE	-	MASS CONSUMED BIOLOGICA

This approach, that is considering the multiple pathways by which pollutants can be removed during biological treatment, is used in the following section.

Multimedia Models Of Biological Treatability

Hwang Model

Hwang (1980a) has developed a model describing the dynamics of substrate removal in a continuous activated sludge process with sludge recycle, incorporating the mechanisms of biodegradation, air stripping, and adsorption on sludge. The components of Hwang's model are described briefly below.

Several models describing the kinetics of biological degradation were evaluated in the development of the Hwang Model, including those using first order kinetics for the substrate reaction (e.g., Eckenfelder and McKinney), those which apply Monod kinetics in their material balance formulations (e.g., Lawrence and McCarty, and Gaudy), and the Grau model (which treats the substrate removal rate as a function of the remaining substrate concentration as compared to the original concentration). Experimental data from several sources[1] served as the basis for judging the suitability of each of the treatability models to predict removal of the specific toxic compounds from waste streams. The Grau method best described the data and, as a result, is used in the Hwang multimedia model to describe the biodegradation of priority pollutants; this model is represented by:

$$-\frac{dS}{dt} = k_{n(s)} \left[\frac{S}{S_o}\right]^n$$

where:

S = substrate concentration, g/liter

t = time, days

[1]Data were obtained from the following sources: EPA, Cincinnati, Union Carbide Corporation, Catalytic, Inc., and the literature.

$k_{n(s)}$ = rate constant in the Grau equation, l/day when n=l

S_o = initial substrate concentration, µg/liter.

To describe air stripping kinetics in the Hwang model, experimental data on air stripping rate constants are used where available. The expression for air stripping of volatile components is represented as:

$$\frac{dS}{dt} = k_a S$$

where:

k_a = air stripping rate constant, l/day

S = substrate concentration in the liquid, µg/liter.

Where experimental data on air stripping rate constants are not available, rates can be determined by combining the individual liquid and gas phase mass-transfer constants as follows:

$$1/k_a = 1/k_{aL} + 1/Kk_{aG}$$

where:

k_a = air stripping rate constant, l/day

k_{aL} = individual liquid mass-transfer constant

k_{aG} = individual gas mass-transfer constant

K = equilibrium distribution coefficient.

Adsorption on activated sludge provides an additional route of removal for some organic pollutants, cyanides, and metals. Adsorbed toxic compounds are removed from the biological system when the activated sludge is wasted. In Hwang's model, removal of a specific toxic pollutant in sludge is represented by the multicomponent Langmuir type

adsorption equation, which is based on the concept of binary characterization of wastewater adsorption:

$$[B \cdot S] = \frac{K_1 \; X' \; S}{1 + K_1 \; S + K_T \; S_r}$$

where:

[B·S] = concentration of a substrate in sludge, μg/liter

S = substrate concentration in the liquid, μg/liter

S_r = concentration of total substrates minus substrate S under consideration, μg/liter

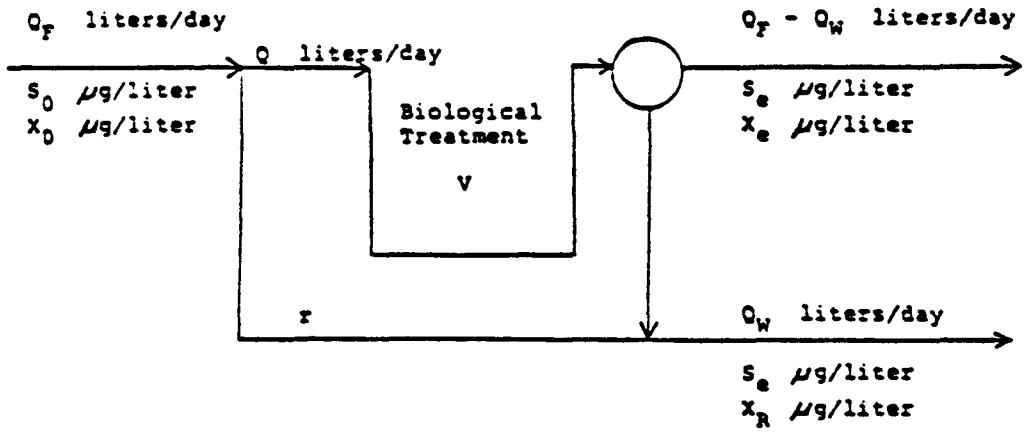
K_1, K_T = adsorption constants

X' = the maximum amount of the substrate adsorbed on sludge, μg/liter.

Hwang's unified model (see Figure E-3) for removal of toxic chemicals by multimedia pathways incorporates the above three mechanisms. The model is described below in terms of the schematic of the activated sludge process and by the equations which follow.

FIGURE E-3

A SCHEMATIC OF THE ACTIVATED SLUDGE PROCESS



SOURCE: Hwang, 1980.

S_o = substrate concentration in influent, μ g/liter

S_e = substrate concentration in effluent, μ g/liter

X_o, X_e, X_R = concentration of substrate on sludge in influent, effluent, and return sludge, respectively, μ g/liter

V = bioreactor volume, liter

r = recycle ratio, volume flow rate recycle/volume flow rate feed

Q = flow rate to biological treatment (=(1+r)Q_F), liter/day

Q_F = flow rate of the fresh feed

Q_W = flow rate of the waste sludge.

$$Q_F S_o - (Q_F - Q_W) S_e = \frac{k_{1(s)} X S_e}{S_o} V + k_a S_e V + Q_W \frac{K_1 X' S_e}{1 + K_T S_r + K_1 S_e}$$

or

$$S_o - (1 - \frac{Q_W}{Q}) S_e = \frac{K_{1(s)} X S_e t_c}{S_o} + k_a S_e t_c + \frac{K_1 X' S_e}{1 + K_T S_r + K_1 S_e} \frac{Q_w}{Q}$$

where:

t_c = hydraulic residence time, day

X = sludge concentration in the reactor, μ g/liter

X' = maximum concentration of a substrate on sludge, μg/liter

$k_{1(s)}$ = rate constant in the Grau equation, 1/day when n=1

k_a = air stripping rate constant, 1/day

K_1 and K_T = adsorption constants

S_r = concentration of total substrates minus substrate S under consideration, μ g/liter.

Using the relationship $Q_F = \frac{Q}{1+r}$, one gets

$$S_o - (1 - W(1+r))S_e = \frac{k_1 X S_e}{S_o}(1+r)t_c + k_a S_e(1+r)t_c + (1+r)_W \frac{k_1 X' S_e}{1 + K_T S_r + K_1 S_e}$$

where $W=Q_W/Q_F$ (waste sludge flow/fresh feed flow).

Since Hwang found the W(1+r) term to be small, the equation may be reduced to:

$$(S_o - S_e) = \frac{k_{1(s)} X S_e}{S_o}(1+r)t_c + k_a(1+r)S_e t_c + (1+r)_W \frac{k_1 X' S_e}{1 + K_T S_r + K_1 S_e}$$

Monsanto Model

In addition to the Hwang model, a number of other models have been developed to describe the removal of organic compounds by multiple pathways during biological treatment. Monsanto Company has described an approach for use in predicting the rate of air stripping and biological degradation (Freeman, 1979). Mathematical predictions based on the Monsanto model for the treatability of the priority pollutant acrylonitrile were verified using an experimental activated sludge system (Freeman et al., 1980).

In the Monsanto model, the biological oxidation component is represented by the model of Gerber. This model involves the solution of the following three simultaneous non-linear equations in three unknowns; solution by use of a digital computer is recommended by the authors. Use of the Gerber model to describe biological treatability

differs from the one constant Grau model used by Hwang; further, the Gerber model accounts for the impact of substrate, oxygen, and biota concentrations.

$$r_{O_2} = \frac{k_5 B_o C_o O_o}{\frac{k_5}{k_1} O_o + K_{O_2} K_s + C_o O_o + K_{O_2} C_o}$$

$$r_B = t r_{O_2} \frac{Mw_B}{Mw_{O_2}}$$

$$r_A = \frac{t}{s} r_{O_2} \frac{Mw_A}{Mw_{O_2}}$$

where:

r_{O_2} = Rate of oxygen use, lb/hr-ft^3,

r_B = Rate of microorganism growth, lb/hr-ft^3,

r_A = Rate of organic disappearance, lb/hr-ft^3,

B_o = Concentration of microorganisms from the basin, lb/ft^3,

C_o = Concentration of organics from the basin, lb/ft^3,

O_o = Oxygen concentration in the basin liquid, lb/ft^3,

k_1 and k_5 = Reaction rate constants,

K_{O_2} and K_s = Constants,

t = Oxygen use factor, lb mole $C_5H_7NO_2$ produced/lb mole oxygen used,

S = Substrate use factor, lb mole $C_5H_7NO_2$ produced/lb mole substrate consumed, and

MW_A, MW_B, = molecular weights of the organic compound, micro
and MW_{O_2} organisms, and oxygen, respectively, lb/lb mole.

The rate of air stripping in the model is represented by the following equation, in which the rate of stripping to the atmosphere varies with the organic concentration in the liquid phase.

$$N_a = \pi \frac{D^2}{4} N K_a^T (X_a - X_a^*) + (A_s - \frac{\pi D^2 N}{4}) K_a^c (X_a - X_a^*)) \quad Mw_a$$

where:

N_a = Loss of organic to the atmosphere, lb/hr,

D = Diameter of region of effect of an aerator for mass transfer, ft,

N = Number of aerators in basin,

K_a^T and K_a^c = Overall mass transfer coefficients for the turbulent and convective regions, respectively, lb mole/hr ft^2,

X_a = Mole fraction of organic in liquid,

X_a^* = Mole fraction of organic in liquid at equilibrium,

A_s = Total surface area of basin, ft^2, and

M_{w_a} = Molecular weight of organic compound, lb/lb mole.

Limitations exist in the application of this approach, however; extensive kinetic data is necessary for the biological degradation component (Gerber model) since several biokinetic constants are used to describe the behavior of the activated sludge process rather than the one combined constant used in the Grau model. Also, the adsorption of organic components onto the surface of wasted solids and the subsequent removal in the sludge is ignored.

The treatability of acrylonitrile was studied to verify the predictability of this model. The model indicated that if sufficient aeration capacity is maintained (2 ppm or above), 99 percent of the feed acrylonitrile will be biodegraded and less than 1 percent will be air stripped (Freeman, 1979). These results were confirmed in series of bench-scale continuous flow activated sludge treatment systems, in which biological treatability efficiencies of greater than 99.9 percent were found (Freeman et al., 1980). When the treatment system was run under sterile conditions (i.e., no seed microorganisms were present), it was found that 18 percent of the acrylonitrile was air stripped.

Data Development for Multimedia Models

Indicatory Fate Study

The data base for predicting the fate of individual compounds by multiple routes is presently limited. One of the first attempts to determine the fate of specific priority pollutants as they pass through a biological system was conducted in the EPA-sponsored Indicatory Fate Study (EPA, 1979a). Three plants belonging to the organics and plastics industries participated in a screening study to provide an indication of the removal of specific priority pollutants via air, water, or residuals routes. The types of treatment processes used to treat the industrial effluents are shown in Appendix G. Analyses were conducted for priority pollutants which had been identified in previous screening and verification sampling under the direction of EPA's Effluent Guidelines Division, Organic Chemical Branch. Analyses were performed on composite samples (influent, effluent, air, and residuals) and on grab samples (influent and effluent). The data generated from these analyses provide only an indication of the route of removal of specific priority pollutants, and were not intended to represent a mass balance across a biological treatment system. Analysis for organic compounds in the three plants sampled revealed no discernible patterns concerning the fate of specific organic compounds or classes of compounds in the effluent, sludge/sediment, or air. Heavy metals occurred at the highest concentrations in

all three plants in the activated return sludge or the sediment of aerated lagoons. The heavy metals included arsenic, copper, chromium, nickel, zinc, and lead in return sludge, and additionally selenium, cadmium, beryllium, antimony, silver, and thallium in lagoon sediments.

EPA-Sponsored Bench-Scale Studies

Kincannon *et al.* (1981) and Kincannon and Stover (1981c), in work supported in part by the EPA, have evaluated the removal of priority pollutants by biodegradation and stripping. Experimental results were obtained using a bench scale-continuous flow activated sludge reactor used to treat a synthetic wastewater containing selected priority pollutants. The reactor was operated as a nonbiological system to determine the strippability of the chemical compound, and as a biological activated sludge system to determine biodegradation and stripping, and adsorption for a limited number of compounds. These results are summarized in Table E-7.

Total percent removal of the specific compounds varied from 93 to 99.9 percent. Stripping during biological treatment accounted for essentially all of the tetrachloroethane, 1,2-dichloropropane, and 1,2-dichloroethane removed. These studies show that volatile organic compounds can be removed by concurrent stripping and biological oxidation. Further, the results indicate that the failure to recognize stripping as a removal mechanism will affect the experimentally derived biokinetic constants for pollutants.

It is interesting to note that the stripping that takes place in a nonbiological system does not necessarily predict the degree of stripping in a biological system. While approximately 100 percent of 1,2-dichloropropane, methylene chloride, benzene, and 1,2-dichlorobenzene are stripped in nonbiological systems, only 1,2-dichloropropane is highly stripped during treatment with the biological systems.

TABLE E-7

FATE OF SPECIFIC POLLUTANTS

Compound	Biological System[a]				Nonbiological System[b]
	Total Percent Removed	Percent Stripped	Percent Adsorbed	Percent Degraded	Percent Stripped
Tetrachloro-ethane	93	93	-	0	-
Nitrobenzene	97	0	-	97.0	-
2,4-Dichloro-phenol	94	0	-	94.0	-
Acrolein	99.9	0	-	99.9	-
Acrylonitrile	99.9	0	-	99.9	-
1,2-Dichloro-propane	99.9	99	-	0	98.8
Methylene Chloride	99.5	5	-	94.5	99.4
Ethyl Acetate	99.8	17	-	82.8	81.9
Benzene	99.9	15	-	84.9	99.3
1,2-Dichloro-ethane	98.5	97.5	1	0	96.1
Phenol	99.9	0	0	99.9	1.9
1,2-Dichloro-benzene	99.9	24	0	75.9	84.7

[a] SOURCE: Kincannon and Stover, 1981c.

[b] SOURCE: Kincannon *et al.*, 1981.

A limited evaluation of the fate of priority pollutants in batch and continous flow bench-scale activated sludge pilot plants was performed by Gaudy et al. (1979) in their study of the effects of 24 priority pollutants on the activated sludge process. ¼See Biological Treatability Studies--Toxicity of Priority Pollutants for a discussion of the test methods used.½ The results of these analyses appear in Appendix H. Although the data are limited, most compounds were removed effectively (with the exception of nitrobenzene, 2-chlorophenol and 3-methylphenol under certain experimental conditions). Anthracene and fluorene concentrations were high in the mixed liquor while concentrations were low in the settled effluent. This indicates that the compounds were present as part of the biological solids, possibly adsorbed to the surface. Although these analyses do not provide specific information on the removal routes of the priority pollutants, the results indicate there was no evidence for massive pass-through in the effluents of any of the compounds. Small quantities of some of the compounds were detected in the effluents, however; more detailed analytical procedures are needed to adequately address the question of pass-through of small concentrations of the priority pollutants.

Fate of Priority Pollutants in Publicly Owned Treatment Works

The fate of priority pollutants in publicly owned treatment works (POTW) was presented in an interim report, in which the preliminary results from 20 of the 40 POTW's selected by the EPA's Effluent Guideline Division were reported (Feiler, 1980). The treatment processes included among the 20 plants were conventional activated sludge and modifications of activated sludge such as contact stabilization, Kraus, and pure oxygen, as well as some advanced waste treatment processes, notably mixed media filters. Samples of influent, effluent, and sludge streams were analyzed for conventional, non-conventional, and priority pollutants. Based on their analyses of 93 priority pollutants,

the following preliminary findings relating to the fate of priority pollutants were reported:

1. For five of the treatment plants (four activated sludge and one trickling filter) where the mass balances were assumed to be relatively accurate it was observed that metallic priority pollutant mass balance was good, but some organic priority pollutants in the influent were always not accounted for in the effluent or sludges. This indicates that, in general, a portion of organic priority pollutants are biodegraded or, in the case of volatiles, stripped out of the wastewater.
2. Based on the 20 POTW data base, half of secondary treatment plants achieved at least 76 percent reduction of total priority pollutant metals, 85 percent reduction of total volatile priority pollutants, and 70 percent reduction of total acid-base-neutral priority pollutants. Tertiary treatment was slightly more effective than secondary treatment in reducing priority pollutants, and primary treatment the least effective.
3. For many conventional and priority pollutants, as influent concentrations increased, effluent concentrations also increased. This trend held for all metals (except for mercury) with correlation coefficients ranging from 0.204 to 0.995, and, in general, for volatile priority pollutants, correlation coefficients ranging from 0.262 to 0.937.
4. Some pollutants not measured in POTW influents were regularly measured at high levels in the corresponding sludge streams. This phenomenon was observed for 25 priority pollutants, including metals and organic compounds. This observation was most likely due to concentration of the pollutant in the sludge stream to detectable levels.
5. Eleven priority pollutant chlorinated hydrocarbons increased in concentration during chlorine disinfection.

Conclusions

The complexities of the removal of organic pollutants during biological treatment are receiving greater attention, both in terms of modeling and the design of experimental protocols to determine treatability constants for pollutants during biological treatment. The multimedia models reflect the recognition that biological treatment not only involves oxidation of organic compounds, but removal through air stripping and waste sludge as well. Failure to incorporate these additional routes of pollutant removal can result in inaccurate determination of biokinetic constants and failure to predict possible air and solid waste pollution problems. Thus, a model of the activated sludge process which incorporates a single biokinetic constant is not an accurate mechanistic model of the complete treatment process. Further experimental work is required in order to determine accurate kinetic constants for each of the competing treatability mechanisms that occur during biological treatment.

ACTIVATED CARBON ADSORPTION

Introduction

Adsorption processes can be used to remove contaminants from aqueous wastewaters by the preferential adsorption (either physical or chemical) of the contaminants on solid surfaces. Pollution parameters affected by activated carbon include BOD, COD, TOC, specific organic priority pollutants, and to a lesser degree four specific non-organics: cadmium, chromium, cyanide, and mercury.

There are approximately 100 large scale industrial or municipal wastewater activated carbon treatment systems in domestic use (Hydroscience, 1981). Large scale industrial activated carbon treatment systems require extensive pilot plant study before scaling up to optimize the system and assure compatibility between the system and the characteristics of the waste stream. This is due, in part, to the competition among individual components in a multicomponent stream for active adsorption sites. For example, the adsorptive capacity of a particular system with respect to a specific compound may be lessened if another compound is added to the waste stream. Additionally, system designers must consider the best economic configuration of the adsorption process itself and, if used in conjunction with other treatment modules (e.g., biological treatment), the optimal configuration of the entire treatment scheme. This is especially true for multicomponent wastestreams where target pollution parameters are of the utmost concern. Recent EPA research efforts have been directed toward modeling systems to assess the applicability of activated carbon treatment systems to differing waste stream types and to provide preliminary design data for scaling up.

Background

Activated carbon adsorption treatment systems may be implemented on a commercial scale in several differing design modes using either granulated or powdered carbon. The most common type of system in use will be discussed here. That system uses

granulated carbon in a fixed bed. The design criteria of a system are numerous: consideration must be given to the presence of suspended solids in the wastestream, the potential for biological growth in the adsorper, carbon regeneration costs, and cycling time. The most critical design parameter is the adsorptive capacity of the bed which dictates the performance of the system.

The effectiveness of granular carbon in removing a given pollutant from solution is typically evaluated in terms of its adsorption capacity at constant temperature. Adsorption capacity measures the amount of solute adsorbed per unit weight of adsorbent as a function of solute (pollutant) concentration in bulk solution.

The actual selection of the adsorber configuration is dependent on the required carbon dosage and contact time (flow rate/adsorber volume), which requires bench and/or pilot scale testing to determine the rate of adsorption. By dynamic column testing over a range of contact times, the concentration of solute (contaminant) remaining in solution (C/C_o) can be plotted versus volume of solution (wastewater) through the column to give the breakthrough curve. Depending on the complexity of the wastewater, the shape of the breakthrough curve may vary but, it is characteristically "S-shaped." The breakpoint represents the point on the curve at which the column is in equilibrium with the influent wastewater, and little additional removal of the contaminant(s) will occur. Generally, time to breakpoint may be extended by increasing the carbon bed depth and lowering the flow rate; however, several other factors, such as the characteristics and concentration of the solute, the pH of the solution, and the characteristics of the carbon selected influence the overall capacity of the adsorption system (i.e., height and rate of movement of the mass transfer zone, capacity of adsorbent, etc.). The objective, in any application, is to design a system in which the most economical, yet practicable, carbon exhaustion rates (the time necessary to reach the breakpoint) can be achieved.

Another major consideration in the design and operation of a carbon adsorption system is the means of replacing exhausted carbon. This may be accomplished by removing the carbon from the adsorber for permanent disposal (i.e., throwaway carbon), or more typically, for reactivation. Reactivation is any means by which the carbon is restored to its original adsorptive capacity. Organic impurities may be removed by thermal, alkaline, acid, hot gas (steam), solvent, or biological regeneration. However, in treating wastewaters containing a mixture of organics, thermal regeneration of the carbon in either multihearth or rotary tube furnaces provides the most reliable reactivation process and, thus, is the most widely applied. Thermal regeneration may be carried out on-site or off-site depending on the scale of operations. As a result of handling and reactivation, attrition losses, requiring fresh make-up carbon, will typically range from 5 to 15 percent by weight.

Under certain conditions (e.g., the presence of biodegradable organics, favorable pH ranges, etc.) biological activity may occur in the carbon adsorption column. In general, anaerobic growth not only results in H_2S production, but reduces the adsorption capacity of the column and therefore should be discouraged. Aerobic bacterial activity, depending on the concentration and composition of the waste loading, may enhance treatment efficiency. In certain cases, biological degradation of organic contaminants complements the adsorption process, increasing adsorption capacity and providing partial regeneration of the carbon.

State of the Art

As indicated above, the most critical design parameter for carbon adsorption treatment systems is the adsorption capacity of the carbon which determines the cycle time of the adsorption bed. The length of the adsorption cycle can be determined by two lab techniques and pilot scale tests. The lab scale tests are the use of isotherms and the Dynamic Mini-column Adsorption Technique (DMCAT).

Isotherms are usually determined under static conditions which assume the resistance to mass transfer to be neglible. EPA (1980) has adopted an alternate approach, DMCAT, that considers non-equilibrium effects to take into account the nature of the driving forces which control the transport phenomena of solutes from solution. Breakthrough curves for complex waste streams with nonlinear isotherms are usually empirically determined. Recent efforts have focused on modeling efforts for multicomponent wastestreams using psuedo-contaminant parameters which represent an empirical theoretical approach to the problem of optimizing dynamic adsorbent systems.

Data gathered through isotherm and DMCAT testing can be extended in some cases through the use of mathematic models which simulate the kinetics of adsorption in full scale systems. The lumped parameter model combines the diffusional resistances described by a pore diffusion model and homogeneous solid diffusion model into a single parameter. This model has been developed based on data from full scale operations and pilot plant data on several priority pollutants (EPA, 1980). Adsorption occurs through a four-step process:

1. Transport of a solute from bulk liquid to solid interface
2. Transport across the interface
3. Transport from the interface into the solids
4. Adsorption on the active sites.

Each one of these steps represents a resistance to mass transfer from the bulk liquid to the active sites. Equilibrium models assume the resistance to mass transfer due to steps 1, 2 and 3 to be neglible and that the bulk concentrations of the two phases are in equilibrium which is unrealistic (i.e., equilibrium is not attained in practical systems).

The lumped parameter model is developed by a fundamental material balance for each step in the adsorption process. The mass transfer equations that result from such an analysis are solved numerically and combined into a single mass transfer coefficient. A

brief treatment of this concept is presented below, the reader is referred to Appendices I and J and to EPA (1979b) for a detailed discussion of the lumped parameter model.

In order to utilize the principles of any model it is necessary to obtain experimental data to estimate mass transfer parameters. This requires dynamic column testing to generate breakthrough curves which, when done in a pilot plant scale column, is a timely and costly procedure. This is particularly true of requisite data on specific organic compounds. The Dynamic Mini-column Adsorption Technique (DMCAT) is capable of rapidly generating necessary design data to nominally assess the performance of an adsorption system for single and multi-component wastestreams.

This technique, described by Beaudet et al. (1980), utilizes a high pressure precision metering pump to pass wastewaters through a very small column at pressures up to 6,000 PSI. Meticulous care must be taken to avoid contamination of the absorbent during assembly of the apparatus to assure accurate and reproducible results. Additionally, influent and effluent samples must also be protected from contamination by ambient airborne organics.

Beaudet et al. (1980) conducted several tests with the DMCAT on several single and multi-components, and "real world" wastestreams and them compared their results with those from pilot scale tests. Their results were congruent with pilot scale results. The utility of DMCAT is to rapidly obtain reproducible data that can be used to determine the amenability of a particular wastestream to carbon adsorption and carbon usage rates to estimate system economics.

Applications of Activated Carbon Adsorption

In one laboratory study (Walk, Haydel, 1980a), two commercially available adsorbents were tested on an unspecified industrial wastestream in a laboratory study. The wastestream was from a multi-product effluent that contained five priority pollutants; chlorobenzene (8.8 ppm), p-dichlorobenzene (360 ppm), nitrobenzene (166 ppm),

dinitrotoluene (7.9 ppm), and phenol (30.8 ppm). Carbon adsorption was found to be effective, exhibiting effluent concentrations of 0.1 ppm or less for each of the priority pollutants. These results are based on equilibrium and dynamic testing. Walk, Haydel, (1980b) also compiled data on commercially operating systems in a limited survey. These data are presented in Table E-8. Hydroscience (1981) conducted a more extensive survey of facilities using carbon absorption to treat process wastewaters. Approximately 50 plants primarily engaged in organic chemicals or pesticide manufacture were queried. Other facilities included in the survey were those expected to generate process wastewaters that may be amenable to carbon adsorption treatment, such as refineries, coke plants, and detergent manufacturers. The design characteristics of these full scale adsorption systems are presented in Table E-9 and the performance of these systems in removing priority pollutants is summarized in Table E-10.

The utility of these data are somewhat limited because survey respondents often provided incomplete data. For example, most facilities reported influent streams in terms of BOD, COD, and only one or two priority pollutants. In some survey responses, only influent or only effluent concentrations of pollutants were reported. However, as noted elsewhere, a particular plant in the organic chemicals industry will only have a small number of priority pollutants in its wastestream in addition to conventional organic pollutants. Because the specific wastestream components were not sufficiently identified in this survey, the impact of specific priority pollutants on systems removal performance can only be inferred. In particular, data are inadequate to assess the competition among individual pollutants for active adsorption sites in a multicomponent stream. Of the systems surveyed by Hydroscience (1981), many were effective in the removal of specific priority pollutants.

TABLE E-8

SUMMARY OF COMMERCIALLY OPERATING CARBON ADSORPTION SYSTEMS

PARAMETER \ PLANT	1		2		3		4		5		6		7		8		9		10		11		12	
	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF	INF	EFF
Acenaphthene	297	80.5																						
Benzene	140	1.0	295	10	73500	10									20	0	384	250						
Chloroform	2.0	2.0																						
2-Chlorophenol	639	15.5																						
2,4-Dimethylphenol	259	4.0	67	0	10	0																		
Ethylbenzene	178	10	355	110																				
Fluoranthene	100	233	160	8	25	5																		
Naphthalene	1116	479																						
Phenol	55	7.5	500	20	630	60			650[A]	0.2[A]					226	0	8190	137			210[A]	3[A]	22400	100
Benzo(a)pyrene	5	35																						
Benzo(k)fluoranthene	8	66																						
Chrysene	22	124																						
Acenaphthylene	98	12.5	590	5	140	5																		
Anthracene	446	340																						
Fluorene	222	92.5	100	5	32	5																		
Phenanthene	446	340																						
Pyrene	69	157.5	131	5	6	5																		
Toluene	79	10	380	10	940	5																		
Nitrophenols													7200000	600000			24400	5470						
Chlorobenzene															40	0								
Toxaphene							6	2																
Nitrobenzene																	189000	1180						
Methyl Chloride															1000	0								
Cadmium															.6	.6	32	43						
Chromium															7	4	114	113						
Lead											6000	75			28	25								
Mercury																	7.1	.6						
Nickel																	348	298						
Cyanide																	7550	3730						
Dinitrotoluene																			1830000	1500				
Isophorone			230	0																				
Dimethyl Phthalate			285	5																				
Acrylonitrile					800	10																		

A = lbs/day
INF = influent concentration (ppb)
EFF = effluent concentration (ppb)

SOURCE: Walk, Haydel, 1980.

TABLE E-9

DESIGN CHARACTERISTICS AND OPERATIONAL PARAMETERS FOR FULL-SCALE GRANULAR ACTIVATED-CARBON ADSORPTION SYSTEMS

			PRETREATMENT								CONFIGURATION													
PLANT	FLOW (m^3/day)	FLOW (gal/day)	Equalization	pH Adjustment	Filtration	API Separation	Dissolved-Air Flotation	Biological	Clarification	Resin Adsorbers	Chemical Coagulation	Upflow	Downflow	Series	Parallel	Expanded Bed	Pulsed Bed	Fixed Bed	NUMBER OF ADSORBERS	HEIGHT (m)	HEIGHT (ft)	DIAMETER (m)	DIAMETER (ft)	HYDRAULIC LOADING m^3/(min) (m^2)	HYDRAULIC LOADING (gpm/ft^2)	C[illegible] Temp (°C)
01																										
02	5,184	1,370,000																								105
03	75,686	20,000,000	•	•	•			•	•			•			•	•	•		10	12.2	40	4.9	16	0.32	8.0	30
04	15,894	4,200,000				•	•				•		•		•				12	7.0	23			0.07	1.69	50
05	34.8	9,000																								
06	3,983	1,052,460			•	•						•			•	•	•		3	19.8	65	3.0	10	0.13	3.18	109
07	27.5	7,267			•							•			•			•	4	3.0	10	0.6	2	0.23	0.2	9
08	606	160,000																	4							
09	545	144,000											•	•				•	4	6.1	20	2.4	8	0.06	2.0	75
10	170	45,000										•		•			•		2	3.7	12	1.5	5	0.06	1.0	60
11																										
12																				9.1	30	3.0	10			
13	76	20,000										•					•		2							
14	9,009	2,392,000	•				•					•			•		•		3	21.3	70	3.7	12	0.27	6.6	50
15																										
16																										
17	50.5	13,350				•			•									•								
18	57	15,000	•	•	•	•	•			•									1							
19	18,921	5,000,000										•			•	•			2	13.7	45	5.5	18	0.28	6.8	30
20	246	65,000																								
21	189	50,000	•		•	•						•		•				•	2	3.3	11	3.0	10	0.02	0.44	170
22	1,607	424,000	•					•	•		•			•					3	6.1	20	3.0	10	0.15	3.75	60
23	284	75,000																	4							
24	1,892	500,000	•	•					•			•			•		•		2	11.0	36	3.7	12	0.06	1.5	173
25															•				2							
26	191	50,400	•	•									•	•				•	2							120
27	378	100,000										•		•				•	2	10.7	35	2.4	8	0.06	1.4	190
28	83	22,000																								
29	219	42,000											•	•				•	2	9.1	30	1.2	4	0.13	3.2	140
30	1,907	500,000		•			•												4							
31	30	8,000																								
32	47	15,000											•	•				•								540
33	1,892	500,000										•					•		1					0.19	4.7	
34																										
35	227	60,000	•	•	•							•					•		1	6.7	22	2.9	9.5			
36	38	10,000																								
37	5,449	1,440,000													•				2					0.18	4.4	60
38	436	115,200	•		•				•			•							2							
39	1,090	288,000		•	•			•											3	2.4	8.0	2.4	8.0			
40												•							2					0.02	.60	560
41																								0.01	.32	?
42														•						3.0	10	.61	2	0.09	2.1	15
43														•	•				2					0.03	[illegible]	[illegible]
44				•	•	•								•	•									0.02	.36	?
45														•	•				2						51	?

SOURCE: Hydroscience, 1981.

TABLE E-10

COMPOUNDS REPORTED IN WASTESTREAMS BEING TREATED BY FULL-SCALE GRANULAR ACTIVATED-CARBON UNITS

POLLUTANT	CONCENTRATION (mg/liter) INFLUENT	EFFLUENT	REMOVAL %	PLANT
Acetic Acid				09
Acetic Acid				10
Acetone	10			09
Aldrin	0.0085	0.00019	97.76	-
Aniline		1		02
Aniline				10
Atrazine	29.5	8.78	70.2	-
Benzene	10	1		09
Benzene	0.001	0.0001	90.00	-
Benzene	590	210	64	14
Benzene	0.010	0.100		-
Butanol				10
Butyl Acetate				10
Para-Butylaniline				13
N-Butylaniline				13
Butylphenol	0.300	0.015	95.00	-
Carbofuran	2,250	0.46	99.9	45
Carbon Tetrachloride		0.6		02
Carbon Tetrachloride	0.0011	0.0001	90.91	-
Carbon Tetrachloride	4.34	1.81	58	-
Chloramines				35

TABLE E-10 (Continued)

POLLUTANT	CONCENTRATION (mg/liter) INFLUENT	EFFLUENT	REMOVAL %	PLANT
Chlordane	0.013	0.00035	97.30	-
Chlorocresol				27
Chloroform		0.9		02
Chloroform	190.32	136.07	29	-
para-Chloronitrobenzene	11.6	0.0093	99.9	41
Chlorophenol				27
2-Chlorophenol	8.67	0.62	92.85	-
4-Chlorophenol	5.64	0.010	99.82	-
Chlorophenol				21
Cresol	0.230	0.0081	96.47	-
Cresol				21
Cresol	16.5	0.62	96.2	27
para-Cresol	5,000	350	93	29
Cyclohexylamine				10
Cyclotetramethylene Tetranitroamine (HMX)				-
Cyclotrimethylene Trinitroamine (RDX)	89.5	32.7	64	-
2,4-D	3,577	0.010	99.99	-
2,4-D	58.4	0.037	99.9	40
2,4-D, 2,6-D-Acids (mixed)	459.9			27

TABLE E-10 (Continued)

POLLUTANT	CONCENTRATION (mg/liter) INFLUENT	EFFLUENT	REMOVAL %	PLANT
2,4-D-Butyl Ester	0.010	0.010		-
2,4-D-Isooctyl Ester	1.862	0.586	69	-
2,4-DB	474	0.034	99.99	-
2,4-DB-Isooctyl Ester	0.010	0.010		-
DEET	218	1.26	99.4	44
Diallyl Phthalate				13
Dichlorobenzene		6		02
Dichlorobenzene				20
ortho-Dichlorobenzene				39
para-Dichlorobenzene				39
2,6-Dichlorophenol	3.47	0.26	92.51	-
2,4-Dichlorophenol	42.65	0.64	98.50	-
Dicofol	17.2	10.5	39.1	42
Dieldrin	0.0110	0.00001	99.99	-
Diethyl Formamide				10
Diethyl Toluamide				13
Diethylamine				10
Dimethylamine				10
Dimethylaniline	0.380	0.023	93.95	-
Dimethylphenol	1.220	0.0054	99.56	-
Dinitrobutylphenol	0.008	0.000002	99.98	-

TABLE E-10 (Continued)

POLLUTANT	CONCENTRATION (mg/liter) INFLUENT	EFFLUENT	REMOVAL %	PLANT
Dinitrocresol		0.4		02
Dinitrotoluene	14.5			02
Dinitrotoluene (DNT)				-
1,3-Dioxane				22
Dioxtyladipate	0.360	0.320	11.00	-
Diphenylamine				10
Ethanol				10
Ethyl Acetate				10
Ethyl Acrylate				10
Ethyl Chloroacetate				10
Ethylamine				10
Ethylenediamine				10
Formaldehyde				10
Fumaric Acid		1		02
Heptachlor	0.00610	0.00006	99.02	-
Isopropanol				10
Kepone	4.0	0.0001	99.98	-
Maleic Acid		1		02
Malic Acid		45		02
Methanol				10
Methyl Cyclohexanone		1		02
Methylene Chloride		0.9		02

TABLE E-10 (Continued)

POLLUTANT	CONCENTRATION (mg/liter) INFLUENT	EFFLUENT	REMOVAL %	PLANT
Methylene Chloride	0.190	0.051	73.15	-
Methylene Chloride	108.06	28.88	73	-
Methylene Dianiline		2		02
Monochlorobenzene		1		02
Monochlorobenzene				39
Monomethylamine				10
Mononitrotoluene		4.0		02
Nitrobenzene		3		02
para-Nitrophenol				20
Octyl Alcohols	438.5			27
Octyl Chloride				13
PCB	0.400	0.000075	99.98	-
PCB	0.019	0.0001	99.47	-
Pentachlorophenol	10.0	0.0001	99.98	-
Pentachlorophenol	120	49	59	14
Phenamines				35
Phenol	26.6	1.3	95	10
Phenol	2,500			12
Phenol		0.11		13
Phenol	4.66	0.58	88	14
Phenol	290	0.10	99.9	17
Phenol		0.10		18
Phenol	15	0.10	99	21

TABLE E-10 (Continued)

POLLUTANT	CONCENTRATION (mg/liter) INFLUENT	EFFLUENT	REMOVAL %	PLANT
Phenol		0.02		22
Phenol				01
Phenol		2.7		02
Phenol	1.9	0.7	63	06 [a]
Phenol	32.0	12.9	60	06 [a]
Phenol	20.0	0.023	99.9	06 [a]
Phenol	12.0	4.5	63	06 [a]
Phenol	13.5	15.1		06 [a]
Phenol	300	4	98.6	09
Phenol	420	4.2	99	24
Phenol	52.9	4	92	27
Phenol	77.9	2.32	97	40
Phenol	129	4.26	97	44
Phenol	0.140	0.0001	99.92	-
Phenol	52.9	0.69	98.7	27
Phenol	2.70	0.11	95.92	-
Bis(2-Ethylhexyl)Phthalate		330		14
Piperonyl Butoxide				13
Piperonyl Butoxide	7.57	0.01	99.9	44
Propylenediamine				10
Resorcinol				21
Tetrachlorobenzene				39
Tetryl				-

[a] Data taken at different time periods.

TABLE E-10 (Continued)

POLLUTANT	CONCENTRATION (mg/liter) INFLUENT	EFFLUENT	REMOVAL %	PLANT
Toluene		1		02
Toluene	0.120	0.0003	99.75	-
Toluene	2,500	630	75	14
Toluene	0.016	0.019		-
Toluene Diamine		1		02
Toluidine		1		02
Toxaphene	0.036	0.001	97.22	-
Trichlorobenzene		1		02
Trichloroethane	0.012	0.0001	99.17	-
Trichloroethylene		1		02
Trichloroethylene		1		02
Trichloroethylene	0.021	0.0003	98.57	-
Trichloroethylene	0.010	0.010		-
2,4,6-Trichlorophenol	34.96	0.010	99.97	-
Trifluraline	3.37	0.004	99.9	43
Trimethylamine				10
Trimethylol-Propane Trimethylacrylate				13
Trimethylphenol	0.130	0.010	92.30	-
Trinitrotoluene (TNT)	118.2	2.6	98	-
Xylene	0.140	0.0001	99.92	-
Xylene				21

Although carbon adsorption systems require extensive lab and scale up testing on a particular wastestream the results of the survey suggest that such systems are a viable and acceptable technology for use in priority pollutant removal from multi-component wastestreams generated by full scale plants.

A daily sampling effort was conducted by EPA at an organic chemicals manufacturing plant (EPA, 1981c). Effluent concentrations from a carbon adsorption unit used to treat process wastewaters are presented in Table E-11. These results are from one laboratory only and exhibit some pronounced variability among parameters. Parameters were selected on the basis of the process chemistry and are expected to be present in the effluent. During the study, two other labs also conducted sample analysis which also exhibited a degree of variability. Whether these differences are attributable to system pertubations or analytical deviations is not known because a statistical analysis of these data is not yet available. EPA is currently conducting such an analysis which will better define this particular system's performance.

TABLE E-11

DAILY SAMPLING RESULTS OF FULL SCALE CARBON ADSORPTION SYSTEM EFFLUENT (ppb)

DAY \ PARAMETER	BENZENE	NITROBENZENE	PHENOL	2-NITROPHENOL	4-NITROPHENOL	2,4-DINITROPHENOL	TOTAL PHENOLS	TOTAL CYANIDE	CADMIUM	CHROMIUM	COPPER	LEAD	MERCURY	NICKEL	ZINC
1	<45	98	<10	816	<10	82,100	150	860	35	30	95	130	24	190	80
2	<45	<10	160	303	338	16,400									
3	<45	<10	<10	282	<10	262,000									
4	<45	281	<10	3,990	<10	49,300			50	30	110	180	<.2	180	70
5	<45	2,160	<10	375	<10	39,700									
6	<45	<10	80	5,580	505	68,300									
7	<45	<10	<10	134	<10	42,300	45	720	70	30	80	150	.2	210	80
8	<45	<10	<10	496	<10	235,000									
9	<45	1,960	<10	344	<10	9,990									
10	<45	49	<10	36	<10	887									
11	<45	2,340	<10	95	<10	13,700	15	260	70	40	40	90	<.2	350	90
12	2,720	12	16	68	98	20,400									
13	<45	<10	<10	58	1,450	21,000									
14	<45	26	473	4,420	270	21,700									
15	<45	<10	50	1,000	127	22,700	540	420	80	60	110	100	<.27	340	80
16	<45	96	13	496	<10	26,000									
17	<45	71	<10	258	<10	17,700									
18	543	240	<10	414	<10	13,300									
19	<45	<10	<10	<10	<10	867	<40	250	70	20	70	230	<.2	570	60
20	<45	<10	<10	<10	<10	815									
21	<45	232	<10	37	<10	35,700									
22	<45	234	<10	396	672	35,900									
23	<45	9,800	126	929	1,185	64,200	300	3,000	40	<20	90	220	.24	50	70
24	<45	<10	<10	139	210	14,600									
25	<45	<10	<10	179	<10	21,000			62	167	229	211	0.2	285	85

SOURCE: EPA, 1981.

STEAM STRIPPING

Introduction

Steam stripping is a mass transfer operation that is used to remove volatile organic contaminants from dilute solutions. It is essentially a distillation that uses live steam as its energy source and is typically used for liquids that are immiscible in water. Steam stripping may be employed for binary distillations, and is also amenable to multicomponent streams. Design equations and criteria are well established and in some cases there is little need for extensive pilot plant studies prior to installing a large scale unit.

Background

Steam stripping is accomplished by injecting live steam into a vertical mass transfer column. Steam distillation, a batch process, is commonly used to distill organic liquids, which might decompose if heated to temperatures high enough to cause them to boil in the absence of steam. Stripping columns may be packed, sieve tray, or bubble cap tray. Packed columns are preferred because they maximize the interfacial surfaces available for mass transfer; however, they may not be the most economical. Consideration must also be given to the compatibility of the mixture to be separated with the materials used to construct the column in order to assess the expected lifetime of the packing. Additionally, throughput rate, energy requirements, maintenance, and operating costs may dictate a column design of either the sieve tray or valve tray type. Bubble cap tray columns are seldom used today because they have been replaced by valve trays. Either choice is about twice as expensive (capital cost) as sieve tray columns.

Regardless of the column design the principle of separation of mixture components is the same. Once the column is in a steady state condition, a temperature gradient and a congruent series of gas-liquid equilibrium stages are established. Standard design equations that relate the vapor pressure, activity coefficients, and equilibrium

TABLE E-12

THERMODYNAMIC PROPERTIES AND VAPOR-LIQUID EQUILIBRIUM CONSTANTS FOR PRIORITY POLLUTANTS

POLLUTANT	RANGE OF ACTIVITY COEFFICIENTS	VAPOR PRESS AT 100°C (kPa)	AVERAGE K VALUES
Acrolein	17 - 5.9	400	45.2
Acrylonitrile	17.5 - 14.3	196	30.75
Benzene	654.2 - 665.7	187	1215
Carbon Tetrachloride (tetrachloromethane)	1654	200	3264
Chlorobenzene	1907	38.7	727.7
1,2,4-Trichlorobenzene	24553	3.3	795
Hexachlorobenzene	3,775,000	0.06	2285
1,2-Dichloroethane	173	167	284
1,1,1-Trichloroethane	378	213	796
Hexachloroethane	21387	4.8	1013
1,1-Dichloroethane	249	333	819
1,1,2-Trichloroethane	371	66	240.7
1,1,2,2-Tetrachloroethane	634	25	154.3
Chloroethane	110.7	1173	1282
Bis(chloromethyl) ether	decomposes in water		
Bis(2-chloroethyl) ether	204	7.6	15.3
2-Chloroethyl vinyl ether (mixed)	119	81	95.5
Chloroform (trichloromethane)	210	307	635.5
1,2-Dichlorobenzene	5392	8.1	429.2
1,3-Dichlorobenzene	7121	10.8	758.9
1,4-Dichlorobenzene	14,200	9.7	1364
1,1-Dichloroethylene	1990	720	14139 [illegible]
1,2-Trans-dichloroethylene	1439	493	7005

TABLE E-12 (Continued)

POLLUTANT	RANGE OF ACTIVITY COEFFICIENTS	VAPOR PRESS AT 100°C (kPa)	AVERAGE K VALUES
1,2-Dichloropropane	489	111	535.3
1,3-Dichloropropylene (1,3-Dichloropropene)	694.6	81	557.5
2,4-Dinitrotoluene	5028	0.36	17.9
2,6-Dinitrotoluene	5028	0.36	17.9
1,2-Diphenylhydrazine	19	0.03	0.006
Ethylbenzene	2730	33.9	912.4
Bis(2-chloroisopropyl) ether	985	6.0	58.3
Methylene chloride (dichloromethane)	227 - 159	600	941.4
Methyl chloride (chloromethane)	37.2	4026	1478
Methyl bromide (bromomethane)	1023	1840	18575
Bromoform (tribromomehthane)	881	21.9	190
Dichlorobromomethane	197	135	262
Trichlorofluoromethane	1172	840	9715
Dichlorodifluoromethane	19871	3266	640,578
Chlorodibromomethane	416	57	235
Hexachlorobutadiene	5680	2.7	149.5
Hexachlorocyclopentadiene	72,114	0.72	512.4
Isophorone	207.6	2.5	5.2
Nitrobenzene	621	2.7	16.6
Tetrachloroethylene	6,690	53.3	3,521
Toluene	1,569	74.7	1,156
Trichloroethylene	1,131	150	1,674
Vinyl chloride (chloroethylene)	145	3240	4,627

TABLE E-13

STEAM STRIPPING OF ORGANIC PRIORITY POLLUTANTS WITH AQUEOUS REFLUX
(inlet concentration = solubility limit)

POLLUTANT	STEAM STRIPPER OUTLET CONCENTRATION 10^{-6} g/L (ppb)	NO. OF THEORETICAL TRAYS REQUIRED
Acrolein	50	19
Acrylonitrile	50	13
Benzene	50	5
Carbon Tetrachloride (tetrachloromethane)	50	4
Chlorobenzene	50	5
1,2,4-Trichlorobenzene	50	4
Hexachlorobenzene	50	3
1,2-Dichloroethane	50	6
1,1,1-Trichloroethane	50	6
Hexachloroethane	50	3
1,1-Dichloroethane	50	6
1,1,2-Trichloroethane	50	5
1,1,2,2-Tetrachloroethane	50	5
Chloroethane	50	7
Bis(chloromethyl) ether		
2-Chloroethyl vinyl ether (mixed)	50	6
Chloroform (trichloromethane)	50	6
1,2-Dichlorobenzene	50	4
1,3-Dichlorobenzene	50	3
1,4-Dichlorobenzene	50	4
1,2-Trans-dichloroethylene	50	
1,2-Dichloropropane	50	5
1,3-Dichloropropylene (1,3-Dichloropropene)	50	5
2,4-Dinitrotoluene	50	10
2,6-Dinitrotoluene	50	10
Ethylbenzene	50	3
Bis(2-chloroisopropyl) ether	50	9
Methylene chloride (dichloromethane)	50	6
Methyl chloride (chloromethane)	50	6

TABLE E-13 (Continued)

POLLUTANT	STEAM STRIPPER OUTLET CONCENTRATION 10^{-6} g/L (ppb)	NO. OF THEORETICAL TRAYS REQUIRED
Methyl bromide (bromomethane)	50	3
Bromoform (tribromomethane	50	6
Dichlorobromomethane	50	6
Trichlorofluoromethane	50	3
Dichlorodifluoromethane	50	1
Chlorodibromomethane	50	6
Hexachlorobutadiene	50	1
Hexachlorocyclopentadiene	50	2
Nitrobenzene	50	19
Tetrachloroethylene	50	4
Toluene	50	5
Trichloroethylene	50	4
Vinyl chloride (chloroethylene)	50	4

TABLE E-14

STEAM STRIPPING OF PRIORITY POLLUTANTS WITHOUT REFLUX
(inlet concentration = solubility limit)

POLLUTANT	STEAM STRIPPER OUTLET CONCENTRATION 10^{-6}g/L (ppb)	NO. OF THEORETICAL TRAYS REQUIRED	STRIPPING STEAM REQUIREMENTS kg steam/kg TOTAL FEED	OVERALL COLUMN EFFICIENCY
Acrylonitrile	50	16	0.085	100
Benzene	50	7	0.085	100
Carbon Tetrachloride (tetrachloromethane)	50	6	0.002	100
Chlorobenzene	50	0	0.005	100
1,2,4-Trichlorobenzene	50	6	0.003	99
Hexachlorobenzene	50	5	0.003	78
1,2-Dichloroethane	50	8	0.018	100
1,1,1-Trichloroethane	50	9	0.005	96
Hexachloroethane	50	7	0.003	100
1,1-Dichloroethane	50	8	0.005	100
1,1,2-Trichloroethane	50	8	0.02	100
1,1,2,2-Tetrachloroethane	50	8	0.03	100
Chloroethane	50	10	0.003	95
Bis(chloromethyl) ether	50			
2-Chloroethyl vinyl ether (mixed)	50	8	0.06	100
Chloroform (trichloromethane)	50	8	0.008	100
1,2-Dichlorobenzene	50	8	0.007	96
1,3-Dichlorobenzene	50	5	0.007	98
1,4-Dichlorobenzene	50	4	0.005	100
1,1-Dichloroethylene	50	6	0.0004	100
1,2-Trans-dichloroethylene	50	10	0.0004	100
1,2-Dichloropropane	50	7	0.01	100
1,3-Dichloropropylene (1,3-Dichloropropene)	50	7	0.01	100
2,4-Dinitrotoluene	50	18	0.1	82
2,6-Dinitrotoluene	50	18	0.1	82
Ethylbenzene	50	6	0.004	97

TABLE E-14 (Continued)

POLLUTANT	STEAM STRIPPER OUTLET CONCENTRATION 10^{-6}g/L (ppb)	NO. OF THEORETICAL TRAYS REQUIRED	STRIPPING STEAM REQUIREMENTS kg steam/kg TOTAL FEED	OVERALL COLUMN EFFICIENCY %
Bis(2-chloroisopropyl) ether	50	14	0.06	63
Methylene chloride (dichloromethane)	50	8	0.006	100
Methyl chloride (chloromethane)	50	7	0.004	100
Methyl bromide (bromomethane)	50	4	0.001	100
Bromoform (tribromomethane)	50	9	0.02	100
Dichlorobromomethane	50	8	0.02	100
Trichlorofluoromethane	50	5	0.001	100
Dichlorodifluoromethane	50	2	0.0007	100
Chlorodibromomethane	50	4	0.02	100
Hexachlorobutadiene	50	4	0.02	88
Hexachlorocyclopentadiene	50	4	0.01	85
Tetrachloroethylene	50	6	0.001	98
Toluene	50	8	0.003	96
Trichloroethylene	50	7	0.003	100
Vinyl chloride (chloroethylene)	50	6	0.002	100

constants to the material balance in the staged column (with and without reflux) are presented in Appendix K.

Volatile organic components having a partial pressure at a particular temperature evaporate or distill and are removed at the top of the column while the "stripped" liquid phase water is discharged from the bottom. The presence of steam in the vapor phase reduces the partial pressure of the other components at a fixed total pressure and thus lowers the saturation temperature of the liquid to be distilled. The column tops may be incinerated, partially condensed, or totally condensed. The water from the column tops may be refluxed to the column depending on the economic utility of refluxing. Refluxing offers the advantage of recovering organics as a liquid and increases the degree of separation of the column. It also increases the steam requirements to satisfy the overall heat balance for the column. Again, as with the column design, selection of refluxing options are based upon economic considerations.

State-of-the-Art

Standard design equations that relate the vapor pressure, activity coefficients, and equilibrium constants to the material balance in the staged column (with and without reflux) can be used to predict the column performance. Hwang and Fahrenthold (1980) have compiled thermodynamic data pertaining to 99 (of the 129) priority pollutants potentially amenable to steam stripping. These data were extracted from the literature or calculated from solubility and other data. Data on compounds normally subject to steam stripping have been summarized and are presented in Table E-12 Tables E-13 and E-14 present calculated column efficiencies, tray and steam requirements, and outlet concentrations of the priority pollutants based upon data from five commercial steam stripping columns, several pilot plant studies, and laboratory data. These are calculated data assuming the inlet concentration to the stripper is the solubility limit of the specific pollutant. Additionally, the calculations assume ideal behavior for mixtures (i.e., no

intermolecular interactions among components). Pollutants that require more than 20 trays to effect an outlet concentration of 50 ppb or less were excluded because they may not be economically justifiable for wastewater treatment.

In a recent controlled and comparative bench scale study, the validity of the estimated thermodynamic data presented in Table E-12 was examined (EPA, 1981b). A water solution saturated with three nonpriority pollutants was steam stripped and the effluent concentrations were measured. These results were compared to predicted results that are calculated by assuming five theoretical plates. The measured and calculated results compared favorably (see Table E-15) although the measured effluent concentrations are slightly lower suggesting that the assumption of five theoretical plates was incorrect (i.e., the column has more than five plates). The calculated effluent concentrations were based on K values of these nonpriority pollutants that were derived from published vapor-liquid equilibrium data. As noted earlier, the average K values for priority pollutants presented in Table E-12are more or less based on solubility data rather than vapor-liquid equilibrium data. The experiment was repeated with the calibrated column using six priority pollutants (benzene, chlorobenzene, 1,1,2,2-tetrachlorobenzene, chloroform, ethylbenzene, and tetrachloroethylene).

Saturated aqueous solutions of the six priority pollutants were also stripped individually and in combination with each other. The calculated effluent concentrations, assuming one theoretical plate, were much lower (in some cases up to four orders of magnitude) than the measured values (see Table E-16). The calculated values in Table E-16 for priority pollutants were based on the estimated average K values presented in Table E-12. The assumption of one theoretical plate reduces the system to a simple single stage vapor-liquid equilibrium. These data suggest that, although column efficiencies for the priority pollutant may be less than those of the nonpriority pollutants examined in this study, the accuracy of the estimated K values presented in Table E-12

TABLE E-15

CALCULATED RESULTS FOR STEAM STRIPPING OF SOLUBLE NONPRIORITY POLLUTANTS ASSUMING A 5 THEORETICAL TRAY COLUMN

Component	Mole Fraction Feed X 10^5	Mole Fraction Bottoms X 10^5	Calculated Mole Fraction Bottoms X 10^5
Acetone	2,470	.329	51.2
2-Propanol	2,110	.122	12.9
Methanol	4,610	1,520	2,489

TABLE E-16

CALCULATED RESULTS FOR STEAM STRIPPING OF PRIORITY POLLUTANTS ASSUMING A 1 THEORETICAL TRAY COLUMN

Component	Mole Fraction Feed X 10^5	Mole Fraction Bottoms X 10^5	Calculated Mole Fraction Bottoms X 10^5
Benzene	5.53	.303	.000298
Chloroform	39.8	.0876	.00833
1,1,2,2-Tetrachloroethane	29.1	.364	.0875
Chlorobenzene	3.59	.437	.000794
Ethylbenzene	1.96	.130	.000179
Tetrachloroethylene	15.4	.365	.0000905
Ethylbenzene	.952	.102	.000101
1,1,2,2-Tetrachloroethane	11.2	.692	.0446
1,1,2,2-Tetrachloroethane	16.4	.200	.0452
Benzene	3.01	.0733	.000138
Chloroform	2.53	.0062	.000431
Ethylbenzene	.391	-	.0000409
Chlorobenzene	1.51	.140	.000238
Tetrachloroethylene	.468	.0166	.00000318
Chlorobenzene	1.32	.315	.000283
Ethylbenzene	.204	.0296	.0000280
Tetrachloroethylene	.174	.0129	.00000162
1,1,2,2-Tetrachloroethane	6.79	.0693	.0222
Chlorobenzene	.899	.0175	.00000137
Chloroform	9.52	.149	.00190
1,1,2,2-Tetrachloroethane	4.49	.192	.0188
Chloroform	9.10	.139	.00233
Ethylbenzene	.170	.00334	.0000213
Tetrachloroethylene	.105	.00107	.000000883
1,1,2,2-Tetrachloroethane	3.38	.118	.00932
Benzene	.809	.0859	.0000374
Chlorobenzene	.513	.0184	.0000658
Ethylbenzene	.117	.00372	.00000959
1,1,2,2-Tetrachloroethane	3.27	.00258	.00975
Benzene	3.08	.0555	.000153
Chlorobenzene	.257	.00876	.0000355
Ethylbenzene	.199	.00297	.0000176
Tetrachloroethylene	.0557	.000357	.000000344
Chloroform	.324	.00364	.0000588

may be somewhat limited in the range of influent concentrations investigated in this study and that calculations predicated on these data should be viewed in the context of these limitations.

Column efficiencies are among the most critical of design parameters in that they predict the actual number of trays for a tray column or the height of the packing for a packed column. The efficiency of any column is dependent on several phenomena: the degree of mixing of the liquid, entrainment of liquid in the vapor phase, and the contact time between phases. They are always empirically determined and essentially assess the performance capabilities of the column.

More recently Hwang has investigated tray and packing efficiencies in a follow-up study for mixtures at extremely low contaminant concentrations, utilizing pilot plant data and predictive correlations to revise and expand his earlier model (Hwang, 1980b). In this latter study, Hwang has suggested the grouping of volatile organic compounds based on common parameters (e.g., functional groups and molecular weight) to streamline the model. EPA has solicited industry for additional data concerning priority pollutants that are separated by steam strippers in order to assess the validity and accuracy of the correlations used for column design and modeling. These performance data on commercially operating wastewater steam stripping columns are presented in Table E-17.

TABLE E-17

INDUSTRIAL STEAM STRIPPERS

Column Type	Height (feet)	Diameter (feet)	Flow Rates (lb/hr) Feed	Bottoms	Inlet Conc	Outlet Conc
Packed*	75.42	3	17,500	1,200	2.63% Aniline	<0.001% Aniline
Trays*	52	3.5	6,960	5,789	5.51% TOC 7.18% Aniline 0.79% Benzene	0.042% TOC 0.03% Aniline 0.02% Benzene
Packed*	33.83	2.5	2,375	5,750	5% Aniline	>0.0005% Aniline
Trays*	80	6.5	70,000	99,750	NA	NA
Packed	54	2.5	33,750	34,300	0.52% Nitrobenzene	0.05% Nitrobenzene
Trays*	38	6	40,000- 90,000	13,900- 31,700	NA	NA
Packed*	36.8	2	7,500	8,200	4,980 ppm TOC 0.18 ppm Methylene chloride 1.05 ppm Methyl chloride 0.001 ppm Phenols	2,360 ppm TOC 0.001 ppm Methylene chloride 0.0018 ppm Methyl chloride 0.0065 ppm Phenols
Trays*	54.5	5.5-7.0	100,080	116,600	778 ppm Sulfide 833 ppm Ammonia 510 ppm Phenols	"Nil" Sulfide 36 ppm Ammonia 284 ppm Phenols

* With recycle

TABLE E-17 (Continued)

Column Type	Height (feet)	Diameter (feet)	Flow Rates (lb/hr) Feed	Bottoms	Inlet Conc	Outlet Conc
Trays	27.42	4.5-3	90	5,000	0.3% Methylene chloride	0.03% Methylene chloride
Packed*	42	3.0	25,931	23,154	1.07% Aniline 0.019% Methanol	0.009% Aniline 0.01-0.02% TOC
Packed	NA	2.5	16,886	15,886	0.697% TOC 1.88% BOD 0.75% Aniline 0.10% Methanol	0.01-0.02% TOC 0.23% BOD 0.02% Aniline
Trays and Packed*	30.33	1.66-3.25	3,958	3,916	2.3% TOC 2.98% Aniline	0.077% TOC 0.076% Aniline
Packed*	22	1	3,100	3,387	1.35% DIPA 7.26% Salts	0.03% DIPA 6.64% Salts
Packed	15	1	2,746	3,108	0.91% EDC 4.0% NaCl	3.54% NaCl
Packed*	15	2.0	28,600	29,067	0.79% EDC 1.04% HCl	1.025% HCl
Packed*	26	4	43,150	42,870	9,400 ppm EDC	85 ppm EDC 15 ppm VCM

* With recycle

TABLE E-17 (Continued)

Column Type	Height (feet)	Diameter (feet)	Flow Rates (lb/hr) Feed	Bottoms	Inlet Conc	Outlet Conc
Trays	(not given)	3.5	24,520	25,329	0.0595% TOC 0.076% BOD 0.05% NH_3 0.256% Sulfides	0.034% TOC 0.05% BOD 0.012% NH_3 0.0037% Sulfides
Packed*	8	0.5	1,611	1,603	6,828 ppm Benzothiazole 620 ppm Aniline	<60 ppm Benzothiazole <60 ppm Aniline
Packed	10.5	0.33	253	254	198 ppm of H_2S Trace-CS_2	Trace H_2S and CS_2
Trays	44	3	28,579	28,906	35 ppm Benzene 4,220 ppm MNB 12,440 ppm Na Salts	0 ppm Benzene 800 ppm MNB 12,300 ppm Na Salts
Trays*	24,83	2.5	41,897	41,669	1% Methylene Chloride 0.13% Chlorobenzene 0.00001% Octa-decylamine 5.22% NaCl	0.015% Methylene Chloride 0.0025% Chlorobenzene 5.59% NaCl
Trays*	30	2.5	57,000	55,961	0.35% TOC 1.66% Methylene chloride 0.091% Chlorobenzene	0.008% TOC 0.009% Methylene chloride 0.0007% Chlorobenzene

* With recycle

TABLE E-17 (Continued)

Column Type	Height (feet)	Diameter (feet)	Flow Rates (lb/hr) Feed	Bottoms	Inlet Conc	Outlet Conc
Packed*	17	1.5	0-5,000	0-5,000	800-1,000 ppm Vinyl Chloride	<10 ppm Vinyl Chloride
Packed	42	3.5	119,000	121,000	0.197% TOC 0.158% BOD 0.011% Vinyl Chloride 0.56% Dichloroethane 0.172% Other Organic Chlorides	0.095% TOC 0.112% BOD <0.0001% Vinyl Chloride <0.0002% Dichloroethane 0.017% Other Organic Chlorides
Packed	28	3.5	112,500	115,000	0.32% TOC 0.004% Vinyl Chloride 0.56% Dichloroethane	0.07% TOC <0.0005% Vinyl Chloride 0.021% Dichloroethane
Trays*	53	4	60,000	NA	3.3 ppm O/G 1.59 ppm Phenol 750-1,000 ppm TOC <10-1,000 ppm BOD	2.4 ppm O/G 1.99 ppm Phenol 10-100 ppm TOC 40-300 ppm BOD
Trays*	35	4	52,700	51,533	2% "H.C." "(hydrocarbon?)"	50-260 ppm H.C.

* With recycle

ORGANIC ADSORPTION RESINS

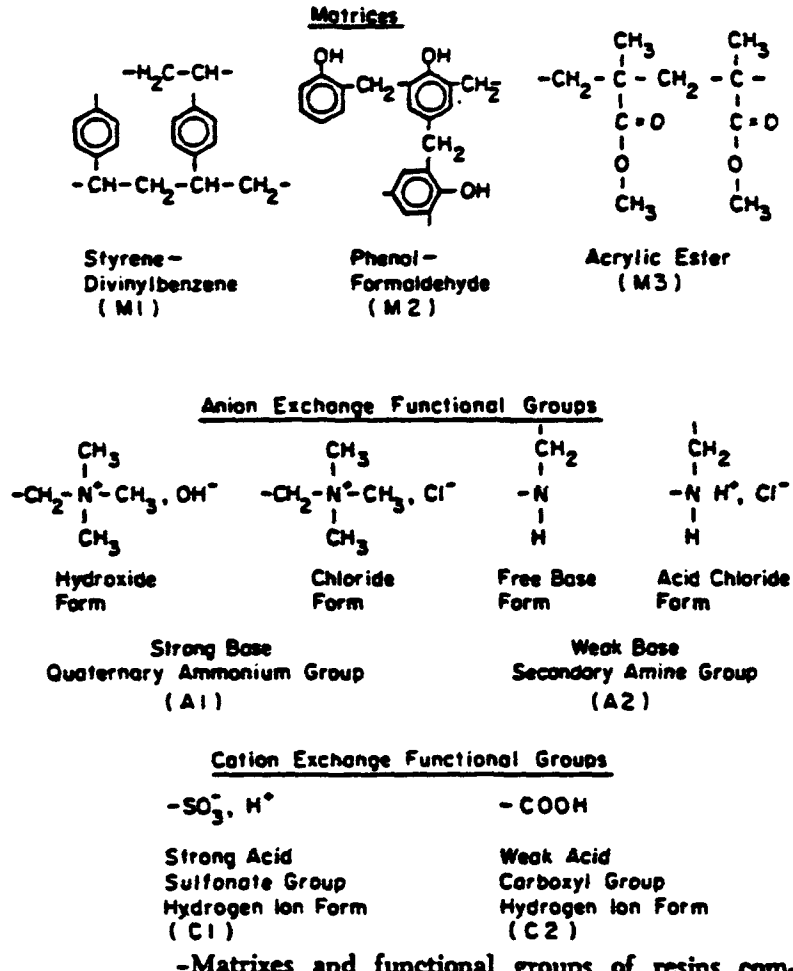
Introduction

Organic adsorption resins are a relatively new system for removing organic chemicals from aqueous streams. Resin adsorption performs like activated carbon: a waste stream passes through a bed of resin beads, which pick up dissolved organic molecules and colloidally suspended organic particles from the waste stream by means of the Van der Waals attraction between the resin and the organic molecules or particles. Resin adsorption has been shown to reduce organic priority pollutant levels well below the parts-per-million range, performing nearly as well as activated carbon. Its most attractive feature is the ease with which a resin bed can be returned to its original adsorption capabilities after use--the major disadvantages of resin adsorption are the high capital cost of the resin units and the problems of disposing of contaminated, spent regenerant.

Organic resin adsorption can be characterized as producing the same result as activated carbon--removing trace organic priority pollutants from a waste stream, and using the same equipment as ion exchange--columns of polymer resin beads. An organic adsorption resin unit is generally a bed of small beads. Each bead is an aggregate of many tiny microbeads of resin, ranging from 10^{-4} mm to 1 mm in diameter. These resin beds consist of agglomerated microbeads; the channels between these tiny spheres provide a large surface area--between 100 and 700 m^2/g per bed, depending on the type of resin--on which adsorption can occur. Some types of resin beads used in organic adsorption have the same structure as the resin beads used in ion exchange. Resin beads which do not have the ionic functional sites are also effective in removing organic compounds from aqueous solution. These beads consist of a polymer framework, or matrix (generally styrene-divinylbenzene copolymer, phenol-formaldehyde copolymer or acrylic ester polymer) which bears ionic acidic or basic functional groups. Figure E-4 shows the most

FIGURE E-4

WIDELY USED POLYMER MATRICES AND FUNCTIONAL GROUPS



-Matrixes and functional groups of resins commonly used for water purification.

SOURCE: Kim et al., 1976.

widely used polymer matrices and functional groups. Polymeric adsorbents have the same basic structure without the functional groups.

In addition to polymeric adsorption resins, there is another set of adsorption materials. These are carbonaceous adsorbents, which consist of black spheres roughly 10^{-5} mm in diameter, intermediate in composition between activated carbon and polymeric resin adsorbents. Several types of carbonaceous adsorbents ("Ambersorb" trade name) are included in the investigations by Rohm and Haas which are discussed later in this report.

For polymeric adsorbents the polymer matrix is the site of organic adsorption. Organic molecules are attracted to the resin's organic matrix by Van der Waals forces. Attractive forces between the adsorbed organic molecules and the adsorbent resin matrix are relatively weak, weaker than the attractive forces in activated carbon adsorption. This means that the resins can be effectively regenerated by solvent elution. The role of the polymer's ionic functionalities, weak and strong acid and base groups, is a secondary one of attracting particular types of polar organic molecules to the surface of the beads to facilitate their adsorption onto the resin matrix. The actual driving force of the adsorption process, however, is the affinity between an organic pollutant molecule and the hydrophobic polymer resin matrix. Adsorption occurs when this affinity is greater than the affinity between the organic pollutant molecule and the aqueous waste stream. Therefore, organic adsorption resins are most effective in situations where a nonpolar organic pollutant is to be removed from an aqueous stream; the presence of ionic functional groups acts to increase the resin's affinity for slightly charged organic molecules by introducing electrostatic attraction.

The pH of the aqueous stream being treated affects the degree to which slightly polar organic molecules are adsorbed onto the resin matrix. pH determines the extent to which an ionizable organic molecule will be polarized. Therefore, since a hydrophobic molecule will be more strongly attracted to the hydrophobic resin matrix than a

hydrophilic molecule, a weak organic acid (such as phenol) will be best adsorbed from an acid solution, where the organic acid will be present in its less water soluble nonionized form--this is the phenomenon known as "salting out."

Nonionic resins have been shown to be particularly effective in removing chlorinated pesticides, detergent compounds, emulsifiers, wetting agents, dispersants and all types of textile dyes. These resins consist of a polymer matrix and do not include any functional ionic groups; they are regenerated with methanol, acetone, isopropanol and similar solvents.

Weakly basic resins have been shown to be effective in removing phenolics, anionic surfactants, carboxylic acids, proteins, anionic textile dyes and kraftpaper waste color bodies; weakly basic resins with phenol-formaldehyde polymer matrices remove phenolic organics particularly well. Sodium hydroxide solutions regenerate these resins.

Strongly acidic and strongly basic resins do not perform as well as weakly acidic and weakly basic resins in terms of removing organic molecules. Furthermore, strongly ionic resins must be eluted with large amounts of acid or base; and disposing of these eluents requires special facilities.

Organic adsorption resins generally are effective in the same situations where activated carbon adsorption is effective. Resin adsorption differs from carbon adsorption in that resins can be manufactured to adsorb specific types of organic molecules by selecting the appropriate ionic functionality on the resin matrix and in that the attractive forces between adsorbent and pollutant molecule are weaker for resin adsorption than for activated carbon adsorption. The fact that pollutants are less strongly adsorbed onto resin than onto activated carbon has two significant implications: first, the effluent stream from a resin unit will have a higher concentration of residual organic pollutants than an effluent from an equivalent activated carbon unit and secondly, resin can be

regenerated with solvents or steam while activated carbon requires expensive thermal regeneration.

Although regeneration by elution restores the resin bed to most of its original effectiveness the elution stream presents a disposal problem. The net effect of an adsorption-elution cycle is to transfer pollutants from the waste stream to an eluent stream. A technique frequently used is to isolate the first portion of eluent to come out of the resin during elution. This eluent stream is relatively concentrated and can be sent to incineration or treated to recover the pollutant. The second portion of eluent will emerge from the resin bed with a lower pollutant concentration. This eluent stream can be recycled into the waste stream entering the resin bed, or it can be used as the first portion in the next elution.

Elution regeneration, nevertheless, effectively restores resin function. Fox, of Rohm and Haas, describes a commercial phenolic removal and recovery system using Amerlite XAD-4: "After 2 1/4-years of operation, the original resin performed the same as it did during startup. Resin capacity measurements made in the laboratory show the used resin has 98 percent of its original capacity for phenol even after 1,300 load, regeneration cycles" (Fox, 1978).

Rohm and Haas applications for their commercial resins are given in Table E-18.

TABLE E-18

ROHM AND HAAS COMMERCIAL RESIN APPLICATIONS

	Adsorbent/Regenerant in Wastewater Treatment	
Waste Stream/Process	US (14 sites)	Foreign (13 sites)
phenols (BPA and others)	XAD-4/Methanol XAD-4,XAD-7/Methanol	IRA-93/Methanol XAD-4/Acetone (3)
brine (phenols and others)	XAD-7/Methanol XAD-74% Caustic XAD-2,XAD-4/Methanol	
pesticides (cl-, NO_2-phenols)	XAD-4/Methanol XAD-4/Isopropanol XAD-4/2% Caustic	XAD-2/4% Caustic
nitroacromatics (TDI and others	XAD-4/Acetone	XAD-4/Methanol
amines (alkyl and aromatic)	XAD-2/Methanol XAD-2/4% Caustic	XAD-4/Toluene (2)
grease (misc. hydro-carbons	XAD-2/steam XAD-4/acetone	CAD-4/steam (2)
dyes	XAD-7/Methanol	CAD-4/Methanol
chlorinated organics unidentified	XAD-4/4% Caustic	XAD-4(?)/steam (2)

Rohm and Haas is a leading manufacturer and distributor of commercial organic adsorption resins; therefore, these listings describe a significant portion of world-wide resin use.

Organic adsorption resins are also used commercially to treat paper mill kraft wastes and are placed upstream to ion exchangers to prevent resin fouling. The latter application exploits the affinity between resins and organic pollutants which caused the fouling problem in the first place.

Preliminary Development of Resin Performance Data

Rohm and Haas Studies

Research aimed at developing reliable equations to predict the performance of any synthetic resin by means of parameters easily obtained experimentally is currently under way; no definitive design/performance equations have yet been developed. Rohm and Haas, a major commercial manufacturer of organic adsorption resins, has undertaken a testing program to develop a simple laboratory test which will evaluate the performance and cost of various synthetic resins in removing organic pollutants from industrial waste streams. This laboratory test is intended to establish applicability of synthetic resin adsorbents for removal of organic pollutants from industrial waste streams, to identify the most useful resin or combination of resins and suitable regenerants for them, and to allow estimation of approximate treatment costs. Results of these tests have been presented by Rohm and Haas as a series of quarterly reports entitled "Synthetic Resin Adsorbents in Treatment of Industrial Waste Streams" (1980, 1981).

The test evaluated in the Rohm and Haas study was the Batch/Rate Test, which measured both the amount of pollutant removed by a known mass of a certain resin and the rate at which the pollutant was adsorbed onto the resin. The Batch/Rate tests were performed on a variety of resins, including resins manufactured by Rohm and Haas, Mitsubishi, Diamond-Shamrock, Montedison, Calgon, and Dow Chemical. Batch/Rate test

results were supplemented by data from adsorption isotherm studies, and also by column loading/regeneration studies. In the column loading/regeneration studies, the effluent concentration of a pollutant-bearing stream passing through a resin column was monitored. Then, when the resin had been saturated with the pollutant, regenerant was sent through the column and analyzed as it was collected leaving the column to give the cumulative amount of pollutant eluted as a function of the volume of eluent pumped through the bed. The resins used in this project are listed and characterized in Table E-12. The synthetic waste streams used were single-solute aqueous solutions of 2-nitrophenol, tetrachloroethylene, 1,2-dichloropropane and 2,4-dinitrotoluene.

Pilot columns, routinely used to evaluate the adsorption performance of activated carbon, cannot be effectively used to evaluate organic adsorption resins, because synthetic resins are manufactured in a wide variety of resin types, with a broad range of performance characteristics, amenable to regeneration with numerous eluent solvents. Determining the optimum adsorbent/regenerant pair from all the combinations available would require a prohibitively expensive and time-consuming set of column experiments.

The first step in the Batch/Rate test is compiling adsorption isotherms for the adsorbents and waste streams being studied.

The Batch/Rate studies measure the capacity of a resin to adsorb pollutants from a stream and the rate at which this adsorption takes place. In the Batch phase of the Batch/Rate tests, the adsorbent is saturated with a pollutant in a stream, which is a solution of one of the four priority pollutants studied. The saturated adsorbent is then eluted with a regenerant solution (acetone or methanol), and the eluent is analyzed for the pollutant. In the Rate part of the Batch/Rate test, the adsorbent is exposed to the waste stream as it is in the Batch test. However, small portions of the resin are taken out of the waste at regular intervals, while the resin is still being saturated with pollutant. The

TABLE E-19

PHYSICAL PROPERTIES OF ADSORBENTS

Adsorbent	Manufacturer	Chemical Nature	Pore Vol (cm^3/g)	Surface Area (m^2/g)	Ave. Pore Dia. (Å)	Mesh Size	Surface Polarity
Amberlite XAD-2	Rohm & Haas	polystyrene	0.68	300	90	20-50	low
Amberlite XAD-4	Rohm & Haas	polystyrene	0.97	725	40	20-50	low
Amberlite XAD-7	Rohm & Haas	acrylic ester	0.99	450	90	20-50	intermediate
Amberlite XAD-8	Rohm & Haas	acrylic ester	0.08	160	225	20-50	intermediate
Ambersorb XE-340	Rohm & Haas	polymer carbon	0.34	400	200, 15[a]	20-50	very low
Ambersorb XE-347	Rohm & Haas	polymer carbon	0.41	350	200, 5[a]	20-50	low
Ambersorb XE-348	Rohm & Haas	polymer carbon	0.58	500	200, 15[a]	20-50	intermediate
Diaion HP-10	Mitsubishi	polystyrene	1.16	720	–[b]	20-50	low
Diaion HP-20	Mitsubishi	polystyrene	0.87	570	–[b]	20-50	low
Diaion HP-30	Mitsubishi	polystryene	0.63	700	–[b]	20-50	low
Filtrasorb 300	Calgon	activated carbon	0.85	1000	–[b]	8-30	intermediate
Filtersorb 400	Calgon	activated carbon	0.94	1125	–[b]	12-40	intermediate

[a] Ave. pore diameter of the macropores and micropores, respectively.
[b] Ave. pore diameter not available.

portions of resin removed are eluted with regenerant and the eluents are analyzed for pollutants; rate test results are presented as adsorption capacity (mg pollutant adsorbed per g of resin) determined as a function of the length of time the resin has been exposed to the pollutant.

The Batch/Rate tests were supplemented by column tests which were somewhat analogous in design to the Batch/Rate tests. Columns were packed with resin to construct a bench-scale resin adsorption unit, and column loading and column regeneration tests were performed. Column loading tests consisted of passing pollutant streams (synthetic single-solute solutions of one of four organic priority pollutants—2-nitrophenol, 2,4-dinitrotoluene, 1,2-dichloropropane and tetrachloroethylene) through a resin bed and monitoring the effluent pollutant concentration. In column regeneration tests, the columns that were saturated with pollutant in the column loading tests are eluted with regenerant. The regenerant leaving the column is analyzed for pollutant.

The adsorption data from isotherm, Batch/Rate, and column loading/regeneration studies are summarized in Table E-20. The batch test has proven to be a very good predictor of saturation column capacity for single component streams. It not only gives a more accurate saturation value than the isotherm test, it also is a much simpler test, requiring a single analysis after exposure to the influent waste stream.

Equations which will predict resin effectiveness in removing organic pollutants from industrial waste streams by means of easily determined parameters do not yet exist. However, Rohm and Haas' study represents the first steps towards these equations. The limitations of the body of data assembled to date (up to and including the third quarterly report, released by Rohm and Haas in August 1981) reflect the fact that synthetic resin adsorption is a new technology in the earliest stages of application.

The most significant limitation of the resin performance data is that no studies have been yet published which describe the performance of systems in the course of

TABLE E-20

SUMMARY OF ADSORPTION CAPACITIES MEASURED BY ISOTHERMS, BATCH TESTS, AND COLUMN EXPERIMENTS
ADSORPTION CAPACITY IN mg/g

Adsorbent	Adsorbate[1]	Isotherm[2,3]	Batch[3,4]	BV (mls)	Flow (BV/hr)	Column Loading	Column Regeneration[4]
XAD-4	Nitrobenzene	859	1310	23.1	8	1330	1250
				11.0	16	1260	1080
XAD-8	Nitrobenzene	456	580	11.6	16	569	530
HP-20	Nitrobenzene	670	1160	24.3	8	1150	1060
XAD-4	1,2-Dichloropropane	747	1060	23.9	8	1310	1150
				10.4	16	1240	1160
XAD-7	1,2-Dichloropropane	409	564	27.1	8	523	544
HP-20	1,2-Dichloropropane	778	1060	11.7	16	1200	1030
XAD-4	2,4-Dinitrotoluene	397	450	10.4	16	434	470
				11.4	24	432	474
XE-340	2,4-Dinitrotoluene	118	138	10.8	16	148	151

[1]Aqueous nitrobenzene concentrations: isotherm = 1900 ppm; batch = 1900 ppm; column = 1800 ppm
Aqueous 1,2-dichloropropane concentrations: isotherm = 2500 ppm; batch = 2450 ppm; column = 2350 ppm
Aqueous 2,4-dinitrotoluene concentrations: isotherm = 180 ppm; batch = 180 ppm; column = 180 ppm

[2]Capacity calculated from the Freundlich parameters assuming a linear isotherm

[3]Nitrobenzene data from the second quaterly progress report, ref. 2.

[4]Adsorbent regenerated with methanol in the nitrobenzene study and with acetone in both the 1,2-dichloropropane study and the 2,4-dinitrotoluene study.

multiple saturation-regeneration cycles. Therefore, estimates of an adsorbent's useful lifetime cannot be made, nor can the effectiveness of a multiply regenerated resin be predicted as a function of the number of its regeneration cycles. Because the ease with which resins can be regenerated is one of their most attractive features, a complete quantitative evaluation of resin performance will include examining the performances of resins which have been subject to more than one regeneration cycle.

A second limitation of the studies is that they were all performed on single-solute synthetic solutions of one of four organic priority pollutants. Performance data obtained from streams more closely resembling industrial waste streams will more reliably characterize resin performance in industrial application.

Although no equations have yet been developed to predict resin peformance, Rohm and Haas' investigation included a preliminary evaluation of the mathematical bases of resin adsorption capacities and rates, considered together with capacity and rate data from the Batch/Rate tests. This evaluation is the beginning of the development of a theory of resin adsorption kinetics and equilibria to be applied to resin system design and economics; it is not yet a reliable means of quantitatively predicting resin adsorption performance.

Walk, Haydel Studies

In addition, a treatability study comparing carbon and resin adsorption was run by Walk, Haydel (1980a) in which equilibrium and dynamic studies on organic resin systems were performed. Five organic priority pollutants were monitored: chlorobenzene, p-dichlorobenzene, nitrobenzene, dinitrotoluene, and phenol. The equilibrium studies were batch operations at three pH levels; it was found that adsorption performance was higher at pH 6 than at pH 9 and that the resin had a low adsorption capacity at low pollutant concentrations, adsorption capacity increased with the strength of the waste stream. Resin was found to be more effective than carbon for priority pollutant removal

at high pollutant concentrations, while carbon performed better than resin with a dilute waste stream; resin performance was more sensitive to waste stream concentration than carbon performance.

The dynamic studies of resin adsorption consisted of six runs with methanol regeneration between runs. Constant leakage, 3 percent of the organic components of the waste stream, was observed almost from the beginning of the runs. Table E-21 presents an economic analysis, based on regenerability observations. The Walk, Haydel studies concluded that resin adsorption of organic contaminants from an industrial wastewater is technically feasible, and that the economic justification rests on a combination of wastewater cleanup and specific compound recovery.

Conclusion

The conclusion to be drawn from the experimental studies by Rohm and Haas and Walk, Haydel is that synthetic resin adsorption may be equivalent to activated carbon adsorption in effectiveness in removing organic priority pollutants from industrial waste streams. The absence of quantitative data describing adsorption capacities and resin regenerability prevents a direct comparison between the two methods. However, if subsequent research indicates that resins can retain their original adsorption capacities after multiple saturation/regeneration cycles, the demonstrated effectiveness of resin priority pollutant adsorption plus the ease with which the regeneration operation can be performed suggest that resin adsorption may be a valuable means of removing organic pollutants from industrial waste streams.

TABLE E-21

TREATMENT COSTS FOR RESIN ADSORPTION

Basis: 1,130 l/min (300 gpm) treatment to 1 ppm p-dichlorobenzene.

Investment $1,700,000

Working Capital $ 370,000

				Treatment Costs	
Materials Cost				$/yr	$/1,000 l
Streams	units/yr	$/unit			
Resin					
Replacement	28.3m^3	8,833.92		250,000	
Royalty				12,000	
On Resin	28.3m^3	424.03			
Methanol	1,239,840 l	0.17		213,000	
			Subtotal	475,000	0.80
Processing Cost					
Labor Directed:					
Direct Labor				105,000	
Supervision 10% Direct Labor				10,000	
Plant Overhead 100% Direct Labor				105,000	
			Subtotal	220,000	0.37
Investment Directed:					
Amortization Cost*				351,000	
Taxes & Insurance		@2% Inv.		34,000	
Maintenance		@4% Inv.		68,000	
Supplies		@1% Inv.		17,000	
			Subtotal	470,000	0.79
Throughput Directed:					
Steam $0.77/100 kg				475,000	
Other Utilities				51,000	
			Subtotal	536,000	0.90
Total Treatment Cost				1,701,000	2.86

*Based on 12 years and 15 percent interst.

SOURCE: Walk, Haydel, 1980a.

METALS REMOVED

The two processes most widely used in industry to remove heavy metals are precipitation, followed by coagulation and flocculation, and ion exchange; both are effective in removing the metallic priority pollutants from waste streams. Precipitation-coagulation-flocculation uses relatively cheap and simple equipment and reagents to reduce high influent metals concentrations (e.g., 100 mg/liter) to concentrations near the ppm level. Ion exchange systems on the other hand can remove metals originally present at low concentrations (e.g., 1-2 ppm), but is complicated and expensive. Moreover, in many cases, the stream entering the ion exchange unit must be pretreated to remove contaminants that would damage the ion-exchange resin.

Precipitation

Precipitation is a common metals removal process based on the solubility of metal salts. While a particular salt of a given metal may be relatively soluble in water, another salt of the same metal may be much less soluble in water. An example is the silver ion: at pH 10, the silver cation in a solution of AgOH has a solubility of 10^2 g/liter, while the silver cation in a solution of Ag_2S has a solubility of 10^{-15} g/liter. Therefore, introducing sulfide to a solution of AgOH (at pH 10) reduces the solubility of silver by 17 orders of magnitude. Silver is precipated as Ag_2S, an insoluble salt.

The solubility of a salt is described quantitatively by its solubility product constant K_{sp}.

$$M_mX_n \rightarrow mM^+ + nX^-$$

For a salt solution in equilibrium with its solid precipitate, $K_{sp} = (M^+)^m(X^{m-})^n$ where m and n are the stoichiometric coefficients of each species.

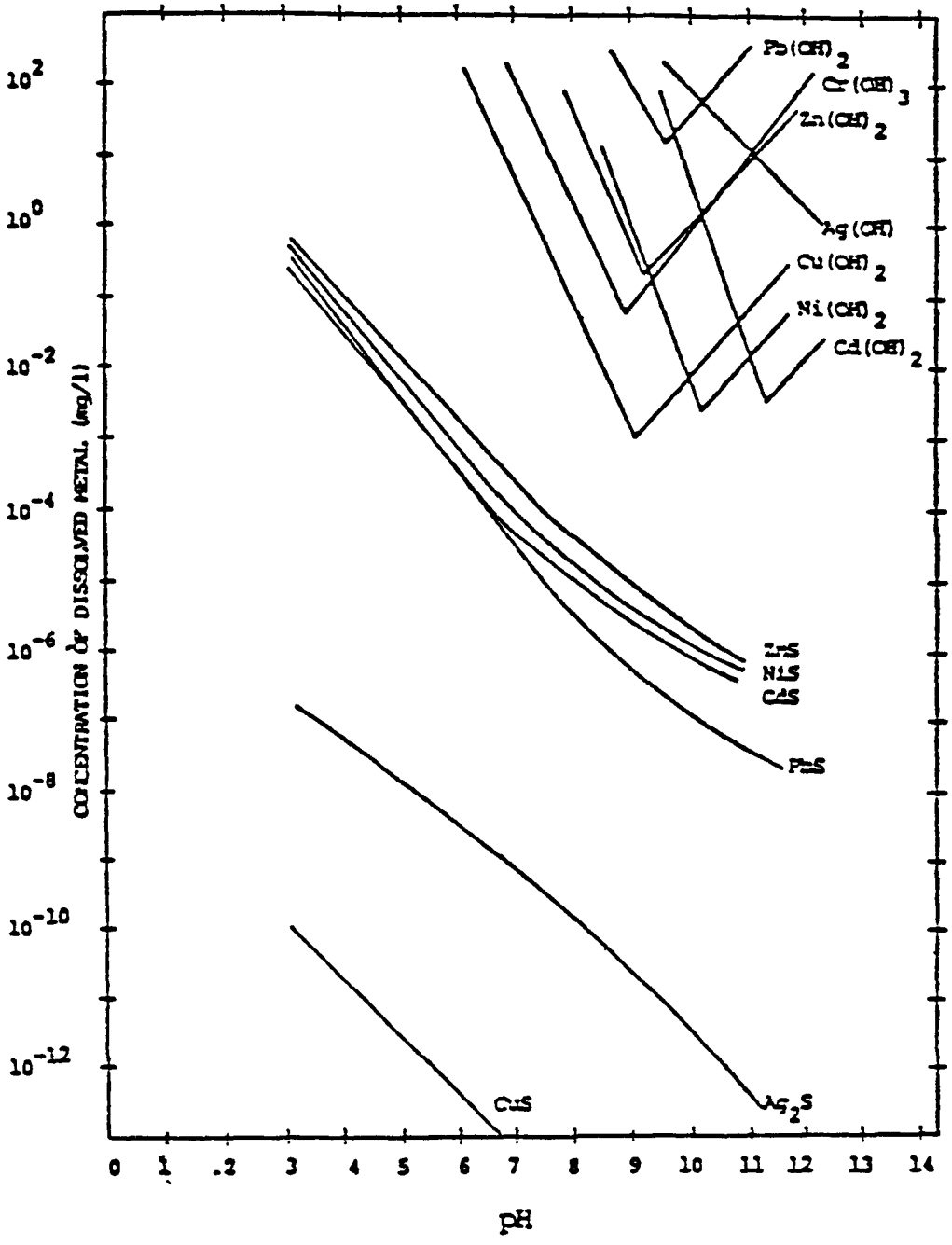
Precipitation is, therefore, the process of introducing to a solution of metal ions a particular anionic specie to form insoluble salts of those metal ions, thus removing them from solution. The choice of the particular anionic species that will most effectively precipitate pollutant metals, however, is rarely straightforward. Industrial waste streams often contain many ions, each of which has a specific solubility product. Additionally, industrial wastewaters often contain species that form water-soluble complexes with metal ions, thus increasing their resistance to precipitation. Amphoteric metals, berryllium, cadmium, chromium, copper, lead, nickel, and zinc, form stable, solvated complexes at both high and low pH. There is therefore a narrow range of solution pH for each metal in which hydroxide precipitation is most effective as shown in Figure E-5.

Four types of chemicals are widely used industrially as precipitants: hydroxides, ferrites, sulfides and xanthates. Metal hydroxides are the most widely used precipitants. Typically lime [$Ca(OH)_2$] and caustic soda (NaOH) are used. Though hydroxide precipitation is a relatively simple and inexpensive procedure, it is limited by its effectiveness in metal removal: soluble metallic complexes form at high pH and, in general, metal concentrations range from several parts per billion to parts per million. Other disadvantages are that hydroxide precipitates tend to form stable colloidal suspensions and that hydroxide precipitate sludge is bulky and presents a disposal problem.

Ferrite coprecipitation can be used to precipitate zinc, cadmium, copper, nickel, lead, and chromium from acidic wastewater. In one ferrite coprecipitation procedure, a ferrous salt is added to the heavy metal containing waste stream; the stream is then neutralized and the resulting heavy metal ferrous hydroxide precipitate oxidized to the stable ferrite coprecipitate. Large particles are formed by this procedure and the resulting sludges are stable enough for safe landfill disposal. Another more energy-intensive ferrite-coprecipitation procedure has been used in Japan primarily for removing

FIGURE E-5

METAL SULFIDE AND HYDROXIDE SOLUBILITIES
AS A FUNCTION OF pH



chromium from acidic waste streams. Chromium is electrolytically reduced from Cr(VI) to Cr(III) in the presence of iron salts and forms insoluble chromium ferrite (iron chromite). The magnetized ferrites can be recovered from sludge by magnetic separation.

Sulfide precipitation is similar to hydroxide precipitation in principle and procedure. Figure E-5 and Table E-22 show that for the metal priority pollutants cadmium, copper, lead, mercury, silver, nickel, and zinc, the metal sulfide is considerably less soluble than the metal hydroxide; additionally, soluble metallic complexes are not formed at high pH. Therefore, adding sulfide ions to a metal-bearing waste stream will precipitate larger quantities of metal from solution than will hydroxide ions, and will result in an effluent containing significantly less metal. Despite this advantage, hydroxide precipitation is more common because lime and caustic soda are less expensive than sodium or ferrous sulfide, and also because sulfide forms hydrogen sulfide (a severe acute health hazard) if introduced to an acidic stream. Thus use of sulfide precipitation is largely limited to a polishing step following hydroxide precipitation, when effluent concentrations below those obtainable by hydroxide precipitation are required. Treatment systems for sulfide precipitation and for hydroxide precipitation are similar, generally consisting of a pH adjustment tank, a flash mixer, a flocculator, settling units with flash storage, and a dual media filter. A pH adjustment to pH 7 to 8 is critical because of the risk of hydrogen sulfide generation.

TABLE E-22

SOLUBILITY PRODUCTS OF TRACE METALS AS HYDROXIDES, SULFIDES AND XANTHATES

Metal	Solubility Product Constant ($-\log K_{sp}$)		
	Metal Hydroxide	Metal Sulfide	Ethyl Xanthate
Cadmium, Cd	13.6	26.1	13.6
Copper, Cu	18.6	35.2	
Ferrous, Fe^{+2}	15.3	16.9	7.1
Lead, Pb	16.1	26.6	16.9
Mercury, Hg	25.4	52.2	37.8
Nickel, Ni	14.8	25.7	11.9
Zinz, Zn	15.7	25.2	8.3
Chromium (VI), Cr^{+6}	8.9	-	

Xanthate precipitation combines aspects of ion exchange with a chemical precipitation process. Xanthates are long-chain starch molecules that bear functional groups capable of forming insoluble complexes with metals. They can be generated by mixing starch or cellulose with carbon disulfide in a caustic medium. Three types of xanthates have been studied: soluble starch xanthate with a cationic polymer, insoluble starch xanthate and fibrous cellulose xanthate. These were tested for their ability to remove cadmium, chromium(III), copper, lead, mercury, nickel, silver, and zinc. In general, xanthates were found to be effective in removing metals over a wide pH range, from 3 to 11, with optimum performance between pH 7 and 9. The studies also concluded

that while cellulose xanthate and starch xanthate were similarly effective in removing trace metals, cellulose xanthate is superior to starch xanthate in terms of sludge settling characteristics, filterability, and handling. Xanthate may also be used as a complexing agent to prevent insoluble hydroxides of amphoteric metals from forming soluble anions as the pH of the stream changes.

Xanthate precipitation, however, is a new technology; reagents, therefore, are not yet available in commercial quantities, and data has not been gathered on dosage rates in continuous flow xanthate precipitation operations. Table E-23 presents qualitative characterizations of the behavior of priority pollutants during treatment by precipitation.

TABLE E-23

QUALITATIVE CHARACTERIZATIONS OF PRIORITY POLLUTANT METAL IONS FOR PRECIPITATION

BERYLLIUM:	Be^{3+}	hydroxide is amphoteric, sulfide decomposes in aqueous solution, sulfate is water soluble
CHROMIUM:	Cr^{+3}	"hydroxide" is hydrated oxide, is amphoteric and dissolves in excess strong base, approximate solubility = 0.00064 mg/liter. Sulfide cannot be made in aqueous solution, sulfate is water soluble
ANTIMONY:	Sb^{+3}	oxide is amphoteric, sulfide can be formed in acid solution (to pH 6) with solubility 0.0018 g/liter (1.3 mg/liter Sb), sulfide is soluble in neutral to alkaline solutions and solutions with excess alkali sulfide
ZINC:	Zn^{+2}	amphoteric hydroxide gelatinous precipitate when formed in aqueous solution. K_{sp} of ZnS = 2×10^{-14}
SELENIUM:	Se^{+4}	oxide is very water soluble, sulfide insoluble in water, but dissolves in excess sulfide reagent
ARSENIC	As^{+3}, As^{+5},	oxides soluble in water, As (V) sulfide water solubility 0.0014 g/liter (0.6 mg/liter As) As(III) sulfide water solubility 0.0005 g/liter (0.3 mg/liter As). Both sulfides soluble at pH 6 and in excess alkali metal sulfide

Coagulation-Flocculation

Coagulation-flocculation is a physical treatment process designed to remove suspended particulate matter known as colloids and as such is an integral part of metal removal processes. Colloids exist because solids in water are always electrically charged; one side of the solid-water interface assumes a positive or negative net electrostatic charge. This causes an equivalent number of oppositely charged ions to form a diffuse layer in the aqueous phase immediately surrounding the metal particle. The electrostatic repulsion between the diffuse outer layers surrounding the metal particles keeps the metal particles in colloidal suspension and precludes their agglomeration and subsequent precipitation. Removing the metal species from colloidal suspension requires that the charged cloud surrounding the metal ion be destabilized.

Coagulation is, therefore, the process of destabilizing colloids suspended in the waste stream by neutralizing the repulsive forces between them. This destabilization is carried out by adding certain ionic species known as chemical coagulants--generally low molecular weight salts of multivalent inorganic ions, usually aluminum salts, iron salts or polyelectrolytes--and then gently stirring the suspension to facilitate contact between the newly destabilized colloids. The result of the coagulation process is that the colloidal particles agglomerate into flocs.

Adding charged species to the waste stream destabilize colloids in two ways. First, raising the electrolyte concentration in the aqueous medium lowers the diffuse outer layers surrounding each metal particle, so the range of electrostatic repulsion decreases and the short-range electrostatic attractive forces take over. Because this phenomenon results from simple electrostatic attraction, the minimum coagulant concentration required to destabilize the colloid is independent of the chemical composition of the colloid. Therefore, quantitative data are not available to describe the response of each individual priority pollutant to coagulation and flocculation. Second,

cations can bring the negatively charged layers surrounding each metal ion closer together by bridging them electrostatically. Multivalent cations are especially effective in bridging heavy metal colloids; a trivalent ion may be 1,000 times as effective as a monovalent ion.

Flocculation is the process of getting these flocs to coalesce still further to form settleable agglomerates. Like coagulation, this process involves adding flocculants--cations that bridge small flocs together by bridging the negatively charged layers surrounding each floc--and then mixing the suspension intensely but not violently.

The two processes, therefore, involve the same procedure: ion addition followed by mild agitation. Alum salts and iron salts are widely used both as coagulants and as flocculants; cationic polyelectrolytes can be used as coagulants, but they are especially effective as flocculants because they are long molecules containing multiple ionic groups and are therefore structurally ideal for bridging flocs. Agitation is generally accomplished by slow stirring with long thin blades; a coagulation/flocculation unit is usually a tank with blades arranged inside as stators and rotors, equipped with meters to dispense measured quantities of coagulants and flocculants. Table E-24 presents a summary (EPA, 1979c) of several coagulation-flocculation-precipitation processes and their effectiveness in treating the metal priority pollutants. Table E-25 presents typical performances for some of these treatment processes as 30-day average effluent metal concentrations.

Ion Exchange

Ion exchange removes metal ions from water by transferring them to a solid material, the ion exchanger, which accepts these undesirable species at acidic or basic exchange sites, giving back to the aqueous phase an equivalent number of a similarly charged species (usually H^+ or OH^-) stored on the ion exchanger skeleton. When the exchange sites become saturated with the undesirable ions, the ion exchanger is washed

TABLE E-24

PERFORMANCE DATA SUMMARIES
ANTIMONY AND ARSENIC REMOVAL

Treatment Technology	pH	Initial Concen-tration (mg/l)	Final Concen-tration (mg/l)	Removal (%)
Antimony				
Lime/Filter	11.5	0.6	0.4	28
Ferric chloride/Filter	6.2	0.5	0.2	65
Alum/Filter	6.4	0.6	0.2	62
Arsenic				
Lime Softening	-	0.2	0.03	85
Sulfide/Filter	6-7	-	0.05	-
Lime (260 mg/l)/Filter	10.0	5.0	1.0	80
Lime (600 mg/l)/Filter	11.5	5.0	1.4	72
Ferric sulfate	5-7.5	0.05	0.005	90
Ferric sulfate	6.0	5.0	0.5	90
Lime/Ferric Chloride/ Filter	10.3	3.0	0.05	98
Activated alumina (2 mg/l)	6.8	0.4-10	<0.4	96-99+
Activated carbon (3 mg/l)	3.1-3.6	0.4-10	<4.0	63-97
Ferric Chloride	-	0.3	0.05	98
Ferric Chloride	-	0.6-0.9	<0.13	-

TABLE E-24 (Continued)

BERYLLIUM AND CADMIUM REMOVAL

Treatment Technology	pH	Initial Concentration (mg/l)	Final Concentration (mg/l)	Removal (%)
Beryllium				
Lime/Filter	11.5	0.1	0.006	99.4
Cadmium				
Lime (260 mg/l)/Filter	10.0	5.0	0.25	95
Lime (600 mg/l)/Filter	11.5	5.0	0.10	98
Lime Softening	5-6.5	0.44-1.0	0.008	92-98
Lime/Sulfide	8.5-11.3	0.3-10	0.006	98+
Ferrous Sulfide (Sulfex)	8.5-9.0	4.0	<0.01	99+
Ferrite coprecipitation/ Filter	neutral	240	0.008	99+

TABLE E-24 (Continued)

CHROMIUM III AND CHROMIUM VI REMOVAL

Treatment Technology	pH	Initial Concentration (mg/l)	Final Concentration (mg/l)	Removal (%)
Chromium				
Lime (260 mg/l)/Filter	10.0	5.0	0.1	98
Lime (600 mg/l)/Filter	11.5	5.0	0.1	98
Reduction/Lime	7-8	140 (as Cr VI)	1.0	—
Reduction/Lime	7-8	1300 (as Cr VI)	0.06 Cr III	—
Lime Softening	10.6-11.3	—	0.15	98+
Lime/Filter	7-9	—	0.05	—
Lime	9.5	15	0.1	—
Lime	9.5	3.2	<0.1	—
Ferrite coprecipitation/ Filter	—	25	0.01	—
Ferric sulfate	6.5-9.3	—	—	98+
Ferric sulfate/Filter	—	5.0	0.05	99
Chromium VI				
Activated carbon (pulverized, Pittsburgh type PC)	3.0	10	1.5	85
Same as above	2.0	10	0.4	96
Activated carbon (granular)	6.0	3	0.05	98
Ferrite coprecipitation	—	0.5	not detectable	—
Sulfur dioxide reduction	—	—	0.01-0.1	—
Bisulfite reduction	—	—	0.05-1.0	—

TABLE E-24 (Continued)

COPPER REMOVAL

Treatment Technology	pH	Initial Concen-tration (mg/l)	Final Concen-tration (mg/l)	Removal (%)
Lime/Filter	8.5-9.0	3.2	0.07	98
Lime (260 mg/l)/Filter	10.0	5.0	0.4	92
Lime (600 mg/l)/Filter	11.5	5.0	0.5	91
Ferric sulfate/Filter	6.0	5.0	0.3	95
Lime	>8.5	10-20	1-2	90
Lime	9.5	3.0	0.2	93
Alum	6.5-7.0	3.0	0.2	93
Lime/Sulfide	5.0-6.5	50-130	<0.5	-
Ferrous sulfide (Sulfex)	8.5-9.0	3.2	0.02	99
Ferrous sulfide (Sulfex)	8.5-9.0	4.0	0.01	99+
Ferrite Coprecipitation/ Filter			0.01	99+

TABLE E-24 (Continued)

LEAD REMOVAL

Treatment Technology	pH	Initial Concentration (mg/l)	Final Concentration (mg/l)	Removal (%)
Lime (260 mg/l)	10.0	5.0	0.25	95.0
Lime/filter	8.5-9.0	189	0.1	99.9
Lime (260 mg/l)/Filter	10.0	5.0	0.075	98.5
Lime (600 mg/l)/Filter	11.5	5.0	0.10	98.0
Ferrous sulfate/Filter	6.0	5.0	0.075	98.5
Sodium hydroxide (1 hour settling)	5.5	—	1.6	—
Sodium hydroxide (24 hour settling)	7.0	—	0.04	—
Sodium hydroxide/Filter	10.5	1700	0.60	99+
Sodium carbonate/Filter	10.1	1260	0.60	99+
Sodium carbonate/Filter	6.4-8.7	10.2-70.0	0.2-3.6	82-99+
Sodium carbonate/Filter	9.0-9.5	5.0	0.01-0.03	99+
Ferrous sulfide (Sulfex)	8.5-9.0	189	0.1	99.9
Ferrite coprecipitation/ Filter	—	480	0.01-0.05	99.9

TABLE E-24 Continued)

MERCURY II REMOVAL

Treatment Technology	pH	Initial Concen-tration (mg/l)	Final Concen-tration (mg/l)	Removal (%)
Sulfide	-	0.3-50.0	0.01-0.12	-
Sulfide	10.0	10.0	1.8	96.4
Sulfide/Filter	5.5	16.0	0.04	99
Sulfide/Filter	4.0	36.0	0.06	99.8
Sulfide/Filter	5.8-8.0	0.3-6.0	0.01-0.125	87-99.2
Ferrite coprecipitation/ Filter	-	6.0-7.4	0.001-0.005	99.9
Activated Carbon	-	0.01-0.05	<0.0005	-
Activated Carbon/Alum	-	0.02-0.03	0.009	-
Activated Carbon	-	0.06-0.09	0.006	-

TABLE E-24 (Continued)

NICKEL REMOVAL

Treatment Technology	pH	Initial Concentration (mg/l)	Final Concentration (mg/l)	Removal (%)
Lime	8.5-9.0	75	1.5	98
Lime (260 mg/l)/Filter	10.0	5.0	0.3	94
Lime (600 mg/l)/Filter	11.5	5.0	0.15	97
Caustic Soda/Filter	11.0	-	0.3	-
Ferrous sulfide (Sulfex)	8.5-9.0	75	0.05	99.9
Ferrite coprecipitation	-	1000	0.20	99.9

TABLE E-24 (Continued)

SILVER REMOVAL

Treatment Technology	pH	Initial Concen-tration (mg/l)	Final Concen-tration (mg/l)	Removal (%)
Sodium hydroxide	9.0	54	15	72
Ferric sulfate (30 mg/l)	6-9	0.15	0.03-0.04	72-83
Lime Softening	9.0-11.5	0.15	0.01-0.03	80-93
Chloride precipitation (alkaline chlorination in the presence of cyanide)	-	105-250	1.0-3.5	97+
Ferric chloride/Filter	6.2	0.5	0.04	98.2
Sulfide precipitation	5-11	-	-	very high

TABLE E-24 (Continued)

SELENIUM AND THALLIUM REMOVAL

Treatment Technology	pH	Initial Concentration (mg/l)	Final Concentration (mg/l)	Removal (%)
Selenium				
Ferric chloride/Filter	6.2	0.1	0.03	75
Ferric chloride/Filter	6.2	0.05	0.01	80
Alum/Filter	6.4	0.5	0.26	48
Ferric sulfate	5.5	0.10	0.02	82
Ferric sulfate	7.0	0.10	0.03	75
Lime/Filter	11.5	0.5	0.3	35
Lime/Filter	11.5	0.06	0.04	38
Thallium				
Lime/Filter	11.5	0.5	0.2	60
Ferric chloride/Filter	6.2	.0.6	0.4	30
Alum/Filter	6.4	0.6	0.4	31

TABLE E-24 (Continued)

ZINC REMOVAL

Treatment Technology	pH	Initial Concentration (mg/l)	Final Concentration (mg/l)	Removal (%)
Lime/Filter	8.5-9.0	3.6	0.25	93
Lime (260 mg/l)	10.0	5.0	0.85	83
Lime (260 mg/l)/Filter	10.0	5.0	0.80	84
Lime (600 mg/l)	11.5	5.0	0.35	93
Lime (600 mg/l)/Filter	11.5	5.0	1.2	77
Lime/Filter	-	16	0.02-0.23	-
Sodium hydroxide	9.0	33	1.0	97
Sulfide	-	42	1.2	97
Ferrous sulfide (Sulfex)	8.5-9.0	3.6	0.02	99+
Ferrite coprecipitation	-	18	0.02	99+

TABLE E-25

ESTIMATED ACHIEVABLE MAXIMUM 30-DAY AVERAGES FOR THE APPLIED TECHNOLOGIES

	Final Concentrations (mg/l)						
	Lime Settling	Lime Filter	Sulfide Filter	Ferrite Coprecip-itation Filter	Soda Ash Settling	Soda Ash Filter	Alum
Antimony, Sb	0.8-1.5	0.4-0.8					
Arsenic V	0.5-1.0	0.5-1.0	0.05-0.1				
Beryllium, Be	0.1-0.5	0.01-0.1					
Cadmium, Cd	0.1-0.5	0.05-0.1	0.01-0.1	<0.05			
Copper, Cu	0.5-1.0	0.4-0.7	0.05-0.5	<0.05			
Chromium III, Cr^{+3}	0.1-0.5	0.05-0.5		0.01			
Lead, Pb	0.3-1.6	0.05-0.6	0.05-0.4	0.20	0.4-0.8	0.1-0.6	
Mercury II, Hg			0.01-0.05	<0.01			
Nickel, Ni	0.2-1.5	0.1-0.5	0.05-0.5				
Silver, Ag	0.4-0.8	0.2-0.4	0.05-0.2				
Selenium, Se	0.2-1.0	0.1-0.5					
Thallium, Tl	0.2-1.0	0.1-0.5					0.2-0.5
Zinc, Zn	0.5-1.5	0.4-1.2	0.02-1.2	0.02-0.5			

TABLE E-25 (Continued)

	Final Concentrations (mg/l)					
	Ferric Chloride	Activated Carbon	SO_2 Reduction	Bisulfite Reduction	Lime/$FeCl_2$ Filter	Alkaline Chlorination
Arsenic V, As	0.05-0.5	0.3			0.02-0.1	
Chromium VI, Cr^{+6}		0.1	0.01-0.1	0.05-0.5		
Mercury II, Hg		0.01				
Silver, Ag	0.05-0.1					
Selenium, Se	0.05-0.1					
Thallium, Tl	0.7					
Cyanide (Free), CN^-						0.1-0.5

with regenerant, a strong solution of the ion originally present on the resin--usually mineral acid or caustic soda. The pollutant species accumulated on the resin are replaced by the original species of ions from the regenerant and the exchanger is returned to its original usable condition.

The ion exchanger is almost always a three-dimensionally cross-linked polymer resin to which particular ionic function-alities are attached. There are four categories of ion-exchange resin, each with a characteristic set of functional groups, which interact most strongly with a particular type of charged species. First are the strongly acid cation-exchange resins. Their general formula is $Res\text{-}SO_4\text{-}H^+$ where "Res" represents the polymeric resin structure. Most typically, strongly acid-cation exchange resins consist of polystyrene sulfonic acid cross-linked with divinylbenzene. Second, there are weakly acidic cation-exchange resins: $Res\text{-}CO_2\text{-}H$. These are generally polyacrylic acid or polymethacrylic acid cross-linked with divinylbenzene. Third, there are strongly basic anion-exchange resins: $Res\text{-}NR_3\text{-}OH$, where R is an aliphatic or aryl-aliphatic radical. Strongly basic anion-exchange resins generally are polyvinylbenzyl trimethyl-ammonium hydroxide cross-linked with divinylbenzene. And fourth, there are weakly basic anion-exchange resins: $Res\text{-}NR_2$. Several varieties of these are available; all contain tertiary aliphatic or aryl-aliphatic amine function-alities on resin matrices ranging from the polystyrene type through polyacrylate to aliphatic polyamine condensation products.

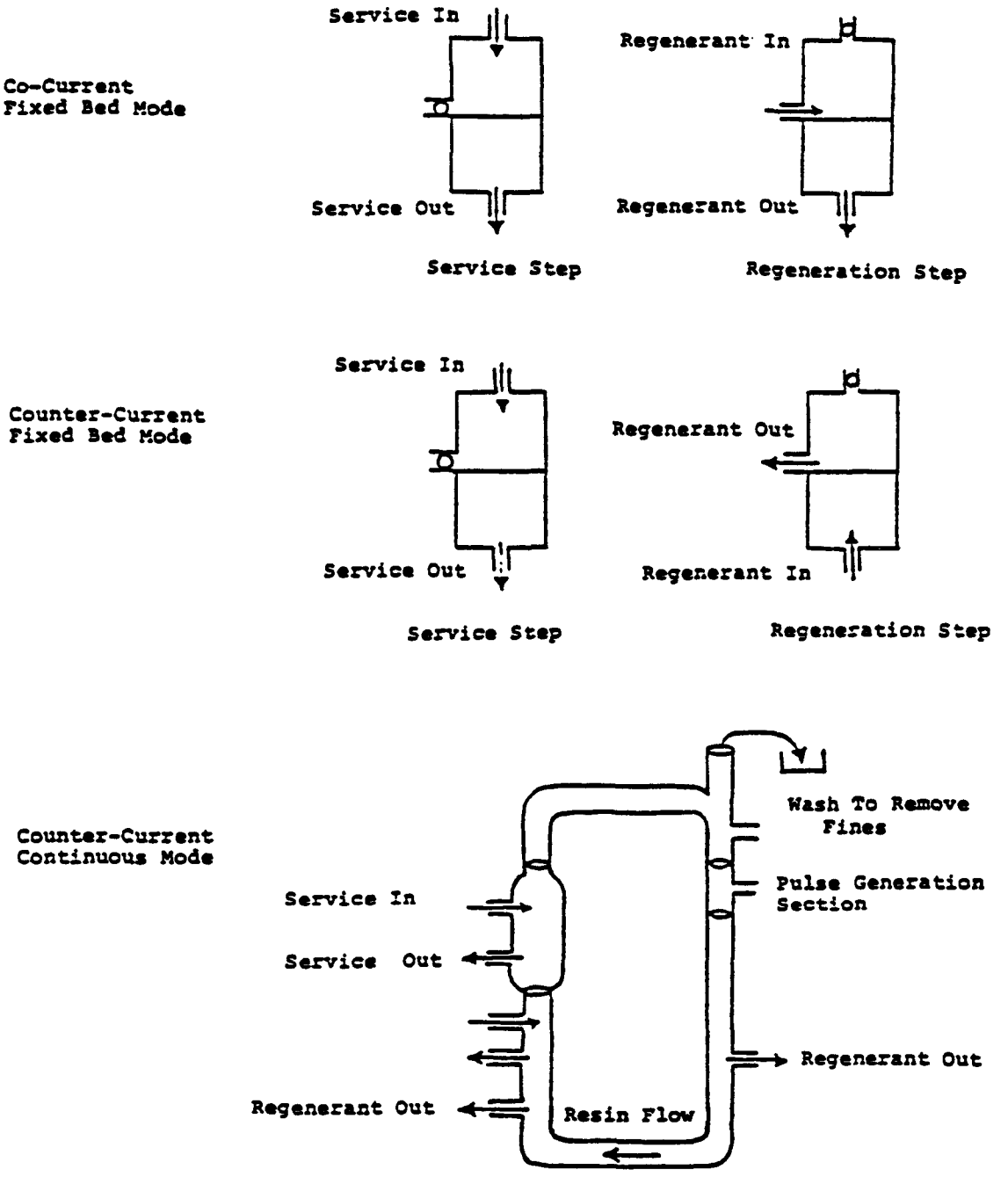
The selectivity of a particular resin is a function of the size and charge of the ions to be exchanged--an exchange resin prefers highly charged multivalent ions. Knowledgeable choice of a particular ion exchange material from the wide range of selective resins commercially available can often allow selective separation of one ion from another, allowing selective removal of an undesirable ion from a stream bearing many other ions. The affinity of a particular ion exchange resin for a particular ion is a function of several factors, including ion size and charge, the composition of the waste

stream, and the functional group and polymer structure of the resin. Extensive commercial literature is available for the engineer intending to design an ion-exchange system to remove particular metals from a particular waste stream.

Commercially available ion exchangers operate in one of two modes: fixed bed or continuous. (Figure E-6 presents their flow diagrams.) Fixed bed units perform in a four-operation cycle. First, service wastewater flows through the ion exchange unit until the point of exhaustion is reached where all available ion-exchange sites are occupied by pollutant ions. Second, backwash water is pumped through the bed in the direction opposite to that of the waste stream during the service phase. Third, regeneration occurs in which a strong solution of the ion originally occupying the exchange sites on the resin is pumped through the bed to dislodge the pollutant ions and return the resin to its original composition. And fourth, the resin is rinsed with water. The backwash phase is necessary to flush out extraneous particles from between the resin beads. Fixed bed units can operate cocurrently--the regenerant is pumped through the unit in the same direction the waste stream flowed-or countercurrently--the regenerant flows in the opposite direction from the waste stream. Countercurrent regeneration is more effective than cocurrent regeneration because the maximum pollutant sorption occurs where the waste stream enters the ion-exchange unit. Sorption sites become progressively less occupied by pollutant ions along the path of the waste stream through the unit. Therefore, the countercurrent method is better because it brings fresh regenerant into contact with the part of the resin bearing the fewest pollutant ions and as the regenerant proceeds through the unit and becomes less concentrated, the resin along the path of the regenerant becomes more heavily occupied with contaminant ions--therefore a mass ratio regenerant ion:sorbed metal ion favorable to regeneration is maintained all along the regenerant path.

FIGURE E-6

ION EXCHANGE BED CONFIGURATIONS



Continuous ion-exchange operations are run countercurrently. Figure M-6 shows how ion-exchange resin beads are circulated through a loop. One segment of the loop is the service segment, through which wastewater to be treated is sent, and another section is the regeneration segment, through which regenerant is passed in a direction opposite to the direction the wastewater took.

Since ion exchange is basically a method for tranferring pollutant ions from the waste stream to the regenerant solution, there arises the problem of disposing of the spent regenerant. In some cases, it is economical to recover the metal pollutant from the regenerant. Disposal or recovery is simplified when the volume of regenerant is minimized. Many fixed bed units minimize regenerant volume with the "staged" or "proportional" regeneration technique. The first part of the regenerant leaving the ion bed is the most enriched in the pollutant species being removed. This portion is sent to treatment or disposal. The second portion of regenerant to leave the ion-exchange bed leaves with a significantly lower pollutant concentration. It is stored and used as the first portion of regenerant in the next service regeneration cycle.

Ion exchange removes metal priority pollutants with outstanding efficiency. Table E-26 summarizes the results of treatability studies on ion exchange removal of priority pollutants. Most removal efficiency percentages are in the high nineties. Table E-24 summarizes ion exchange removal efficiencies for each metal priority pollutant obtained in treatability studies with industrial wastewaters and synthetic solutions.

One chemical company has prepared a summary of treatment and cost data for an industrial ion-exchange system, treating a chromium-bearing waste, which it considers to represent BAT for chromium removal. The waste stream is a cooling tower blowdown, containing chromium added to inhibit growth of fungi and algae.

TABLE E-26

TREATABILITY STUDIES SUMMARY FOR PRIORITY POLLUTANT REMOVAL WITH ION EXCHANGE

METAL	DATA POINTS	EFFLUENT CONC., mg/l MEAN	MED	MIN	MAX		REMOVAL % MEAN	MED	MIN	MAX	
As	19	1.65	0.60	0.0	8.0		87.1	96.5	21	100	*
Cd	17	0.019	0.0003	0.0001	0.1		96.8	99.9+	75	99.9+	*
Cr	12	0.36	0.05	0.01	1.8	(11)	96.7	99.5	88	99.9	
Cu	3	1.8	2.0	0.5	3.0	(4)	96.5	97	93	99+	*
Pb	2	0.03	-	-	-		99.85	-	-		
Hg	5	0.0005	0.0001	0	0.002		99.9+	99.9+	99.9+	100	*
Ag	5	3.5	0.14	0.01	6.5	(7)	93.7	95	90	100	*
Zn	7	2.2	0.4	0	10	(9)	97.7	99	90	100	*

*Data set includes results from synthetic solutions.

() = Data points used for that computation.

This application for ion exchange is relatively new technology. Blowdown is filtered and pH adjusted before passing through weak base anion exchange vessels for chromium removal and then weak acid cation exchangers for zinc and trivalent chrome removal. Upon regeneration of the resins, chrome and zinc can be recovered and recycled back to the cooling towers eliminating a large percentage of the make-up chrome and zinc solutions. Another advantage of ion exchange is the elimination of voluminuous metal sludges formed in the precipitation technique commonly employed for chrome-and-zinc removal in cooling tower blowdown.

Figure E-7 is the flow diagram of the system. Table E-27 summarizes operating parameters and removal data. Tables E-28 and E-29 present daily and rolling average chromium effluent concentrations.

The ion exchange units were installed in January 1978 and have consistently met the NPDES permit. Installed costs were $4.7 million. The operating costs for the first quarter of 1981 were approximately $80,000/month. This figure includes utilities services, wages, salary and payroll overhead, maintenance, chemical requirements, laboratory analyses, technical engineering services, catalyst (resin), fixed indirects, and unit depreciation. Assuming an average flow of 650 gpm, at 8 ppm Cr^{tot} removal across the unit, this comes to roughly $43/pounds of Cr^{tot} or $2.85/1,000 gallons.

A survey of published ion exchange treatability data is presented in Tables M-30 These data were compiled by Calmon, Casana, and Gold of Water Purification Associates (1980); many of these represent results obtained with synthetic solutions.

Minor drawbacks to the application of ion exchange to industrial waste treatment exist. They include the problems of spent regenerant disposal, susceptibility to damage by high temperatures and strong oxidants, and the tendency to contamination by organic matter present in the waste stream. Furthermore, suspended organic solids will foul the resin and aromatic molecules will be irreversibly adsorbed onto the resin.

FIGURE E-7 FLOW DIAGRAM FOR CHROMIUM AND ZINC REMOVAL SYSTEM

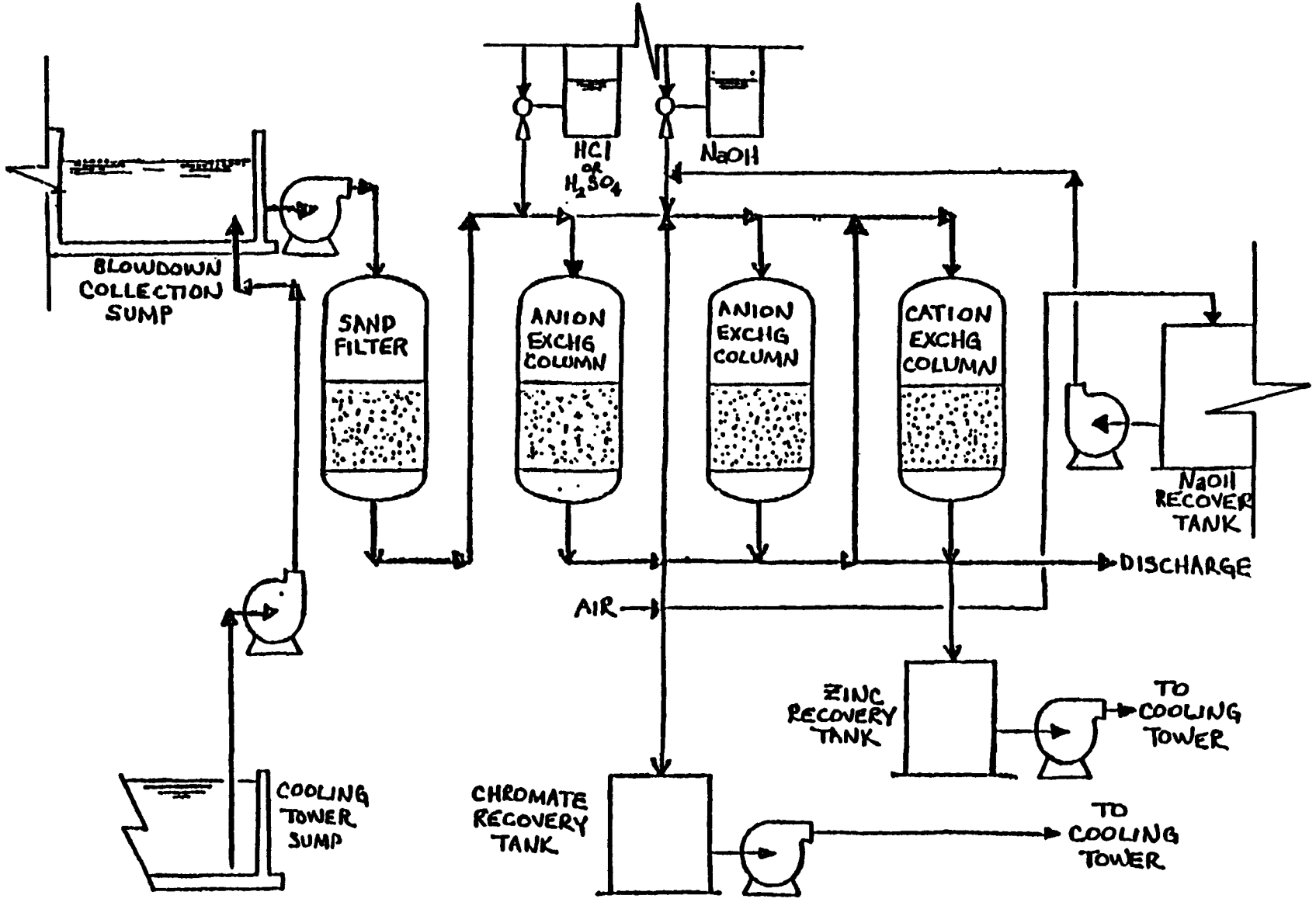


TABLE E-27

CHROMIUM AND ZINC REMOVAL SUMMARY

Treatment Technology: Ion Exchange

Data Source: Cooling Tower Blowdown
Point Source Category: Organic Chemicals
Subcategory:
Plant:
References:

Data Source Status:
Engineering estimate ____
Bench scale ____
Pilot scale ____
Full scale __x__

Use in System: Cr Removal and Reuse
Pretreatment of Influent: Dual Media Filtration (Anthracite/Sand, pH adjustment

Design or Operating Parameters'

Wastewater Flow: 400 gpm Avg; 1000 gpm avg design 1500 gpm max design
Solids Loading Rate: 0.50 lb/day/ft^2 based on 400 gpm @ 40 ppm TSS
Bed Height: Anion 44" Cation 36"
Pressure Drop:
Resin Type: Anion Rohm & Haas IRA-94 Cation Rohm & Haas DP-1
Avg. Run Length: Anion 1 regeneration/day Cation 1 regeneration/3 days (based on 400 gpm)
Regenerant Used: 5% HCl 5% NaOH
Cycle Time: 8 hrs/regeneration time
Backwash Rate:
Resin Pulse Volume:
Unit Configuration: Dual Media Filtration, pH adjustments, Anion Exchange (Chrome Removal), Cation Exchange (Zinc Removal)

REMOVAL DATA

Sampling period: 7/1/79 - 7/31/80

Pollutant/Parameter	Concentration, mg/l Influent	Effluent	Percent Removal
Toxic pollutants: Chromium			
Mean Concentration	10.94	.48	94
99th percentile	36.00	2.58	99
95th percentile	21.78	1.20	99
90th percentile	17.31	0.80	98

TABLE E-28

DAILY CHROMIUM AVERAGES
Plant Operating Data 7/1/79 through 7/31/79

DATA SET	NUMBER OF DATA POINTS	AVG. FLOW (gpm)	DAILY AVERAGES (mg/l) MEAN	MEDIAN	MIN	MAX	99%	95%	90%
Influent Cr^{tot}	473	395	10.94	9.8	0.41	42.00	36.00	21.78	17.31
Effluent Cr^{tot}	473	395	0.48	0.38	0.07	6.74	2.58	1.2	0.8
% Removal Cr^{tot}	473	395	94	96	22	99	99	99	98

TABLE E-29

THRITY DAY CHROMIUM ROLLING AVERAGES
Plant Operating Data 7/1/79 through 7/31/79

30 DAY ROLLING AVERAGE (mg/l)

DATA SET	NUMBER OF DATA POINTS	AVG. FLOW (gpm)	MEAN	MEDIAN	MIN	MAX	99%	95%	90%
Influent Cr^{tot}	362	386	11.32	9.76	7.72	25.13	24.89	22.21	17.29
Effluent Cr^{tot}	362	386	0.49	0.48	0.34	0.72	0.70	0.64	0.60
% Removal Cr^{tot}	362	386	95	95	92	98	98	97	97

TABLE E-30

POLLUTANT REMOVAL SUMMARIES
ARSENIC REMOVAL SUMMARY

Source	As conc (mg/l) Influent	As conc (mg/l) Effluent	Removal (%)
Potable Well	1.06	0.01	91
Potable Well	0.84	0	100
Potable Well	0.09	0	100
Synthetic Drinking Water	0.06	0.003	99.5
"	1.06	0.17	21
"	24.7 *	0	100
"	60.1 *	0.6	99
"	104 *	3.0	97
"	12.8 *	0.5	96.5
"	25.4 *	1.9	92.4
"	34.7 *	5.7	83.5
"	6.54**	1.33	79.6
"	13.4 **	2.3	73.0
"	26.7 **	8.0	70.0
"	6.75**	0.7	88.8
"	13.4 **	2.3	83.0
"	26.7 **	5.5	79.3
Geothermal	100 *	0	100
	100 **	0	100

Combined Data

Source	Data Points	As effluent conc. (mg/l) mean	med	min	max	As Removal (%) mean	med	min	max
Potable Well	3	0.003	0	0	0.01	97	100	91	100
Synthetic	16	1.96	1.01	0	8.0	85.2	90.6	21	100
TOTAL	19	1.65	0.60	0	8.0	87.1	96.5	21	100

TABLE E-30 (Continued)

CADMIUM REMOVAL SUMMARY

Effluent Concentration (mg Cd/l)					Removal (%)				
No. Data Pts.	Mean	Med	Min	Max	No. Data. Pts.	Mean	Med.	Min.	Max.
14	0.0018	0.0002	0.0001	<0.01	14	96.2	99.9+	75+	99.9+
3	0.1	0.1	0.1	0.1	3	99.7	99.8	99.5+	99.9+
17	0.019	0.0003	0.0001	0.1	17	96.8	99.9+	75+	99.9+

Source	Initial Concentration (mg/l Cd)	Other Ions Present	Pretreatment	Volume	Effluent Removal (mg/l Cd)	(%)
$CdSO_4$ Synthetic solution	5	-	pH adj. 8.6	500	0.0001*	99.9+
				1000	0.0002*	99.9+
				1600	0.0003*	99.9+
				3000	0.008*	99.9+
				4800	0.0018*	99.9+
Gas Washing Wastewater	0.039	Hg;Cu	pH adj. S.S. Removal	1000	<0.01**	75+
				2000	<0.01**	75+
W.W. from Photo Plant	1.7	Organics & Inorganic Sales	pH adj. S.S. rem.	100	<0.001	99.9+
WW from Cd Plating	4.6	CN & Inorganics	pH adj. S.S. rem. NaClO rem. CN	100	<0.001	99.9+
WW from Cd Battery Plant	0.02	-	pH adj. 9.0 SS removed	100	0.0002	99
				100	0.0002	99
				300	0.0002	99
Flue gas Treatment Solution	0.8	Hg Cr Pb Inorg.Salts	pH adj. 9.0	1000	0.0002*	99.9+
				3000	0.0003*	99.9+

* Two columns in series.
** Other heavy metals reduced to very low levels.

TABLE E-30 (Continued)

CHROMIUM REMOVAL SUMMARY
Combined Data

Source	Effluent Conc. (mgCr/l)					Removal (%)					Capacity ($HCrO_4^{-2}/ft^3$)				
	No. Data Points	Mean	Med	Min	Max	No. Data Points	Mean	Med	Min	Max	No. Data Points	Mean	Med	Min	Max
Cooling Tower Blowdown	8	0.52	0.075	0.01	1.8	7	94.9	97.7	88	98.8	5	3.5	3.0	2.5	5.5
Electroplating	2	0.017	0.017	0.01	0.025	2	99.85	99.85	99.8	99.9	2	3.8	3.8	1.85	5.75
Pigment Manufacture Waste	1	<0.05	<0.05	<0.05	<0.05	1	99.9+	99.9+	99.9+	99.9+	0	–	–	–	–
Wool Dyeing Wastewater	1	0.05	0.05	0.05	0.05	1	99.6	99.6	99.6	99.6	1	5.6	5.6	5.6	5.6
Total Combined Data	12	0.36	0.05	0.01	1.8	11	96.7	99.5	88	99.9	8	3.8	3.0	1.85	5.75

TABLE E-30 (Continued)

CHROMIUM REMOVAL SUMMARY

Source	Concentration (mg/l as Cr) Influent	Effluent	Removal (%)	Capacity ($\#CrO_4/ft^3$)
Plating Waste (rinse water)	44.8	0.025	99.9	1.7 - 2.0
Cooling Tower Blowdown (Electroplating)	9.8	0.02	99.8	3.0
Electroplating Waste (Rinse Water)	41.6	0.01	99.8	5.2 - 6.3
Cooling Tower Blowdown	17.9	1.8	90	5-6
Cooling Tower Blowdown	-	0.1	-	-
Cooling Tower Blowdown	10	1	90	2.5 - 4.5
Cooling Tower Blowdown	7.4-10.3	1	86-90	-
Cooling Tower Blowdown	20	< 0.05	99.4	-
Cooling Tower Blowdown	10	0.05	99.5	0.6 - 5.2
Wool Dyeing Wastewater	10-20	0.05	99.5-99.75	0.6 - 5.2
Cooling Tower Blowdown (Chemical Complex)	8.96	0.2	97.7	2.51
Pigment Manufacture Wastewater	1210	<0.5	99.9+	-

TABLE E-30 (Continued)

COPPER REMOVAL SUMMARY

Source	Eff (mg/l Cu)	Removal (%)	Capacity (meq/l)
Synthetic	-	-	-
Synthetic	-	-	1220 (pH 4)
Synthetic	-	93	-
Synthetic	0.5	99	1250 (H^+ form
Rayon Wastewater	3.0	99+	1412
Pickle Rinse Soln.	2.0	95	1330

Combined Data

Effluent Cu (mg/l)

No data pts.	Mean	Med	Min	Max
3	1.8	2.0	0.5	3.0

Removal %

No data pts.	Mean	Med	Min	Max
4	96.5	97	93	99+

Capacity (meq/l)

No data pts.	Mean	Med	Min	Max
4	1303	1290	1220	1412

TABLE E-30 (Continued)

LEAD REMOVAL SUMMARY

Source	Inf (mg/l Pb)	Eff (mg/l Pb)	Removal (%)	Capacity
Ammunition Plant Wastewaters	6.5	0.01	99.8	
Synthetic Solution	50	0.05	99.9	4.4 lb/ft^3
-	28.5	0.03	99.85	4.4 lb/ft^3

TABLE E-30 (Continued)

MERCURY REMOVAL SUMMARY

Source Water	Influent (mg/l Hg)	Effluent (mg/l Hg)	Removal (%)	Capacity
Salt Electrodialysis Plant	10	0.002	99.9+	$60x10^3$ to $200x10^3$ mgHg/l resin
Mercury Cell Plant Waste & Brine Solns.	0.01 to 50	<0.0001	99.9+	From 0.4 to 680 mgHg/gm resin, depnd. on influent conc.
Synthetic Solution	125 to 138	0	100	0.1064 gmHg per gm resin
Synthetic Solution	0.1	0	100	550 mgHg per gm dry resin
Chlorine-Caustic	20	0.0005	99.9+	1200 meq/liter of resin

	Combined Data				
	Data Pts	Mean	Med	Min	Max
Effluent Conc. (mg/l Hg)	5	0.0005	0.0001	0	0.002
Removal (%)	5	99.9+	99.9+	99.9+	100

TABLE E-30 (Continued)

SILVER REMOVAL SUMMARY
Combined Data

Influent	Effluent Silver Concentration (mg/l)					Percent Removal					Capacity (meq Ag/l bed)				
	No.Pts	Mean	Med	Min	Max	No.Pts	Mean	Med	Min	Max	No.Pts	Mean	Med	Min	Max
Synthetic Solutions	1*	50	50	50	50	2	95	95	90	100	3	463	300	90	1000
Treated Sewage	2	0.075	0.075	0.01	0.14	2	95.4	95.0	91.7	99.4	0	-	-	-	-
Photographic Processing Wastes	3	5.8	6.5	< 1	10	3	91.6	90	90	95	3	352	80	75	900
All Data Combined	5	3.5	0.14	0.01	6.5	7	93.7	95	90	100	7	563	300	75	1300

*Extremely high influent concentration (500 mg/l).

Deleted from combined data.

TABLE E-30 (Continued)

SILVER REMOVAL SUMMARY

Influent	Effluent Conc. (mg/l Ag)	Removal %	Capacity meq/l bed
Synthetic Soln.	50	90	90
Synthetic Soln.	Not reported	∿100	300-1000
Sand/Carbon Treated Sewage	0.01	99.4	Not reported
Lime/Sand Treated Sewage	0.14	91.7	Not reported
Photographic wastes	10	95	278
Photographic wastes	<1	>90	75
Photographic wastes	6.5	90	900-1300

TABLE E-30 (Continued)

SILVER REMOVAL SUMMARY

Source of Solution	Resin Used	Eluting Agent	Capacity/Comments
Silver Plating	Cation ion exch. + Weak base anion exch.	5% KCN + 5% NaOH	92.9 gm Ag/l anionic resin
Non-ferrous Metal Treatment Cyanide Waste	Anionic		High flow rates
Cyanide Liquor Plating Process	Medium base Anion exchange	Potassium Thiocianates. HCl in methanol. HCl in acetate	
Lab Wastes			Silver reduced to 5ug/l
Color Photography process.	Anion Weak Base WA-21		Quantitative adsorption pH 6.5
Cyanide Plating Waste	Strong Base	NH_4Cl	

TABLE E-30 (Continued)

ZINC REMOVAL SUMMARY

Source	Combined Data														
	Effluent Conc. (mg/l Zn)					Percent Removal					Capacity (meq/l)				
	No. Data Points	Mean	Med.	Min	Max	No. Data Points	Mean	Med	Min	Max	No. Data Points	Mean	Med	Min	Max
Cooling Tower Blowdown	2	0.2	0.2	0	0.4	2	97.6	97.6	95.2	100	2	844	844	109	1,600
Rayon Production Wastewater	1	~0	-	-	-	1	100	-	-	-	1	444	-	-	-
Pickle Liquor From Galvanizing Operation	1	<1000*	-	-	-	1	>97.5	-	-	-	2	2,295	2,295	2,142	2,448
Wash Water From Kaolin Processing	1	<0.4	-	-	-	1	>98.5	-	-	-	1	835	-	-	-
Zinc Phosphate Bonderizing Wastewater	1	10	-	-	-	2	99.45	99.45	99	99.7	1	1,374	-	-	-
Synthetic Solution	2	2.75	2.75	0.5	5.0	2	94.5	94.5	90	99	2	440	440	390	490
Total Combined Data	7	2.2	0.4	0	10	9	97.7	99	90	100	9	598	840	109	2,448

*Extremely high influent concentration (40,000 mg/l).

Deleted from total combined data.

TABLE E-30 (Continued)

ZINC REMOVAL SUMMARY

Source	Effluent Con. (mg/l Zn)	Removal (%)	Capacity (meq/l)
Cooling Tower Blowdown	~0	~100	109-165
Rayon Wastewater	~0	~100	1320
Rayon Wastewater	low*	high*	444**
Pickle Liquor from*** Galvanizing Plant	<1000	>97.5	2142-2448
Washwater from Kaolin Processing Plant	<0.4	>98.5	040
Cooling Tower Blowdown	0.4	95.2	1420-1680
Zinc Phosphate Bonderizing Wastewater	10	99-99.7	1374
Synthetic Solution pH 4	0.5	99	390
pH 7 and 9	5.0	90	490

*No specific value reported. .

**Corresponds to "breakthrough" point.

***Zn in influent = 40 g/l.

Conclusion

The vast majority of waste treatment systems in the organic chemicals and plastics industry use coagulation-flocculation-precipitation, or ion exchange, or both, to remove metal priority pollutants from wastewater. However, other metal-removing systems exist and are used to some extent as in the inorganic chemicals processing industry. These are reduction and oxidation processes, membrane processes (reverse osmosis), and carbon adsorption.

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* References from other sections of the 1981 Contractor's Engineering Report also in this reference list.

APPENDIX F

STATISTICAL DETAILS AND DEVELOPMENT OF VARIABILITY FACTORS

APPENDIX F

STATISTICAL DETAILS AND DEVELOPMENT OF VARIABILITY FACTORS

This Appendix presents the major statistical methodologies and data processing procedures used in the development of the proposed effluent limitations from the OCPSF effluent data. As explained in Section IX, variability factors were determined using organic priority pollutant data from the CMA study and heavy metals data from five BPT Daily Data File plants. Organic priority pollutant data from the Verification study were used in conjunction with the CMA data to determine median long term values for calculating the effluent limitations. The Screening data were used to investigate BAT subcategorizaton. Some elementary formulas and definitions are presented first; subsequent sections discuss the rationale for using daily sample averages to model effluent variability, goodness-of-fit tests, derivation of variability factors, example variability factor calculations, and the statistical methodology used to investigate BAT subcategorization.

A. FORMULAS AND DEFINITIONS

Important formulas and definitions of statistical terms used in this appendix include the following:

1. N - number of valid observations used in a particular analysis (e.g., the total number of valid effluent values at a particular plant for a particular pollutant)

2. Mean - arithmetic average: $\bar{X} = \sum_{i=1}^{N} X_i/N$

3. Variance (unbiased estimate): $S^2 = \frac{1}{N - 1} \sum_{i=1}^{N} (X_i - \bar{X})^2$

(The standard deviation is $S = \sqrt{S^2}$.)

4. Minimum - the smallest value in a set of N observations.

5. Maximum - the largest value in a set of N observations.

6. Range - the minimum subtracted from the maximum

7. Median - the middle value in a set of N observations. If N is odd (N = 2k - 1 for some integer k), the median is the kth order statistic, C(k). If N is even (N = 2k), the median is

$$1/2[C(k) + C(k + 1)].$$

B. RATIONALE FOR USING DAILY SAMPLE AVERAGES IN MODELING EFFLUENT VARIABILITY

In the CMA Five-Plant Study, multiple measurements of organic pollutant concentrations in daily samples were made by one or more laboratories. Thus, several reported values of a specific pollutant concentration were available for a particular daily sample. Because NPDES permits require a single reported value, the Agency considered several alternate approaches for characterizing the variability of the pollutant concentration of individual daily samples. The following approaches were considered.

- Arbitrarily selecting one measurement per day and calculating a variance from the selected measurements

- Calculating daily sample averages of multiple measurements and computing a variance of the results

- Performing a variance component analysis using replicate measurements and adding estimates of relevant components to estimate the variance of a single measurement.

The first alternative was rejected because it does not use all the data and gives different answers depending on which measurements are selected. The second method is a way duplicate measurements can be handled in NPDES reporting; it is more straightforward than the last alternative, which eventually also was rejected. The variance component method is described briefly below.

A variance component analysis of data for a given plant and pollutant characterizes variation in effluent monitoring results at the plant by estimating a variance component for each relevant source of variation. Because of the way the CMA study was conducted, it was possible to estimate variance components for the following sources:

- Within-day variation attributable to short-term replication errors within a laboratory

- Between-day variation attributable to process, treatment, sampling, and longer-term within-laboratory variation

- Between-laboratory variation.

The between-day and between-lab variance components generally are larger than the within-day variance component because many more factors affect between-day and between-laboratory variability.

Although it was possible to estimate a between-laboratory variance component from the CMA data, it was not necessary to do so to characterize effluent variability. In practice, a single laboratory generally performs all monitoring analyses of a given pollutant at a given plant, so between-laboratory differences do not affect observed effluent variation at a plant. (Between-lab differences do contribute to between-plant differences, which are reflected in the observed long-term performance of the industry.)

Given estimates of the within- and between-day variance components σ_w^2 and σ_b^2 for a specific plant and pollutant, the variance of a single measurement is estimated from

$$\sigma^2 = \sigma_w^2 + \sigma_b^2.$$

Estimates of σ_w^2 and σ_b^2 can be obtained from data for a given plant, lab and pollutant using formulas in Linear Models (Searle, 1971).

A comparison of the daily sample average and variance component approaches resulted in selection of the daily sample average because of its relative simplicity, its conformance to general monitoring practice, and its tendency to give conservative variance estimates. The conservativeness of the daily sample average approach results from including measurements from different laboratories in the daily average. This tends to make the variances of daily average data larger than corresponding variance component estimates.

TABLE F-1 shows a comparison of standard deviations estimated from the CMA data by the two approaches. The variance component estimates in the table were based on $\log_e$ (C-10) for individual concentrations (C) above 10 µg/l; the daily sample average estimates were based on $\log_e$ ($\overline{C}$-10) for daily sample averages ($\overline{C}$) above 10. Note that in 8 out of 13 plant-pollutant comparisons, the daily sample average result was larger. The median standard deviation for the daily sample average method was 1.54 compared to 1.37 for the variance component method.

Daily sample averages were computed by averaging replicate measurements within laboratories and then averaging the results across laboratories. Before computing daily sample averages for organic pollutants, it was necessary to decide how to handle results below the detection limit (<10 µg/l). Alternatives considered were to exclude such values or to replace them with some number between zero and the limit. In order to use all the data and to be conservative from the standpoint of effluent levels plants can

TABLE F-1

COMPARISON OF STANDARD DEVIATIONS
ESTIMATED BY TWO METHODS

ORGANIC POLLUTANT		PLANT	LAB	METHOD: Variance Component	METHOD: Daily Average
(8)	1,2,4-trichlorobenzene	P4	8	.95	
			9	.73	
				.84	.88
(10)	1,2-dichloroethane	P1	1	2.20	
			4	2.32	
				2.26	2.54
		P3	3	1.31	1.39
(21)	2,4,6-trichlorophenol	P3	3	1.50	
			6	.84	
			8	1.35	
				1.23	1.55
(23)	Chloroform	P1	1	1.19	
			4	1.25	
			8	1.02	
				1.15	.92
(25)	1,2-dichlorobenzene	P4	9	1.37	1.54
(31)	2,4-dichlorophenol	P4	9	2.69	2.12
(44)	Methylene Chloride	P1	1	1.13	1.07
(59)	2,4-dinitrophenol	P3	6	1.77	1.80
(64)	Pentachlorophenol	P3	-	-	1.48
(65)	Phenol	P3	-	-	1.25
		P5	-	-	.24

NOTE: The average standard deviation is given for the variance component method where there are estimates for more than one laboratory.

TABLE F-1
(concluded)

ORGANIC POLLUTANT	PLANT	LAB	METHOD Variance Component	METHOD Daily Average
(66) Bis(2-ethylhexyl) Phthalate	P3	3	1.54	
		6	2.34	
		8	1.00	
			1.63	1.50
(68) Di-n-butyl Phthalate	P3	3	1.29	1.20
(70) Diethyl Phthalate	P3	3	1.52	1.65
(86) Toluene	P3	3	1.71	1.99

achieve, a replacement value equal to the detection limit was chosen. For example, suppose duplicate measurements on a sample resulted in C1 = 20 μg/l and C2 = ND (not detected). Setting C2 = 0 gives an average concentration of $\overline{C}$ = 10 μg/l; setting C2 = 10 gives $\overline{C}$ = 15 μg/l. Note that the use of 10 not only yields a higher average, but results in counting the pollutant as detected in the sample (with detected defined as $\overline{C} > 10$).

C. GOODNESS-OF-FIT TESTS

The statistical distribution used to model the effluent data assumes that $X = \log (C-D)_e$ is NORMAL for $C > D$, where C is the daily effluent concentration and D is the analytical detection limit. To assess the validity of that assumption, goodness-of-fit tests were performed using the studentized range test based on the statistic

$$U = R/S,$$

with the range (R) and standard deviation (S) defined in A. Critical values of the U-test are given in Biometrika Tables (Pearson and Hartley, 1969). An upper tail test was used to guard against alternative distributions with heavier tails than the lognormal distribution: if such alternatives were appropriate, the lognormal distribution would tend to underestimate the 99th percentile.

A test was run using daily sample averages above the detection limit for each plant-pollutant data set. The criterion for distribution rejection was a statistical significance level of $\alpha = 0.01$ for each test. As the results

in TABLE F-2 show, the model was rejected for only one of the 27 data sets tested (lead at Plant 113). The Agency concluded that the lognormal distribution is appropriate for modeling the data above the analytical detection limit.

For organic pollutants, a detection limit of D = 10 μg/l was used; no metals readings were reported as being at or below a detection limit, therefore D = 0 was used.

D. DERIVATION OF VARIABILITY FACTORS

To develop variability factors for each pollutant at each plant, the Agency assumed that the concentration C has a delta distribution modified to have its origin at D, the analytical detection limit (Aitchison and Brown, 1957). This assumption implies that a result under the detection limit has probability δ of occurring, and $x = \log(C - D)$ for $C > D$ is normally distributed with mean μ and variance σ^2. The 99th percentile value of the concentration is then

$$C_{0.99} = D + e^{\mu + z\sigma} \qquad (1)$$

with

$$z = \Phi^{-1}\left[(.99 - \delta)/(1 - \delta)\right],$$

where $\Phi^{-1}(\bullet)$ is the inverse of the standard normal cumulative distribution function. The mean and variance of C for $C > D$ are

$$\mu_c = D + e^{\mu + 1/2\,\sigma^2} \qquad (2)$$

TABLE F-2

GOODNESS-OF-FIT TESTS FOR VARIABILITY DATA

LOG_e ($\overline{C}$ - D) FOR DAILY AVERAGES $\overline{C}$ OVER D μg/ℓ

POLLUTANT	PLANT	NDAY	U	TEST RESULT*
(8) 1,2,4-trichlorobenzene	P4	11	3.00	N.S.
(10) 1,2-dichloroethane	P1	7	2.73	N.S.
	P3	7	2.65	N.S.
(21) 2,4,6-trichlorophenol	P3	18	3.18	N.S.
(23) Chloroform	P1	14	3.05	N.S.
(25) 1,2-dichlorobenzene	P4	6	2.48	N.S.
(31) 2,4-dichlorophenol	P4	5	2.53	N.S.
(44) Methylene chloride	P1	13	4.23	N.S.
(59) 2,4-dinitrophenol	P3	7	3.02	N.S.
(64) Pentachlorophenol	P3	4	2.44	N.S.
(65) Phenol	P3	3	1.73	N.S.
	P5	4	2.00	N.S.
(66) Bis(2-ethylhexyl) phthalate	P3	26	3.64	N.S.
(68) Di-n-butyl phthalate	P3	10	3.79	N.S.
(70) Diethyl phthalate	P3	24	3.94	N.S.
(86) Toluene	P3	10	3.41	N.S.
(119) Chromium	3	46	3.14	N.S.
	110	8	3.25	N.S.
	113	90	3.18	N.S.
	126	26	5.01	N.S.
(120) Copper	113	145	5.42	N.S.
	118	27	5.18	N.S.
(122) Lead	113	13	4.53	P < 0.01
(128) Zinc	27	158	6.01	N.S.
	110	8	2.63	N.S.
	113	140	6.07	N.S.
(121) Cyanide	P5	27	4.14	N.S.

*Critical values for the studentized range test (α = 0.01, upper tail) are:

n	U.99	n	U.99
3	2.00	13	4.24
4	2.44	14	4.34
5	2.80	18	4.67
6	3.10	20	4.80
7	3.34	25	5.06
8	3.54	30	5.26
10	3.88	45	5.67
11	4.01	90	6.27

N.S. = not significant (U value below critical level).
Pearson and Hartley, 1969

and

$$\sigma_c^2 - e^{2\mu + \sigma^2} (e^{\sigma^2} - 1). \qquad (3)$$

The Agency defined the daily variability factor as

$$VF(1) = \frac{C_{0.99}}{\mu_c} = \frac{D + e^{\mu + z\sigma}}{D + e^{\mu + 1/2\,\sigma^2}} \qquad (4)$$

Estimates of the above quantities are calculated by replacing δ by the proportion of observations below D and replacing μ and σ^2 with the mean and variance of $\log_e(C-D)$ for observations above D.

To obtain the variability factor for a monthly mean $\overline{C}$ of 4 samples per month, only results above the detection limit (i.e., C > D) were averaged. The following assumptions were made: $\overline{C}$ has a modified delta-lognormal distribution with the same mean as C and with variance proportional to σ_c^2 (Barakat, 1976), and measurements are uncorrelated. The last assumption implies that the probability of obtaining no results above D in the four monthly samples for a month is

$$\delta_4 = \delta^4. \qquad (5)$$

The other assumptions give

$$\mu_{\overline{c}} = D + e^{\mu_4 + 1/2\,\sigma_4^2}, \qquad (6)$$

$$\sigma^2_{\bar{c}} = e^{2\mu_4 + \sigma_4^2}(e^{\sigma_4^2} - 1). \qquad (7)$$

and

$$\bar{C}_{0.95} = D + e^{\mu_4 + z_4 \sigma_4} \qquad (8)$$

with

$$z_4 = \Phi^{-1}\left[(.95 - \delta_4)/(1 - \delta_4)\right].$$

The Agency defined the 4-day monthly variability factor as

$$VF(4) = \frac{\bar{C}_{0.95}}{\mu_{\bar{c}}} = \frac{D + e^{\mu_4 + z_4 \sigma_4}}{D + e^{\mu_4 + 1/2\, \sigma_4^2}} \qquad (9)$$

To estimate VF(4), it is necessary to express μ_4 and σ_4^2 in terms of δ, μ, and σ. Since $\mu_{\bar{c}} = \mu_c$, it can be shown using (2) and (6) that

$$\mu_4 = \mu + \frac{\sigma^2 - \sigma_4^2}{2}. \qquad (10)$$

To relate σ_4^2 to parameters of the original distribtuion, $\sigma^2_{\bar{c}}$ must be expressed as a function of σ_c^2. When k out of 4 tests in a month are above D (k > 0), the variance of $\bar{C}$ for that month is σ_c^2/k. In addition, k varies randomly from month to month with probability distribution

$$Pr\left[K = k\right] = \binom{4}{k}(1 - \delta)^k \delta^{4-k} \qquad (11)$$

for k = 0, 1, 2, 3, 4 (K has a binomial distribution with parameter $1 - \delta$). Thus $\sigma^2_{\bar{c}}$ is a weighted average of values σ^2_c/k for different k:

$$\sigma^2_{\bar{c}} = \sum_{k=1}^{4} \frac{Pr(K=k)}{1 - \delta^4} \frac{(\sigma^2_c)}{k} \tag{12}$$

$$= f(\delta)e^{2\mu + \sigma^2}(e^{\sigma^2} - 1)$$

with

$$f(\delta) = (1 - \delta^4)^{-1} \sum_{k=1}^{4} Pr(K = k)/k.$$

Equating the expressions in (7) and (12) and simplifying with the aid of (10) gives

$$\sigma^2_4 = \log\left[1 + f(\delta)(e^{\sigma^2} - 1)\right]. \tag{13}$$

Equations (5), (10), and (13) now express the parameters δ_4, μ_4, and σ^2_4 in terms of the original parameters δ, μ, and σ^2. Substituting the values calculated earlier for these three parameters into the three equations and substituting the results into equation (9) gives an estimate of VF(4).

For organic priority pollutants a detection limit of D = 10 μg/l was used in the model. For metals and cyanide, no readings had been reported as being at or below a detection limit.

E. EXAMPLE OF VARIABILITY FACTOR CALCULATIONS

Use of the above formulas to estimate variability factors is illustrated below with data for 1,2-dichloroethane (10) from CMA plant 3. To avoid rounding errors and reproduce computer results, intermediate calculations were carried out to several decimal places. Measurements of 1,2-dichloroethane were made on samples from 33 days, but the pollutant was detected on only 7 days. The daily sample average values ($\bar{C}$) and their corresponding values of log ($\bar{C}$ - D) are listed in TABLE F-3.

The estimated mean and variance of x are then

$$\hat{\mu} = \bar{x} = 1.40442$$

and

$$\hat{\sigma}^2 = s^2 = 1.93184$$

(Definitions for $\bar{x}$ and s^2 are given under Formulas and Definitions).

The estimated probability of obtaining a daily sample average at or below the detection limit is

$$\hat{\delta} = 26/33 = 0.787879,$$

so

$$z = \Phi^{-1}(0.952857) = 1.67321.$$

TABLE F-3

VALUES OF $\bar{C}$ AND X FOR 1,2-DICHLOROETHANE RESULTS

Daily Sample Average Concentration ($\bar{C}$)	$x = \log_e(\bar{C} - 10)$
13	1.09861
25.5	2.74084
50	3.68888
11	0
12	0.69315
11	0
15	1.60944

Therefore, by formulas (1), (2), and (4), the estimated 99th percentile, mean, and daily variability factor are

$$\hat{c}_{0.99} = 10 + e^{\hat{\mu} + z\hat{\sigma}} = 51.7,$$

$$\hat{\mu}_c = 10 + e^{\hat{\mu} + 1/2\,\hat{\sigma}^2} = 20.7,$$

and

$$VF(1) = \hat{c}_{0.99}/\hat{\mu}_c = 2.498$$

These results were rounded to 52, 21, and 2.50 and then entered into the variability factor table.

For the four day average variability factor, formula (5) gives

$$\delta_4 = (26/33)^4 = 0.385334;$$

thus

$$z_4 = \Phi^{-1}(0.918655) = 1.39608.$$

Next, $f(\delta)$ in (12) is estimated. By (11),

k	Pr(K = k)
1	$4\hat{\delta}^3(1 - \hat{\delta}) = 0.414975$
2	$6\hat{\delta}^2(1 - \hat{\delta})^2 = 0.167586$
3	$4\hat{\delta}(1 - \hat{\delta})^3 = 0.030080$
4	$(1 - \hat{\delta})^4 = 0.002025$

Thus

$$f(\hat{\delta}) = (1 - \hat{\delta}_4)^{-1} \sum_{k=1}^{4} \Pr(K = k)/k = 0.828582.$$

By formulas (10) and (13) then,

$$\hat{\sigma}_4^2 = \log\left[1 + f(\hat{\delta})(e^{\hat{\sigma}^2} - 1)\right]$$

$$= 1.77333$$

and

$$\hat{\mu}_4 = \hat{\mu} + (\hat{\sigma}^2 - \hat{\sigma}_4^2)/2$$

$$= 1.48368.$$

Using equations (8), (6), and (9), respectively, the estimated 95th percentile, mean, and variability factor for 4-day averages are

$$\hat{\bar{c}}_{0.95} = 10 + e^{\hat{\mu}_4 + z_4\hat{\sigma}_4} = 38.3,$$

$$\hat{\mu}_{\bar{c}} = 10 + e^{\hat{\mu}_4 + 1/2\,\hat{\sigma}_4^2} = 20.7,$$

and

$$VF(4) = \hat{\bar{c}}_{0.95}/\hat{\mu}_{\bar{c}} = 1.850.$$

The variability factor was rounded to 1.85 and then entered into the summary table.

F. SUBCATEGORIZATION

The data selection and reduction and statistical test procedures employed in the subcategorization analyses are described in this section. The wastewater characteristics examined were untreated influent concentrations of all priority pollutants except pesticides and asbestos.

Data from the Screening study were used for subcategorization because that study provided the most comprehensive assessment on the presence of priority pollutants at OCPSF plants (in terms of plant coverage and number of pollutants per plant). Untreated influent data were summarized as follows to create one observation for each plant:

- Not detected, trace, and values under 10 μg/ℓ were replaced by 10.

- Since the Screening data was used, pollutant concentration levels at a plant were usually based on a single analysis measurement. For plants where multiple analysis determinations were made, a sample average was generated for that pollutant.

- The value 10 μg/ℓ was inserted for any compound for which there was no measurement at a plant (i.e., if a value was not reported for a pollutant, it was considered not detected).

- Natural logarithms of the resulting concentrations were computed.

The initial data base produced by the above process continued observations for 143 plants.

Many priority pollutants were considered in the subcategorization analysis--88 organic compounds and 14 metals plus cyanide. Furthermore, the correlations among measurements of different pollutants caused by common analytical, sampling, and matrix effects made one-pollutant-at-a-time analyses inappropriate. Therefore, alternate multivariate analysis procedures were considered.

The classical multivariate technique for comparing the means of two populations (e.g., two possible subcategories of plants) involves comparing an F-statistic to tabled critical values of the F distribution with p and $N_1 + N_2 - p - 1$ degrees of freedom, where p is the number of pollutants and N_1 and N_2 are the numbers of plants with data in the two groups (Morrison, 1967, p. 125-126). It can be seen, therefore, that the number of plants with

measurements must exceed the number of pollutants measured in order to use this technique; that is,

$$N_1 + N_2 > p + 1$$

(since $N_1 + N_2 - p - 1$ must be greater than zero). There are pollutant measurements on 143 plants in the Screening file, but it was necessary to use the BPT Summary file to obtain other plant characteristics such as product/processes employed. Many of the plants in the Screening file could not be identified or had incomplete information in the Summary file. Thus less than 143 plants could be used in statistical comparisons based on the classical test. For example, there are 14 Plastics-Only and 78 Not Plastics-Only plants identifiable in the Screening file; there are 13 Organics-Only and 31 Mixed Organics/Plastics plants identifiable. It can be seen from these examples that it was not possible to include all 102 priority pollutants of interest (88 organics and 14 metals/cyanide) in a single multivariate comparison of groups of plants--splitting the pollutants into groups would have been necessary.

Another difficulty with using the classical multivariate test was the analytical limitations of the Screening data. These well-documented limitations made the use of a nonparametric procedure preferable, since such procedures are based on less restrictive assumptions than the classical multivariate procedure. Unfortunately, the well-known nonparametric procedures are univariate (statistical analyses of only one variable at a time). To address these problems, another multivariate technique called

principal components was used to define a few new uncorrelated variables based on the original pollutant variables, and the nonparametric test was applied to the new variables. The principal component analysis defined new variables as weighted averages of the original pollutant-specific variables; weights were selected to retain as much information as possible in the original data (Morrison, 1967, p. 222-230). Principal components were derived separately for organics and metals/cyanide because their measurements are based on different analytical methods; the derivation for organics is described below (the derivation for metals was similar).

For each plant, let $X_1, \ldots, X_{88}$ represent the original 88 organics variables (logs of mean concentrations of organic priority pollutants in the acid, base/neutral, and volatile fractions). Let $\overline{X}_i$ and s_i^2 represent the mean and variance of the ith variable, and $R = (r_{ij})$ the matrix of pairwise correlations among the 88 variables $\overline{X}_i$, s_i^2, and r_{ij} were computed from the 143 plant-specific observations described above). The first principal component, Y_1, was defined as the weighted average

$$Y_1 = \sum_{i=1}^{88} a_{i1}(X_i - \overline{X}_i)/s_i \tag{14}$$

whose coefficients a_{i1} were chosen to make the sample variance

$$s_{Y1}^2 = \sum_{i=1}^{88} \sum_{j=1}^{88} a_{i1}a_{j1}r_{ij}$$

as large as possible given that

$$\sum_{i=1}^{88} a_{i1}^2 = 1.$$

The second principal component was defined as the weighted average

$$Y_2 = \sum_{i=1}^{88} a_{i2}(X_i - \overline{X}_i)/s_i \tag{15}$$

whose coefficients a_{i2} were chosen to make the sample variance

$$s_{Y2}^2 = \sum_{i=1}^{88} \sum_{j=1}^{88} a_{i2}a_{j2}r_{ij}$$

as large as possible given that

$$\sum_{i=1}^{88} a_{i2}^2 = 1$$

and

$$\sum_{i=1}^{88} a_{i1}a_{i2} = 0.$$

(The last condition makes Y_1 and Y_2 uncorrelated.) Additional principal components were defined in similar fashion. The weights for each component were computed by the PRINCOMP procedure in SAS (SAS Institute, 1982, p. 347-361). The value of the first principal component at a given plant was obtained by substituting the log-mean concentrations for the 88 pollutants at that plant for the X_i in formula (14). Plant-specific values of other

principal components were obtained from corresponding formulas for those components.

When a principal component analysis is based on the correlation matrix R as above, the total variation for all 88 components (the sum of the s_Y^2's) equals the number of original variables, 88. Thus the ratio $s_{Yi}^2/88$ indicates the proportion of the total variation accounted for by the ith component. Because of the way principal components are defined, the proportion of variation accounted for by successive components generally decreases (and never increases).

Principal components analysis has the following advantages as a variable-reduction procedure:

- It indicates through $s_{Yi}^2/88$ how many components are needed to describe the data.
- The first few components summarize most of the information in the data when the original variables are highly correlated.
- The principal components themselves are uncorrelated so interpretation of statistical analyses based on them is straightforward.
- Principal components often have a physical meaning that can be identified from the magnitudes of the weights (a_{ij}).

For the organics Screening data, the first 5 components accounted for 74 percent of the total variation; for metals and cyanide the first five components accounted for 78 percent of the total variation. Morrison suggests

that up to five principal components be retained for subsequent analysis if those components account for at least 75 percent of the total variation.

The weights defining the first five principal components for organics and for metals/cyanide are shown in TABLES F-4 AND F-5, respectively.

In the final stage of the statistical subcategorization analysis, the first five principal components were evaluated for each plant, and plants belonging to groups of interest (e.g., Plastics-Only or Not Plastics-Only producers) were identified using information from the BPT Summary file. Plants not classifiable were excluded from the remainder of the analysis, which consisted of using a normal-scores test (Bradley, 1968) to compare group medians of principal component scores.

Results of the comparison of Plastics-Only and Not Plastics-Only plants are given in TABLE F-6. Differences between the two groups were found for the first and fifth organics components. Based on an examination of the relative magnitudes of the weights for individual components in Table F-4, these two components can be roughly interpreted as the average for all 88 compounds and the average for benzene, chlorobenzene, ethylbenzene and toluene, respectively. The statistical test indicates that Not Plastics-Only plants had higher median levels of these two weighted averages than the Plastics-Only plants.

A further analysis based on subdividing Not Plastics-Only plants into Organics-Only and Mixed Organics/Plastics producers showed no significant

TABLE F-4

PRINCIPAL COMPONENT WEIGHTS FOR ORGANICS DATA

NAME	PRIN1	PRIN2	PRIN3	PRIN4	PRIN5
(001) ACENAPHTHENE	0.11832	-0.09204	0.12450	0.02471	-0.07431
(002) ACROLEIN	0.10254	0.03237	-0.13206	-0.07823	0.15138
(003) ACRYLONITRILE	0.09594	0.02801	-0.14006	-0.07650	0.15403
(004) BENZENE	0.03579	0.02401	0.24430	-0.00345	0.39891
(005) BENZIDINE	0.11649	-0.06185	-0.00499	-0.02740	0.03294
(006) CARBON TETRACHLORIDE	0.04990	0.14408	0.09154	-0.04663	0.11093
(007) CHLOROBENZENE	0.02269	0.08494	0.01713	-0.00106	0.39068
(008) 1,2,4-TRICHLOROBENZENE	0.12806	-0.07363	-0.04961	-0.06440	0.07410
(009) HEXACHLOROBENZENE	0.12283	-0.06556	-0.00722	0.03107	0.03824
(010) 1,2-DICHLOROETHANE	0.06166	0.14630	0.03189	-0.05613	0.02358
(011) 1,1,1-TRICHLOROETHANE	0.07084	0.12300	0.06478	-0.01088	-0.08441
(012) HEXACHLOROETHANE	0.13167	-0.07959	0.01439	0.00687	-0.01359
(013) 1,1-DICHLOROETHANE	0.10759	0.17180	-0.05333	-0.06053	0.06081
(014) 1,1,2-TRICHLOROETHANE	0.11434	0.19300	-0.01635	-0.05458	-0.12345
(015) 1,1,2,2-TETRACHLOROETHANE	0.09515	0.19678	-0.00526	-0.04839	-0.15969
(016) CHLOROETHANE	0.08960	0.17613	-0.03963	-0.03809	-0.08165
(017) BIS (CHLOROMETHYL) ETHER	0.10805	0.14920	-0.04864	-0.08669	0.06753
(018) BIS (2-CHLOROETHYL) ETHER	0.13682	-0.07077	-0.03315	-0.05069	-0.00900
(019) 2-CHLOROETHYLVINYL ETHER	0.10458	0.16754	0.04113	-0.02569	0.05771
(020) 2-CHLORONAPHTHALENE	0.13611	-0.06862	-0.04357	-0.04806	0.00417
(021) 2,4,6-TRICHLOROPHENOL	0.08193	0.02875	-0.14453	0.29237	0.06094
(022) 4-CHLORO-M-CRESOL	0.09946	0.04933	-0.11297	0.30597	0.02545
(023) CHLOROFORM	0.02229	0.09740	0.18054	0.06215	0.05912
(024) 2-CHLOROPHENOL	0.07882	0.05713	-0.14389	0.28065	0.06125
(025) 1,2-DICHLOROBENZENE	0.11444	-0.04583	-0.02831	-0.07489	0.11233
(026) 1,3-DICHLOROBENZENE	0.12127	-0.02829	-0.01400	-0.06128	0.04829
(027) 1,4-DICHLOROBENZENE	0.10948	-0.04410	0.00434	0.03403	0.03100
(028) 3,3-DICHLOROBENZIDINE	0.13667	-0.07195	-0.05525	-0.06190	0.02805
(029) 1,1-DICHLOROETHYLENE	0.08101	0.13390	0.14023	0.04340	0.17501
(030) 1,2-TRANSDICHLOROETHYLENE	0.11179	0.20678	0.02071	-0.05852	-0.08186
(031) 2,4-DICHLOROPHENOL	0.07089	0.04118	-0.07943	0.34257	0.05122
(032) 1,2-DICHLOROPROPANE	0.11051	0.19151	-0.00915	-0.05173	-0.06060
(033) 1,3-DICHLOROPROPYLENE	0.09710	0.19114	0.07285	-0.04893	-0.14428
(034) 2,4-DIMETHYLPHENOL	0.07025	0.05421	0.05740	0.28435	-0.06726
(035) 2,4-DINITROTOLUENE	0.13105	-0.05846	-0.06753	-0.06536	0.03616
(036) 2,6-DINITROTOLUENE	0.11739	-0.05911	-0.07384	-0.05341	0.04886
(037) 1,2-DIPHENYLHYDRAZINE	0.12806	-0.05186	-0.05715	-0.02549	-0.00964
(038) ETHYLBENZENE	0.06442	0.03413	0.13828	0.05917	0.26989
(039) FLUORANTHENE	0.12304	-0.10152	0.14610	0.03246	-0.08005
(040) 4-CHLOROPHENYLPHENYL ETHER	0.13420	-0.07890	-0.03595	-0.09384	0.02231
(041) 4-BROMOPHENYLPHENYL ETHER	0.13231	-0.07321	-0.06447	-0.07584	0.04262

TABLE F-4 (concluded)

NAME	PRIN1	PRIN2	PRIN3	PRIN4	PRIN5
(042) BIS-(2-CHLOROISOPROPYL) ETHER	0.12328	-0.08636	-0.04923	-0.08711	0.02223
(043) BIS-(2-CHLOROETHOXY) METHANE	0.12994	-0.05707	-0.04904	-0.07580	0.02963
(044) METHYLENE CHLORIDE	0.01847	0.14937	0.29997	0.03189	0.07741
(045) METHYL CHLORIDE	0.06459	0.14575	-0.03850	-0.08328	0.02434
(046) METHYL BROMIDE	0.11343	0.16426	-0.06482	-0.05632	0.06992
(047) BROMOFORM	0.09996	0.16523	-0.03763	-0.03338	-0.08045
(048) DICHLOROBROMOMETHANE	0.10759	0.18494	0.02639	-0.08996	-0.13525
(049) TRICHLOROFLUOROMETHANE	0.03985	0.18125	0.16554	0.00962	0.05837
(050) DICHLORODIFLUOROMETHANE	0.10350	0.14501	-0.07459	-0.05124	0.06669
(051) CHLORODIBROMOMETHANE	0.10936	0.19881	-0.01253	-0.05321	-0.12406
(052) HEXACHLOROBUTADIENE	0.13577	-0.07686	-0.06010	-0.06901	0.04334
(053) HEXACHLOROCYCLOPENTADIENE	0.13372	-0.07690	-0.04000	-0.09478	0.02735
(054) ISOPHORONE	0.12654	-0.07552	0.00808	0.05459	-0.04904
(055) NAPHTHALENE	0.08261	-0.08181	0.24327	0.11219	-0.07097
(056) NITROBENZENE	0.10075	-0.03826	-0.00322	-0.01309	0.07289
(057) 2-NITROPHENOL	0.09850	0.04016	-0.16553	0.26254	0.03919
(058) 4-NITROPHENOL	0.09867	0.02423	-0.15526	0.21177	-0.01496
(059) 2,4-DINITROPHENOL	0.09247	-0.03557	-0.16634	0.21247	0.08969
(060) 4,6-DINITRO-O-CRESOL	0.11066	0.01715	-0.15425	0.20787	-0.03203
(061) N-NITROSODIMETHYLAMINE	0.12811	-0.10043	-0.07234	-0.08064	0.11704
(062) N-NITROSODIPHENYLAMINE	0.13042	-0.05853	-0.05274	-0.04155	0.05756
(063) N-NITROSODI-N-PROPYLAMINE	0.13280	-0.08230	-0.05791	-0.08475	0.05837
(064) PENTACHLOROPHENOL	0.10152	0.11060	-0.08187	0.16777	-0.06747
(065) PHENOL	0.03146	0.04506	0.09045	0.18532	0.07148
(066) BIS-(2-ETHYLHEXYL) PHTHALATE	0.07348	-0.01975	0.22260	0.11881	0.04058
(067) BUTYLBENZYL PHTHALATE	0.10984	-0.08687	0.03570	-0.06840	-0.06570
(068) DI-N-BUTYL PHTHALATE	0.09483	-0.03585	0.14928	0.11303	-0.12809
(069) DI-N-OCTYL PHTHALATE	0.12487	-0.05723	0.05483	0.00643	-0.08868
(070) DIETHYL PHTHALATE	0.11862	-0.10368	0.01156	-0.05015	-0.09015
(071) DIMETHYL PHTHALATE	0.11160	-0.08238	0.02932	0.02961	-0.09997
(072) BENZO(A)ANTHRACENE	0.12087	-0.12152	0.00108	-0.01091	0.07808
(073) BENZO(A)PYRENE	0.13441	-0.05759	0.00831	-0.08044	-0.01869
(074) 3,4-BENZOFLUORANTHENE	0.13487	-0.05981	-0.02084	-0.04020	-0.02449
(075) BENZO(K)FLUORANTHENE	0.12990	-0.08323	-0.05720	-0.03931	0.06108
(076) CHRYSENE	0.11579	-0.10256	0.06777	0.03394	-0.10366
(077) ACENAPHTHYLENE	0.10319	-0.10236	0.21682	0.10626	-0.12002
(078) ANTHRACENE	0.09696	-0.11007	0.21446	0.07784	0.01894
(079) BENZO(GHI)PERYLENE	0.13456	-0.07662	0.00658	-0.07446	-0.05279
(080) FLUORENE	0.11169	-0.10541	0.20816	0.06221	-0.08166
(081) PHENANTHRENE	0.11080	-0.06292	0.15200	0.02978	-0.12882
(082) DIBENZO(A,H)ANTHRACENE	0.13666	-0.07228	-0.02773	-0.05851	-0.03735
(083) INDENO(1,2,3-C,D)PYRENE	0.13549	-0.05998	-0.01665	-0.05135	-0.06193
(084) PYRENE	0.11794	-0.09883	0.17964	0.06655	-0.11559
(085) TETRACHLOROETHYLENE	0.07083	0.12133	0.07433	0.03379	0.10503
(086) TOLUENE	0.03813	-0.00456	0.22582	-0.04796	0.28578
(087) TRICHLOROETHYLENE	0.07514	0.18593	0.09676	-0.02850	-0.11590
(088) VINYL CHLORIDE	0.10825	0.17387	-0.02209	-0.06305	-0.11654

TABLE F-5
PRINCIPAL COMPONENT WEIGHTS FOR METALS/CYANIDE DATA

NAME	PRIN1	PRIN2	PRIN3	PRIN4	PRIN5
(114) ANTIMONY	0.30083	0.14570	-0.06916	0.10133	0.23727
(115) ARSENIC	0.19453	0.32669	0.11624	0.61894	-0.26333
(117) BERYLLIUM	0.27386	0.13534	-0.18238	-0.52114	-0.06186
(118) CADMIUM	0.32574	0.03418	0.02977	-0.21213	-0.19271
(119) CHROMIUM	0.23323	-0.40820	0.31476	0.07162	0.03567
(120) COPPER	0.28649	-0.09643	0.17821	-0.04895	0.07541
(121) CYANIDE	0.04380	0.48968	0.67704	-0.23871	0.38796
(122) LEAD	0.28694	-0.17496	0.14908	0.07995	0.23695
(123) MERCURY	0.24750	-0.11203	-0.42100	-0.07369	0.51283
(124) NICKEL	0.28106	-0.07280	0.18551	-0.05328	-0.43498
(125) SELENIUM	0.28805	0.19861	-0.18106	0.35473	0.15347
(126) SILVER	0.31248	0.11945	-0.09996	-0.25184	-0.37689
(127) THALLIUM	0.31597	0.21691	-0.20932	0.09984	0.01308
(128) ZINC	0.22166	-0.53631	0.19460	0.10805	0.04248

TABLE F-6

COMPARISON OF PLASTICS-ONLY PLANTS WITH OTHER PLANTS

PRINCIPAL COMPONENT	ORGANICS		METALS/CYANIDE	
	Cumulative % Variation	Significance Level*	Cumulative % Variation	Significance Level*
1	56	<.001	49	.473
2	63	.520	58	.338
3	68	.125	66	.130
4	72	.445	73	.757
5	74	<.001	78	.498

* Based on the Terry-Hoeffding (normal scores) test comparing medians for principal component score for Plastics-Only and Not Plastics-Only plants. There were 92 plants involved in the comparisons, 14 Plastics-Only and 78 Not Plastics-Only.

differences (see TABLE F-7). Likewise, no significant differences were found among the three BPT subcategories for Not Plastics-Only (TABLE F-8). These analyses employed the same principal components as the Plastics-Only/Not Plastics-Only comparison; they included all Screening plants that could be identified as belonging to the groups of interest.

TABLE F-7

COMPARISON OF ORGANICS-ONLY PLANTS WITH
MIXED ORGANICS/PLASTICS PLANTS

PRINCIPAL COMPONENT	ORGANICS		METALS/CYANIDE	
	Cumulative % Variation	Significance Level*	Cumulative % Variation	Significance Level*
1	56	.717	49	.694
2	63	.871	58	.550
3	68	.502	66	.258
4	72	.837	73	.948
5	74	.738	78	.410

* Based on the Terry-Hoeffding (normal scores) test comparing median of principal component scores for Organics-Only and Mixed Organics/Plastics plants. There were 44 plants involved in the comparisons, 13 Organics-Only and 31 Mixed Organics/Plastics.

TABLE F-8

COMPARISON OF THREE BPT SUBCATEGORIES
FOR NOT PLASTICS-ONLY PLANTS*

PRINCIPAL COMPONENT	ORGANICS		METALS/CYANIDE	
	Cumulative % Variation	Significance Level**	Cumulative % Variation	Significance Level**
1	56	.575	49	.710
2	63	.705	58	.166
3	68	.370	66	.822
4	72	.122	73	.978
5	74	.342	78	.270

* The 3 subcategories are Type I with Oxidation (27 plants), Type I without oxidation (8 plants), and Not Type I (9 plants).

** Based on the normal scores test comparing medians of principal component scores for plants in the 3 subcategories (with 44 plants involved in the comparisons). The test was run using PROC NPAR/WAY in SAS Institute (1982), pages 205-211, with the van der Waerden option.

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APPENDIX G

CHEMICAL TREES OF THE GENERALIZED PLANT CONFIGURATIONS (GPCs)

Code	Product/Process	Average Daily Prod. x 1000 lb.
1770-01	Ethylene--Pyrolysis of ethane/propane/butane/LPG	1700
3090-02	Propylene--Pyrolysis of ethane/propane/butane/LPG	400
3008-01	Polyethylene resin--Solution polymerization (HDPE)	400
3008-04	Polyethylene resin--High pressure polymerization (LDPE)	1200

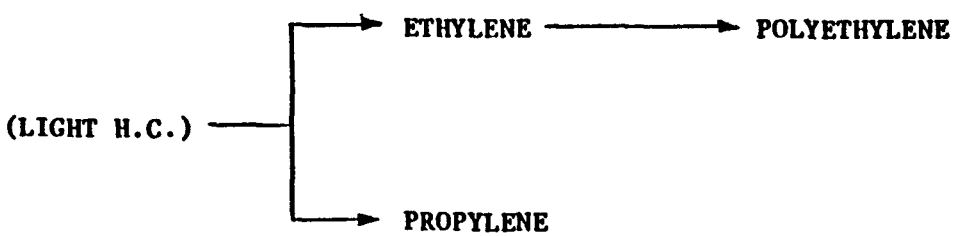

Figure 1. Chemical Tree - GPC 500 Olefins + Polyethylene

Code	Product/Process	Average Daily Prod. x 1000 lb.
1770-02	Ethylene--Pyrolysis of napththa a/o gas oil	3000
3090-06	Propylene--Pyrolysis of naphtha a/o gas oil	1400
0590-01	Butadiene--Extractive dist. of C_4 pyrolyzates	300
0720-01	Butylenes--Extractive dist. of C_4 pyrolyzates	450
2265-01	Isobutylene--Extract from C_4 pyrolyzate	300
2350-02	Isoprene-- Extractive dist. of C_5 pyrolyzate	50
0380-09	Benzene--Dist. of BTX extract/pyrolysis gasoline	750
3349-07	Toluene--Dist. of BTX extract/pyrolysis gasoline	1000
3541-01	Xylenes (Mixed)--Bottom BTX extract/pyrolysis gas	450

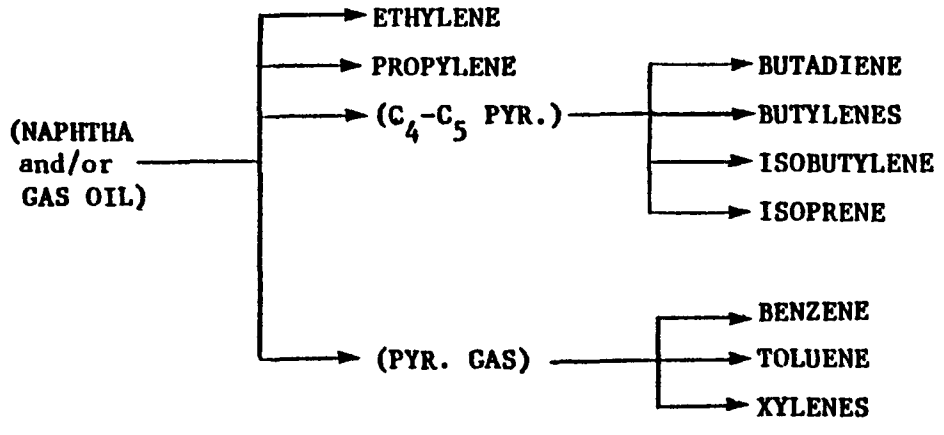

Figure 2. Chemical Tree - GPC 501 Olefins/Aromatics

Code	Product/Process	Average Daily Prod. x 1000 lb.
1770-02	Ethylene--Pyrolysis of naphtha a/o gas oil	1600
1770-04	Ethylene--Pyrolysis of naphtha, propane, ethane a/o butane	500
3090-06	Propylene--Pyrolysis of naphtha a/o gas oil	1200
3090-11	Propylene--Pyrolysis of naphtha, propane, ethane a/o butane	200
0590-01	Butadiene--Extractive dist. of C4 pyrolyzates	200
0720-01	Butylenes--Extractive dist. of C4 pyrolyzates	250
2350-02	Isoprene--Extractive dist. of C5 pyrolyzates	250
1171-01	Cyclopentadiene dimer--Ext. dist. C5 pyrolyzates & dimerization	100
0130-03	Acetylene--By-product of ethylene	25
0380-09	Benzene--Dist. of BTX extract/pyrolysis gasoline	950
0380-01	Benzene--Hydrodealkylation of toluene a/o xylene	350
3349-07	Toluene--Dist. of BTX extract/pyrolysis gasoline	500
3541-01	Xylenes (mixed)--Bottom BTX ext/pyrolysis gasoline	150
3541-08	Xylenes (mixed)--m-,p-xylenes - bottoms from xylene sep.	50
3560-01	o-Xylene--Dist. from mixed xylenes	50
2701-02	Naphthalene--Dist. from pyrolysis gasoline	150
1710-02	Ethyl benzene--Separation from BTX extract	15
2856-01	Petroleum hydrocarbon resins--From C5-C8 unsaturates	200
1980-01	Ethylene oxide--Direct oxidation of ethylene	1650
1830-01	Ethylene glycol--Hydrolysis of ethylene oxide	1600
1300-01	Diethylene glycol--Co-product of ethylene glycol from EO	150
3460-01	Triethylene glycol--Co-product of ethylene glycol from EO	20
0140-01	Acrolein--Oxidation of propylene	180

Figure 3. Chemical Tree - GPC 502 Olefins/Aromatics/Derivatives

1060-01	Cumene--Alkylation of benzene by propylene	1250
1442-01	Diisopropyl benzene--By-product of cumene by alkylation	20
0090-01	Acetone--Cumene oxidation and cleavage	550
2910-02	Phenol--Cumene oxidation and cleavage	900
2690-01	α-Methylstyrene--By-product of acetone/phenol	80

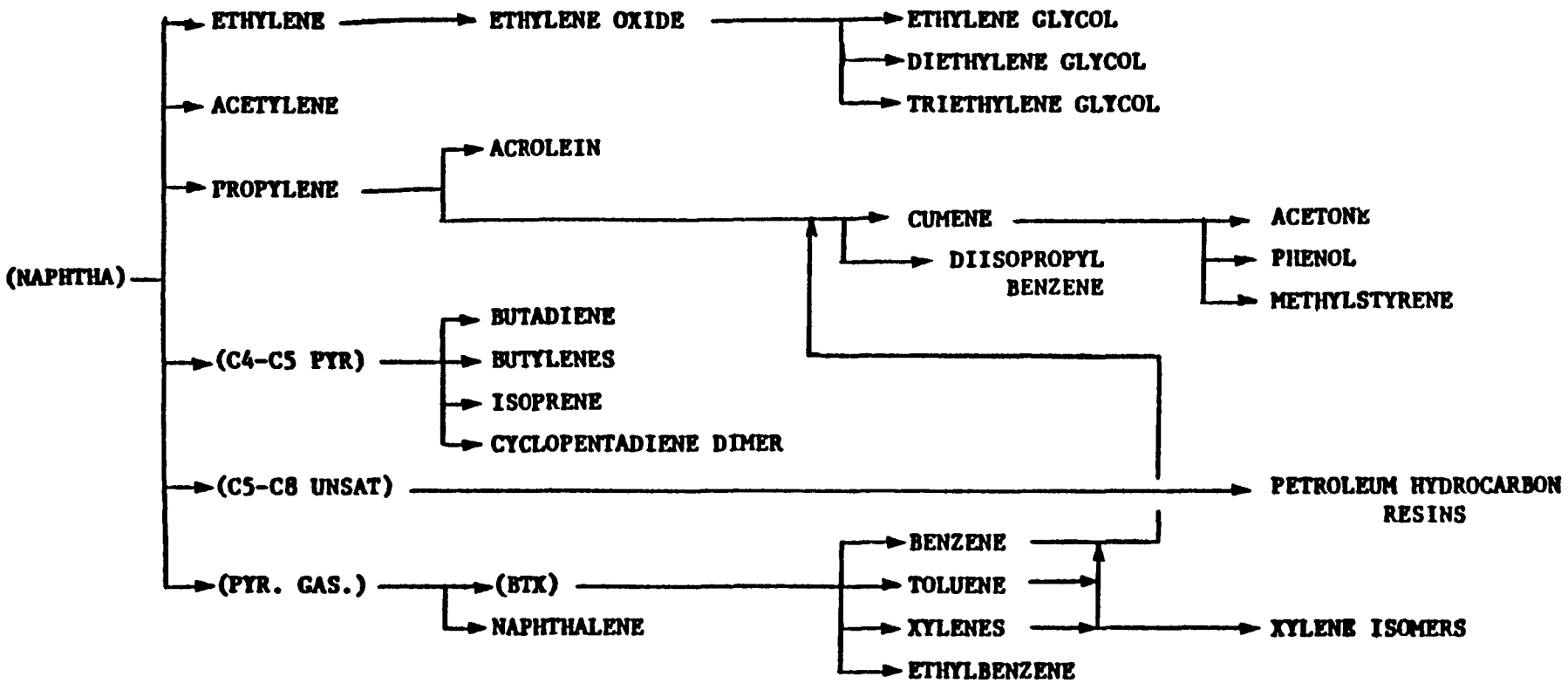

Figure 3. (Continued)

Code	Product/Process	Average Daily Prod. x 1000 lb.
1770-01	Ethylene--Pyrolysis of ethane/propane/butane/LPG	2000
3090-02	Propylene--Pyrolysis of ethane/propane/butane/LPG	900
0130-03	Acetylene--By-product of ethylene	100
0590-01	Butadiene--Extractive dist. of C4 pyrolyzates	150
1980-01	Ethylene oxide--Direct oxidation of ethylene	750
1980-02	Ethylene oxide--Via ethylene chlorohydrin process	150
1830-01	Ethylene glycol--Hydrolysis of ethylene oxide	800
1300-01	Diethylene glycol--Co-product of ethylene glycol from E.O.	100
3460-01	Triethylene glycol--Co-product of ethylene glycol from E.O.	10
3120-02	Propylene oxide--From propylene via chlorohydrin	750
3025-01	Polyoxypropylene glycol--React. of prop. glycol + prop. oxide	900
3025-02	Polyoxypropylene glycol--Propoxylation of glycerine	125
3008-04	Polyethylene resin--High pressure polymerization (LDPE)	850
3020-03	Polypropylene resin--Solution polymerization	200

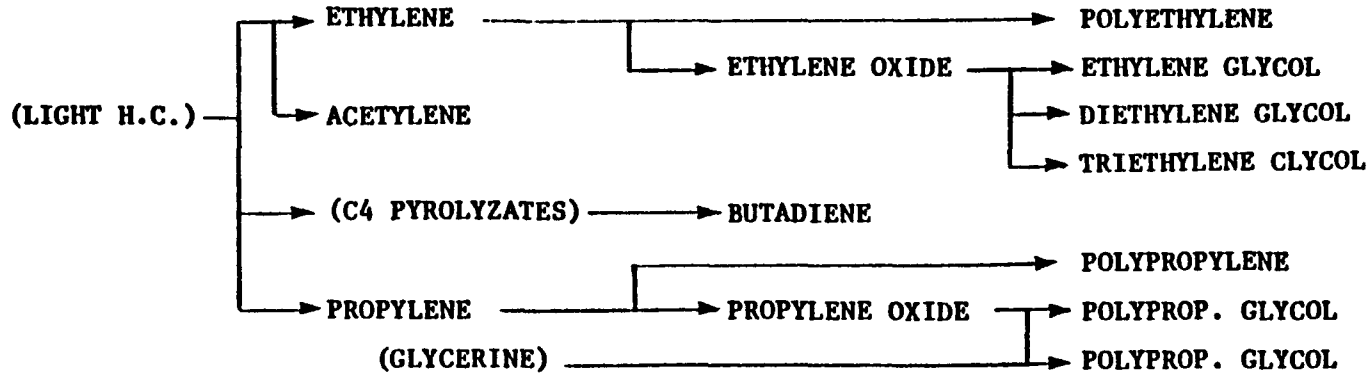

Figure 4. Chemical Tree - GPC 503 Olefins & Glycols & Polymers

Code	Product/Process	Average Daily Prod. x 1000 lb.
1770-01	Ethylene--Pyrolysis of ethane/propane/butane/LPG	500
3090-02	Propylene--Pyrolysis of ethane/propane/butane/LPG	200
0130-03	Acetylene--By-product of ethylene	25
0590-01	Butadiene--Extractive dist. of C4 pyrolyzates	40
1710-01	Ethylbenzene--Liquid phase benzene alkylation	1800
3230-01	Styrene--Dehydrogenation of ethyl benzene	1750

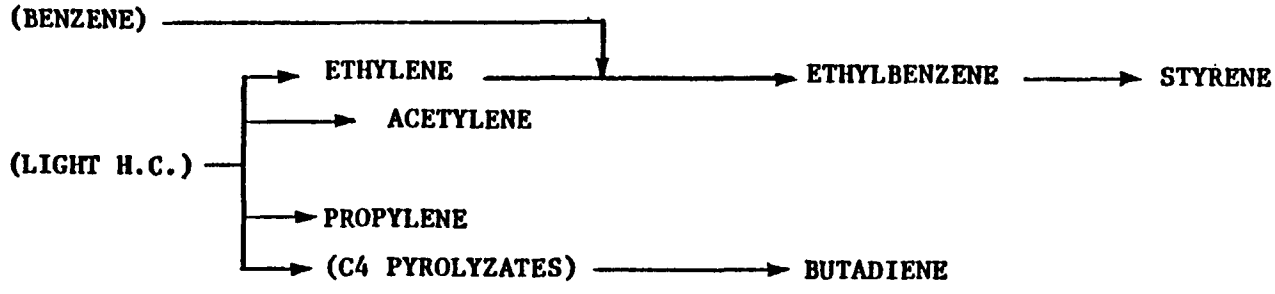

Figure 5. Chemical Tree - GPC 504: Olefins + Ethylbenzene/Styrene

Code	Product/Process	Average Daily Prod. x 1000 lb.
1770-01	Ethylene--Pyrolysis of ethane/propane/butane/LPG	1500
3090-02	Propylene--Pyrolysis of ethane/propane/butane/LPG	600
0720-01	Butylenes--Extractive dist. of C4 pyrolyzates	75
2265-01	Isobutylene--Extractive dist. of C4 pyrolyzates	20
3050-01	Propionaldehyde--Hydroformylation of ethylene - oxo process	250
3070-01	n-Propyl alcohol--Hydrogenation of propionaldehyde - oxo process	120
3068-01	n-Propyl acetate--Reaction of acetic acid + n-propanol	200
3066-01	Propionic acid--Air oxidation of propionaldehyde	175
0640-02	n-Butyl alcohol--Hydrogenation of n-butyraldehyde - oxo process	300
2000-01	2-Ethyl hexanol--Aldol condensation/hydro. of n-butyraldehyde	550
2250-01	Isobutanol--Hydrogenation of isobutyraldehyde - oxo process	50
2831-02	Oxo aldehydes/alcohols--Amyl alcohol (mixed)	100
0240-01	Amyl acetates--Reaction of acetic acid and amyl alcohols	150
2750-01	Neopentanoic acid--From isobutylene via oxo process	35

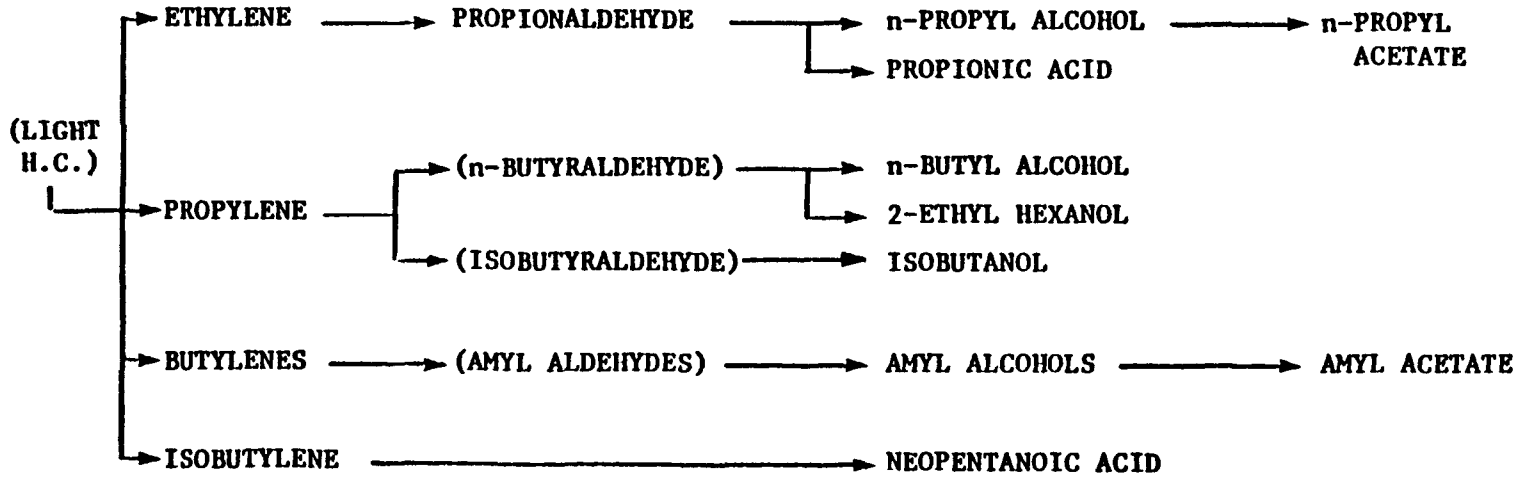

Figure 6. Chemical Tree - GPC 505 Olefins & Oxo Chemicals

Code	Product/Process	Average Daily Prod. x 1000 lb.
1770-01	Ethylene--Pyrolysis of ethane/propane/butane/LPG	3500
3090-02	Propylene--Pyrolysis of ethane/propane/butane/LPG	1200
0590-01	Butadiene--Ext. distillation of C4 pyrolyzates	250
1244-01	1,2-Dichloroethane (EDC)--Direct chlorination of ethylene	2700
3520-03	Vinyl chloride--Thermal cracking of EDC	850
3520-80	Vinyl chloride--From ethylene via EDC by chlor/oxy-chlor.	400
3530-02	Vinylidene chloride--Dehydrochlorination of 1,1,2-trichloroethane	140
3410-03	Trichloroethylene--Chlorination of EDC & other chlor. H.C.	140
3295-02	Tetrachloroethylene--Chlorination of EDC & other chlor. H.C.	180
0810-04	Carbon tetrachloride--Coproduct of tetrachloroethylene	90
1800-01	Ethylene diamine--Amination of EDC	75
3011-01	Polyethylene polyamines--Ethylene diamine + EDC + NH_3	25
3510-05	Vinyl acetate--Vapor phase reaction of ethylene + acetic acid	1000
1660-01	Ethanol--Direct hydration of ethylene	800
1700-01	Ethylamine--Ammonolysis of ethanol	50
0192-04	Alkylamines, C_{11} - C_{19}--From olefin + HCN + H_2	25
2360-01	Isopropanol--Hydrolysis of propylene	1000
0090-11	Acetone--Byproduct of H_2O_2 by oxidation of isopropanol	150
0210-01	Allyl chloride--Chlorination of propylene	* 250
1650-01	Epichlorohydrin--From allyl chloride via dichlorohydrin	250
2090-03	Glycerine--Hydrolysis of epichlorohydrin via allyl chloride	70
0560-01	Bisphenol A--Condensation of acetone with phenol	380
1656-01	Epoxy resins--Epichlorohydrin + bisphenol-A	200
0140-01	Acrolein--Oxidation of propylene	100
0030-06	Acetaldehyde--Byproduct of acrolein by prop. oxidation	10

Figure 7. Chemical Tree - GPC 506 Olefins & Derivatives

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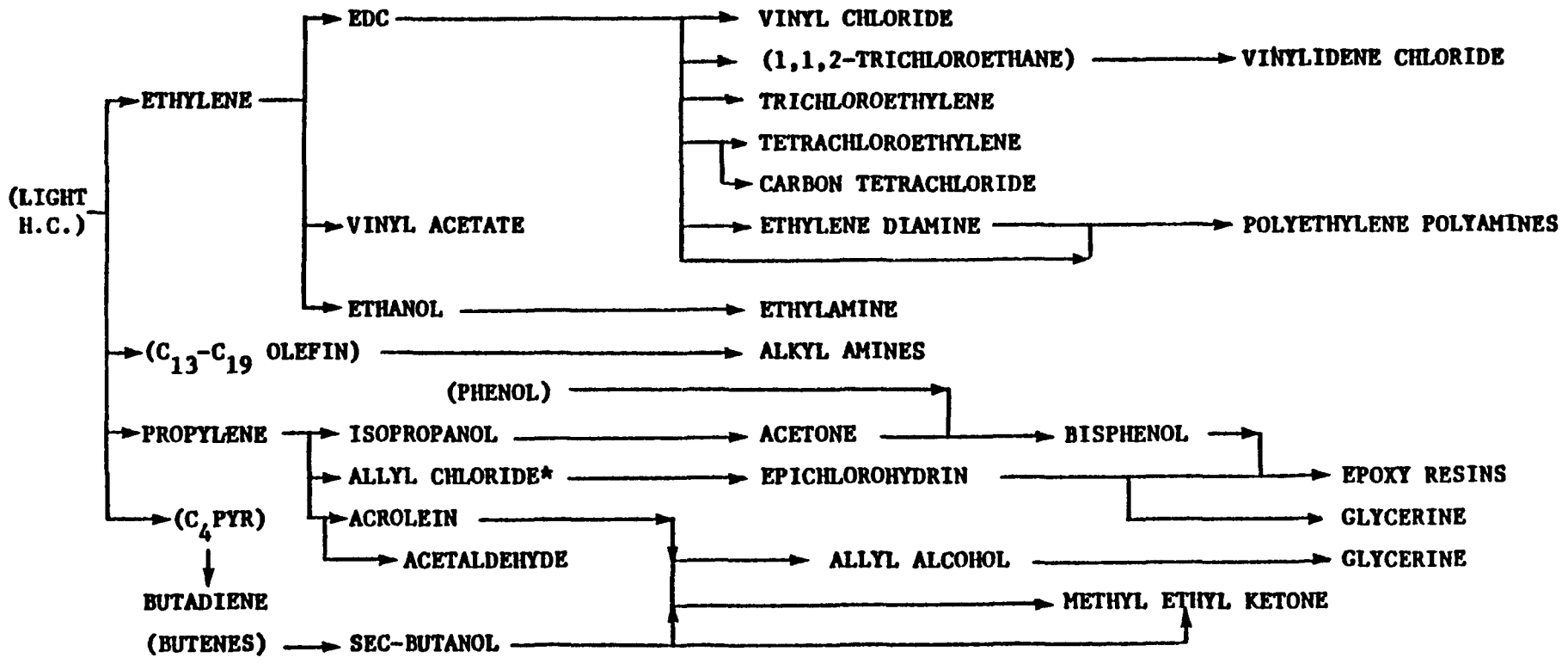
0650-01	Sec-Butanol--Indirect hydration of butenes	150
0200-01	Allyl alcohol--Redox of acrolein + sec-butanol	80
2090-01	Glycerine--Hydroxylation of allyl alcohol	100
2640-07	Methyl ethyl ketone--Redox of acrolein + sec-butanol	100
2640-01	Methyl ethyl ketone--Dehydrogenation of sec-butanol	100



* No wastewater reported in verification.

Figure 7. Chemical Tree - GPC 506 Olefins & Derivatives

Page 2

Code	Product/Process	Average Daily Prod. x 1000 lb.
3070-01	n-Propyl alcohol--Hydrogenation of propionaldehyde - oxo process	60
3068-01	n-Propyl acetate--Reaction of acetic acid + n-propanol	30
3066-01	Propionic acid--Air oxidation of propionaldehyde	125
0640-02	n-Butyl alcohol--Hydrogenation of n-butyraldehyde - oxo process	200
2000-01	2-Ethyl hexanol--Aldol cond./hydrogenation of n-butyraldehyde	300
2250-01	Isobutanol--Hydrogenation of isobutyraldehyde - oxo process	100

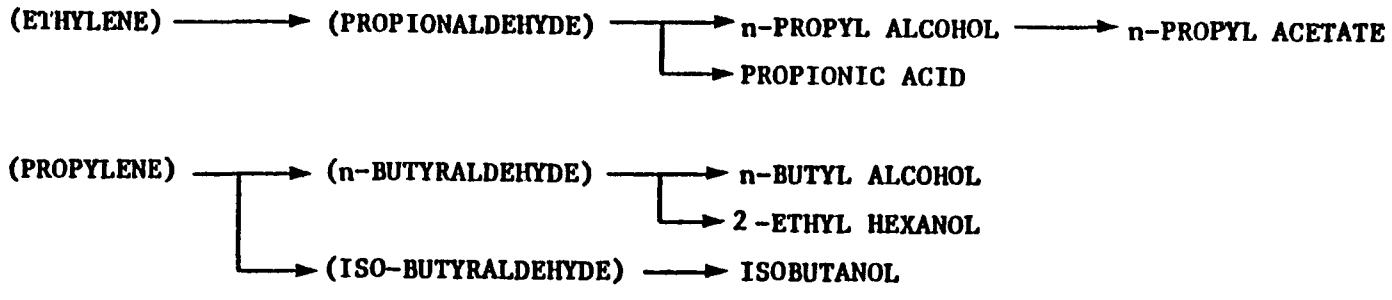


Figure 8. Chemical Tree - GPC 507 Oxo Chemicals

Code	Product/Process	Average Daily Prod. x 1000 lb.
1980-01	Ethylene oxide--Direct oxidation of ethylene	750
1830-01	Ethylene glycol--Hydrolysis of ethylene oxide	500
1300-01	Diethylene glycol--Co-product of ethylene glycol from E.O.	50
3460-01	Triethylene glycol--Co-product of ethylene glycol from E.O.	10
3460-03	Triethylene glycol--From ethylene glycol still bottoms	200
3325-02	Tetraethylene glycol--From ethylene glycol still bottoms	30
1666-03	C_{11},C_{12} Ethoxylates--Linear alcohols + ethylene glycol	700
3120-02	Propylene oxide--From propylene via chlorohydrin	400
3025-01	Polyoxypropylene glycol--Reaction of propylene glycol + prop. oxide	300
3025-02	Polyoxypropylene glycol--Propoxylation of glycerine	200

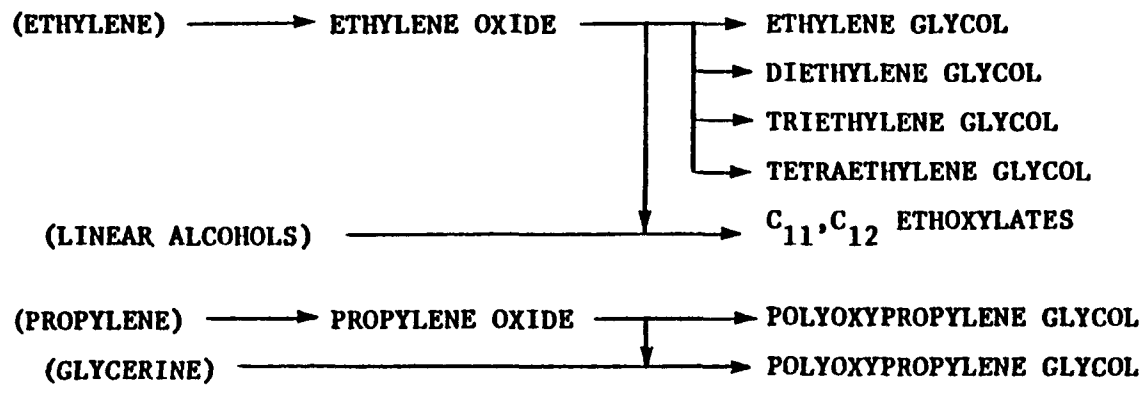


Figure 9. Chemical Tree - GPC 508 Oxides/Glycols

Code	Product/Process	Average Daily Prod. x 1000 lb.
1710-01	Ethylbenzene--Liquid phase benzene alkylation	2050
3230-01	Styrene--Dehydrogenation of ethylbenzene	2000

Figure 10. Chemical Tree - GPC 509 Ethylbenzene/Styrene

Code	Product/Process	Average Daily Prod. x 1000 lb.
0380-02	Benzene--Dist. of BTX extract/cat. reformate	800
3349-01	Toluene--Dist. of BTX extract/cat. reformate	600
3541-03	Xylenes (mixed)--Bottom BTX extract/cat. reformate	1300
3560-01	o-Xylene--Distillation from mixed xylenes	400
3570-02	p-Xylene--Isomerization/crystallization of mixed xylenes	450
1710-02	Ethylbenzene--Separation from BTX extract	100
2265-02	Isobutylene--Dehydration of purchased tert-butanol	200

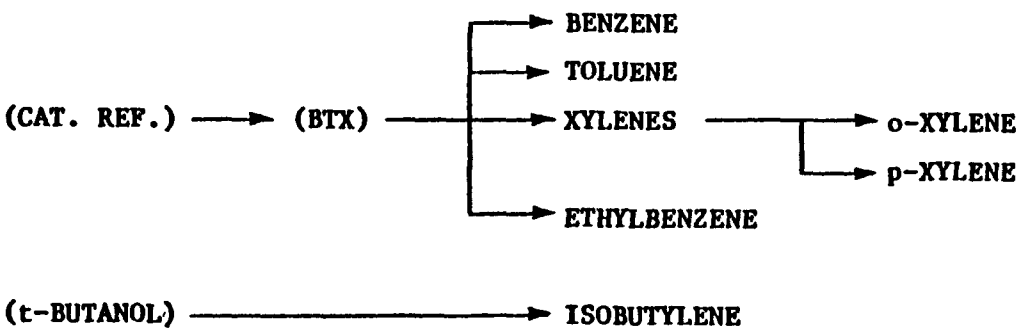


Figure 11. Chemical Tree - GPC 510 Aromatics From Cat. Reformate

Code	Product/Process	Average Daily Prod. x 1000 lb.
0380-04	Benzene--Dist. of BTX extract/coal tar light oil	100
3349-02	Toluene--Dist. of BTX extract/coal tar light oil	20
3541-04	Xylenes (mixed)--Bottom BTX extract/coal tar light oil	10
2701-01	Naphthalene--Separation from coal tar distillate	20

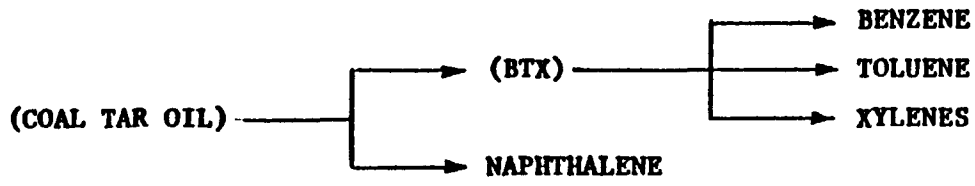


Figure 12. Chemical Tree - GPC 511 Aromatics From Coal Tar

Code	Product/Process	Average Daily Prod. x 1000 lb.
0380-04	Benzene--Dist. of BTX extract/coal tar light oil	950
3349-02	Toluene--Dist. of BTX extract/coal tar light oil	150
3541-04	Xylenes (mixed)--Bottom BTX extract/coal tar light oil	50
2701-01	Naphthalene--Separation from coal tar distillate	600
1007-01	Creosote--Distillation of coal tar light oil	400
2981-01	Pitch tar residue--Separation from coal tar light oil dist.	3000

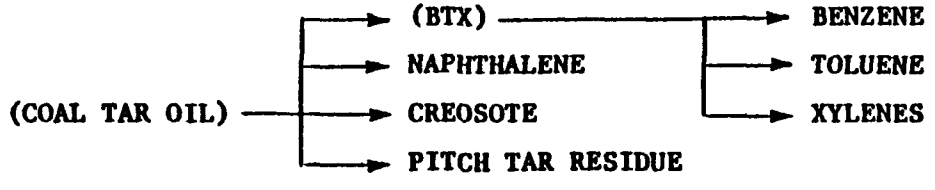


Figure 13. Chemical Tree - GPC 512 Coal Tar Products

Code	Product/Process	Average Daily Prod. x 1000 lb.
1244-01	1,2-Dichloroethane (EDC)--Direct chlorination of ethylene	1200
3520-03	Vinyl chloride--Thermal cracking of EDC	725

(ETHYLENE) ⟶ 1,2-DICHLOROETHANE ⟶ VINYL CHLORIDE

Figure 14. Chemical Tree - GPC 513 EDC/Vinyl Chloride

Code	Product/Process	Average Daily Prod. x 1000 lb.
3295-02	Tetrachloroethylene--Chlorination of EDC	150
0810-04	Carbon tetrachloride--Co-product of tetrachloroethylene	100
3410-03	Trichloroethylene--Chlorination of EDC	75
3530-02	Vinylidene chloride--Dehydrochlorination of 1,1,2-tri-chloroethane	50

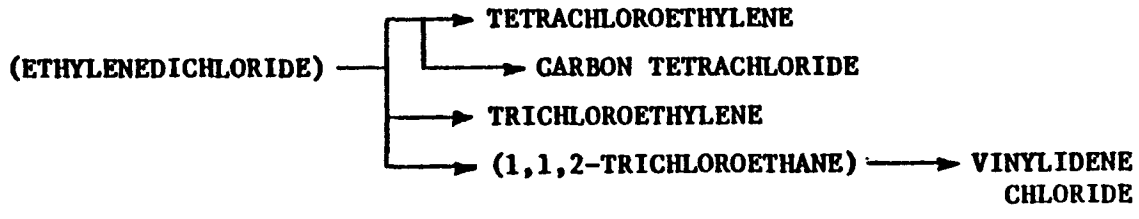


Figure 15. Chemical Tree - GPC 514 Chlorinated Solvents

Code	Product/Process	Average Daily Prod. x 1000 lb.
0810-01	Carbon tetrachloride--Chlorination of methane	150
0930-02	Chloroform--Chlorination of methane	250
2620-02	Methylene chloride--Chlorination of methane	500
2560-03	Methyl chloride--Chlorination of methane	100

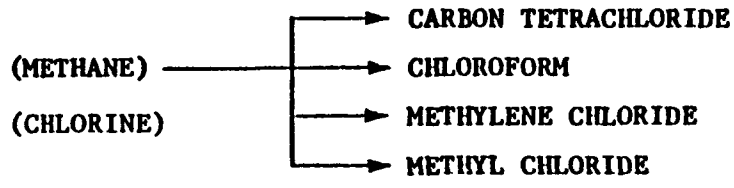


Figure 16. Chemical Tree - GPC 515 Chloromethanes

Code	Product/Process	Average Daily Prod. x 1000 lb.
0890-01	Chlorobenzene--Chlorination of benzene	140
1216-01	o-Dichlorobenzene--Chlorination of benzene	30
1220-01	p-Dichlorobenzene--Chlorination of benzene	70
3393-01	1,2,4-Trichlorobenzene--By-product of benzene chlorination	5
0530-01	Benzyl chloride--Chlorination of toluene	110
0949-01	m-Chloronitrobenzene--Chlorination of nitrobenzene	20

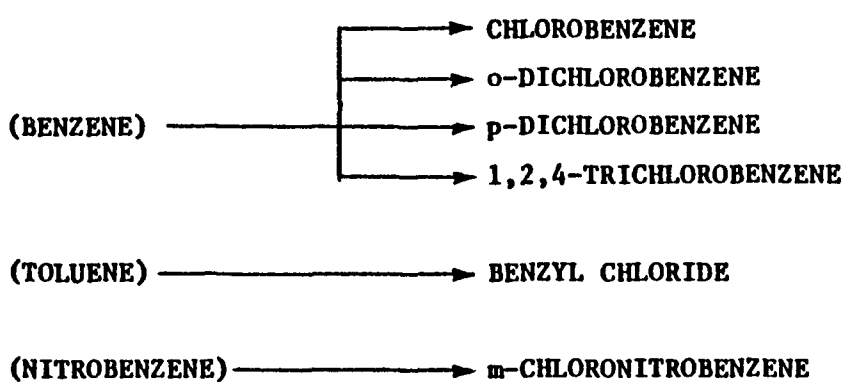


Figure 17. Chemical Tree - GPC 516 Chloro-Benzene/Toluene

Code	Product/Process	Average Daily Prod. x 1000 lb.
0300-04	Aniline-Nitrobenzene hydrogenation	400
2770-01	Nitrobenzene	500

(BENZENE) ⟶ NITROBENZENE ⟶ ANILINE

Figure 18. Chemical Tree- GPC 517 Nitrobenzene/ Aniline

Code	Product/Process	Average Daily Prod. x 1000 lb.
2770-01	Nitrobenzene--Nitration of benzene	310
0300-04	Aniline--Nitrobenzene hydrogenation	230
1550-01	Dinitrotoluene (mixed)--Nitration of toluene	360
3351-01	Toluene diamine (mixture)--Cat. hydrogenation of DNT	260
3354-01	Toluene 2,4-diisocyanate--Phosgenation of 2,4-TDA	200
3355-01	Toluene diisocyanates (mixture)--Phosgenation of TDA	100
3013-01	Polymeric methylene dianiline--React. aniline + formaldehyde	225
3015-01	Polymeric methylene diphenyl diisocyanate--MDA + phosgene	250
2200-01	Hydroquinone--Oxidation of aniline via quinone	50

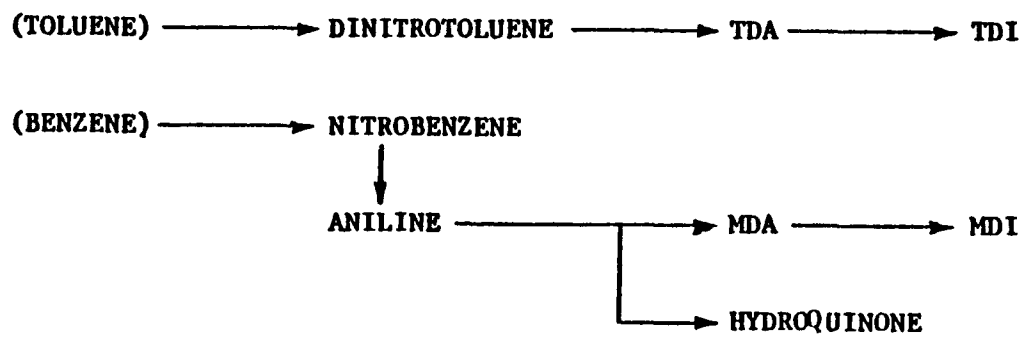


Figure 19. Chemical Tree - GPC 518 Polyurethane/Intermediates

Code	Product/Process	Average Daily Prod. x 1000 lb.
2960-02	Phthalic anhydride--Oxidation of o-xylene	225

(o-XYLENE) ⟶ PHTHALIC ANHYDRIDE

Figure 20. Chemical Tree - GPC 519: Phthalic Anhydride

Code	Product/Process	Average Daily Prod. x 1000 lb.
2430-01	Maleic anhydride--Benzene oxidation	95

(BENZENE) ⟶ MALEIC ANHYDRIDE

Figure 21. CHEMICAL TREE - GPC 520 - MALEIC ANHYDRIDE

Code	Product/Process	Average Daily Prod. x 1000 lb.
2960-01	Phthalic anhydride--Oxidation of naphthalene	350
2859-01	Bis 2-ethylhexyl phthalate--Phthalic anhyd. + alcohol esterification	250
2863-01	C_{11}-C_{14} phthalates--Phthalic anhyd. + alcohol esterification	150
2883-01	Diethyl phthalate--Phthalic anhyd. + alcohol esterification	50
2886-01	Butyl benzyl phthalate--P.A. ester. of alcohol + benzyl chloride	150
3300-01	Tetrachlorophthalic anhydride--Chlorination of phthalic anhydride	25

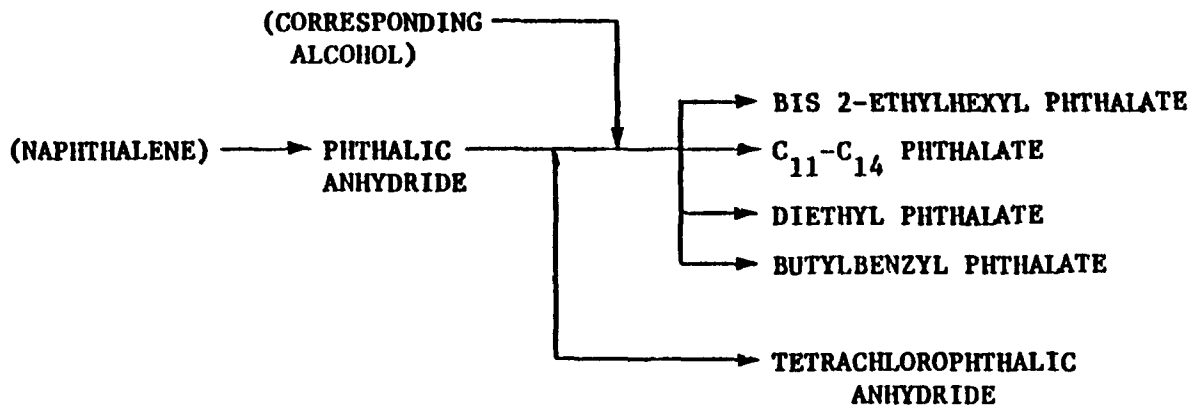


Figure 22. Chemical Tree - GPC 521 Phthalate Esters

Code	Product/Process	Average Daily Prod. x 1000 lb.
0195-01	Nonyl/octyl phenols--Alkylation of phenol	25
1666-02	Alkylphenol ethoxylates--Phenol + ethylene oxide	75
2951-02	Diphenylisodecyl phosphate--$POCl_3$ + isodecyl alcohol	75

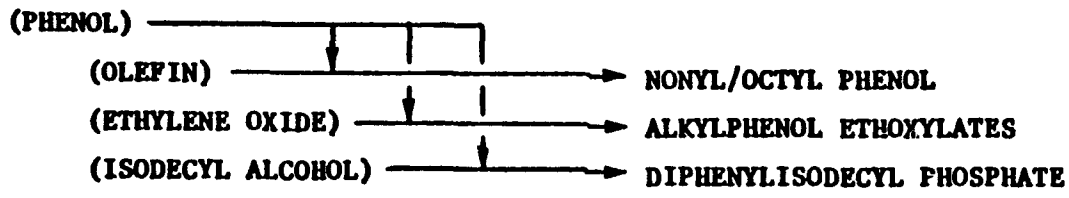


Figure 23. Chemical Tree - GPC 522: Miscellaneous Phenol Derivatives

Code	Product/Process	Average Daily Prod. x 1000 lb.
2910-02	Phenol--Cumene oxidation and cleavage	450
0090-01	Acetone--Cumene oxidation and cleavage	275
2690-01	α-Methylstyrene--By-product of acetone/phenol	50
0560-01	Bisphenol-A---Condensation of acetone with phenol	600

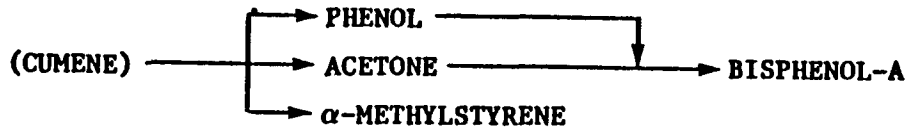


Figure 24. Chemical Tree - GPC 523 Phenol/Acetone

Code	Product/Process	Average Daily Prod. x 1000 lb.
0160-80	Acrylic acid--Oxidation of propylene via acrolein	500
0165-09	n-Butyl acrylate--Acrylic acid + n-butanol	75
0165-10	Ethyl acrylate--Acrylic acid + ethanol	500
0165-11	Ethylhexyl acrylate--Acrylic acid + ethylhexanol	25
0165-12	Isobutyl acrylate--Acrylic acid + isobutanol	125
2460-01	Methacrylic acid--Acetone cyanohydrin process	250
2470-02	Butyl methacrylate--Esterification of methacrylic acid + butanol	75
2665-02	Methyl methacrylate--Methanolysis of acetone cyanohydrin	250

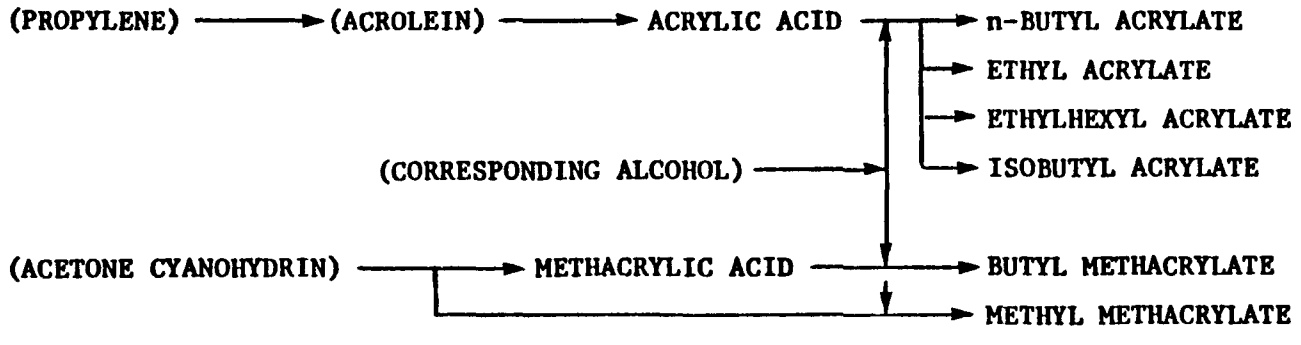


Figure 25. CHEMICAL TREE - GPC 524 - ACRYLATES/METHACRYLATES

Code	Product/Process	Average Daily Prod. x 1000 lb.
0130-01	Acetylene--Partial oxidation of methane	80
0160-03	Acrylic acid--From acetylene, CO and water	200
2500-02	Methanol--L.P. synthesis from natural gas via synthetic gas	900
2040-01	Formaldehyde--Dehydro./oxidation of methanol-silver process	950
0070-05	Acetic acid--Carbonylation of methanol	750

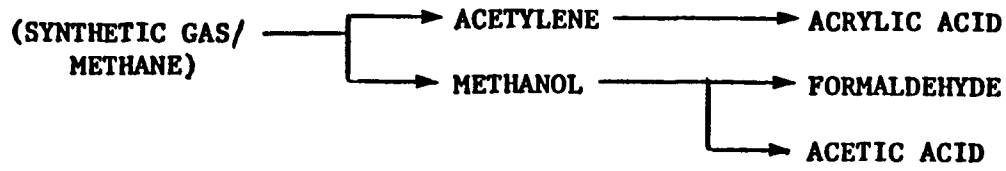


Figure 26. CHEMICAL TREE - GPC 525 - DERIVATIVES OF METHANE/SYNTHETIC GAS

Code	Product/Process	Average Daily Prod. x 1000 lb.
0030-02	Acetaldehyde--Oxidation of ethylene	1150
0070-04	Acetic acid--Oxidation of acetaldehyde	1000
0080-02	Acetic anhydride--From acetic acid by ketene process	1000
0820-03	Cellulose acetate resin--Cellulose + acetic anhydride/ hydrolysis (acid cat.)	500
1450-01	Diketene--Dimerization of ketene-acetic acid	40

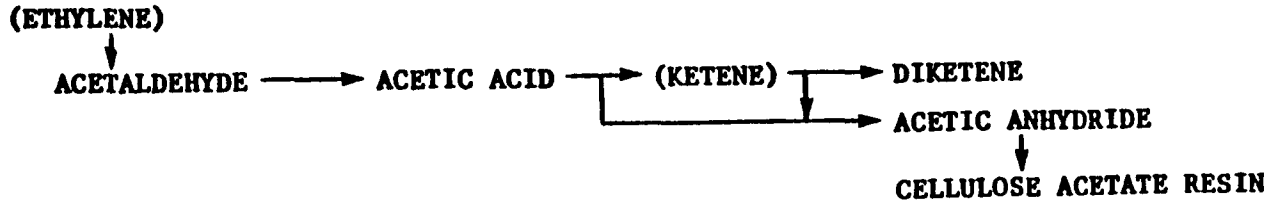


Figure 27. Chemical Tree - GPC 526 Cellulose Acetate and Intermediates

Code	Product/Process	Average Daily Prod. x 1000 lb.
3570-02	p-Xylene--Isomerization/crystallization of mixed xylenes	1050
3280-01	Terephthalic acid--Cat. oxidation of p-xylene	1550
0070-16	Acetic acid--Co-product of TPA by oxidation of acetaldehyde	350
1530-01	Dimethyl terephthalate--Esterification of TPA	1800

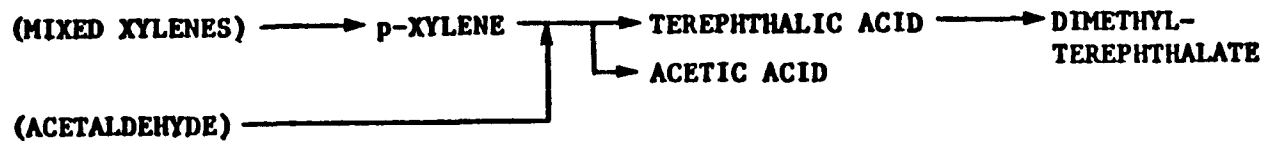


Figure 28. Chemical Tree - GPC 527 Polyester Intermediates

Code	Product/Process	Average Daily Prod. x 1000 lb.
1135-01	Cyclohexanol/one (mixed)--Oxidation of cyclohexane	225
0180-01	Adipic acid--Oxidation of cyclohexanol	100
0785-09	Caprolactam--From cyclohexanone via oxime	150
0785-06	Caprolactam--From phenol via cyclohexanone oxime	200
2825-01	Nylon 6 resin--Polycondensation from caprolactam	300

Figure 29. Chemical Tree - GPC 528: Nylon 6 Resin & Intermediates

Code	Product/Process	Average Daily Prod. x 1000 lb.
1135-01	Cyclohexanol/one (mixed)--Oxidation of cyclohexane	550
0180-03	Adipic acid--Oxidation of cyclohexanol/one mix	700
0180-80	Adipic acid--Oxidation of cyclohexane via ol/one	100
0185-04	Adiponitrile--Ammonolysis & dehydration of adipic acid	75
0185-05	Adiponitrile--Electrohydrodimerization of acrylonitrile	475
2165-02	Hexamethylene diamine--Hydrogenation of adiponitrile	550
* 2824-01	Nylon salt--Adipic acid + HMDA/aqueous soln.	1000
2824-02	Nylon salt--Adipic acid + HMDA/methanol soln.	100

* No wastewater reported in verification.

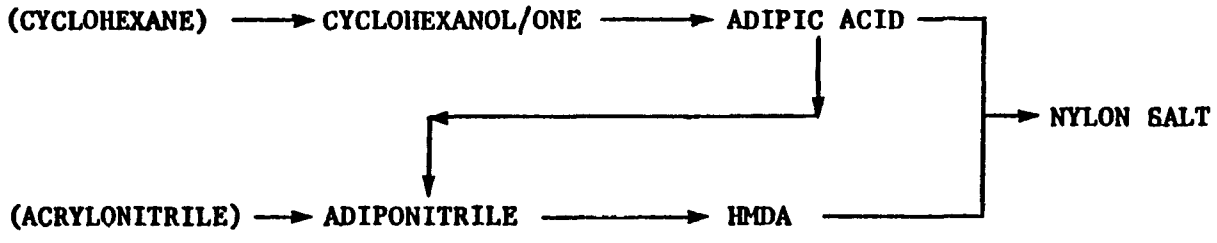


Figure 30. Chemical Tree - GPC 529 Nylon 66 Intermediates

Code	Product/Process	Average Daily Prod. x 1000 lb.
0189-01	Alkyd Resins--Condensation polymerization	50

(POLYHYDRIC ALCOHOL,
POLYBASIC ACID, → ALKYD RESINS
FATTY ACIDS)

Figure 31. Chemical Tree - GPC 530 Alkyd Resins

Code	Product Process	Average Daily Prod. x 1000 lb.
0189-01	Alkyd resins--Condensation polymerization	40
3501-01	Unsaturated polyester resins--React maleic/phthalic/glycol/ styrene/MMA	60

(POLYHYDRIC ALCOHOL, POLYBASIC ACID, FATTY ACIDS) → ALKYD RESINS

(MALEIC ANHYD., PHTHALIC ANHYD., GLYCOLS, ETC.) → UNSATURATED POLYESTER RESINS

Figure 32. Chemical Tree - GPC 531 Alkyds/Unsatd Polyester Resin

Code	Product/Process	Average Daily Prod. x 1000 lb.
0189-01	Alkyd resins--Condensation polymerization	65
0155-03	Acrylic resin--Solution polymerization	40
3501-01	Unsaturated polyester resins--React maleic/phthalic/ glycol/styrene/MAA	85

(POLYHYDRIC ALCOHOL, POLYBASIC ACID, FATTY ACIDS) → ALKYD RESINS

(ACRYLIC ESTERS) → ACRYLIC RESIN

(MALEIC ANHYD., PHTHALIC ANHYD., GLYCOLS, ETC.) → UNSATURATED POLYESTER RESINS

Figure 33. CHEMICAL TREE - GPC 532 - ALKYDS/ACRYLIC/POLYESTER RESINS

Code	Product/Process	Average Daily Prod. x 1000 lb.
3501-01	Unsaturated polyester resins--React maleic/phthalic/ glycol/styrene/MAA	210

(MALEIC ANHYD.,
PHTHALIC ANHYD., ⟶ UNSATURATED POLYESTER RESINS
GLYCOLS, ETC.)

Figure 34. CHEMICAL TREE - GPC 533 - UNSATURATED POLYESTER RESINS

Code	Product/Process	Average Daily Prod. x 1000 lb.
0153-01	Acrylic latex--Emulsion polymerization	220
0155-03	Acrylic resins--Solution polymerization	80

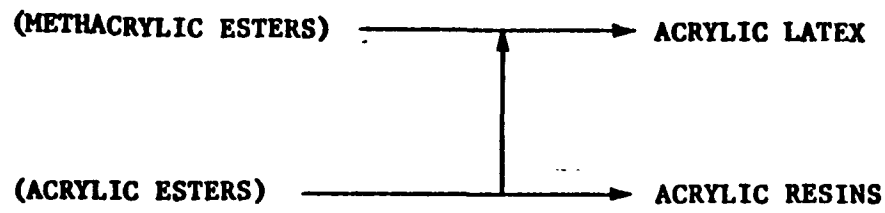


Figure 35. Chemical Tree - GPC 534 - Acrylic Latex/Resins

Code	Product/Process	Average Daily Prod. x 1000 lb.
0153-01	Acrylic latex--Emulsion polymerization	150

(ACRYLIC ESTERS, METHACRYLIC ESTERS) → ACRYLIC LATEX

Figure 36. Chemical Tree - GPC 535 Acrylic Latex

Code	Product/Process	Average Daily Prod. x 1000 lb.
3040-01	Polyvinyl acetate--Emulsion polymerization	100
0153-01	Acrylic latex--Emulsion polymerization	50

(VINYL ACETATE) ⟶ POLYVINYL ACETATE

(ACRYLIC ESTERS, METHACRYLIC ESTERS) ⟶ ACRYLIC LATEX

Figure 37. Chemical Tree - GPC 536 Polyvinyl Acetate/Acrylic Latex

Code	Product/Process	Average Daily Prod. x 1000 lb.
3040-01	Polyvinyl acetate--Emulsion polymerization	60

(VINYL ACETATE) ⟶ POLYVINYL ACETATE

Figure 38. Chemical Tree - GPC 537 Polyvinyl Acetate

Code	Product/Process	Average Daily Prod. x 1000 lb.
3040-01	Polyvinyl acetate--Emulsion polymerization	90
3042-80	Polyvinyl alcohol--Soln. polymerization of vinyl acetate/caustic hydrolysis	140
0070-13	Acetic acid--By-product of polyvinyl alcohol	120

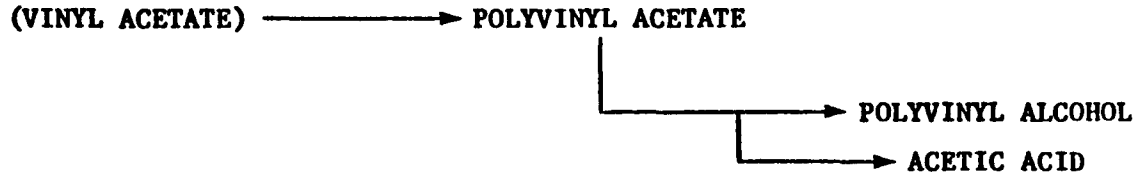


Figure 39. Chemical Tree - GPC 538 Polyvinyl Acetate/Polyvinyl Alcohol

Code	Product/Process	Average Daily Prod. x 1000 lb.
3048-01	Polyvinyl chloride--Emulsion polymerization	100
3048-02	Polyvinyl chloride--Suspension polymerization	340

(VINYL CHLORIDE) ⟶ POLYVINYL CHLORIDE

Figure 40. CHEMICAL TREE - GPC 539 POLYVINYL CHLORIDE

Code	Product/Process	Average Daily Prod. x 1000 lb.
3048-02	Polyvinyl chloride--Suspension polymerization	350

(VINYL CHLORIDE) ⟶ POLYVINYL CHLORIDE

Figure 41. Chemical Tree - GPC 540 Polyvinyl Chloride

Code	Product/Process	Average Daily Prod. x 1000 lb.
3048-02	Polyvinyl chloride--Suspension polymerization	300
3048-04	Polyvinyl chloride--Bulk polymerization	220

(VINYL CHLORIDE) ⟶ POLYVINYL CHLORIDE

Figure 42. Chemical Tree - GPC 541 Polyvinyl Chloride

Code	Product/Process	Average Daily Prod. x 1000 lb.
0005-01	ABS resin--Emulsion polymerization	260
3172-01	SAN resin--Suspension polymerization	150
3235-01	Styrene-Butadiene resin--Emulsion process	70

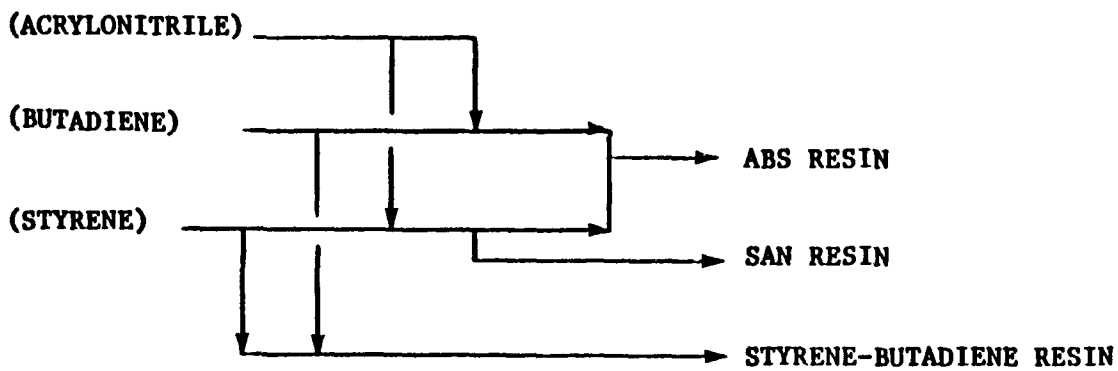


Figure 43. Chemical Tree - GPC 542 ABS/SAN/SB Resins

Code	Product/Process	Average Daily Prod. x 1000 lb.
2825-01	Nylon 6 resin--From caprolactam	85

(CAPROLACTAM) ⟶ NYLON 6 RESIN

Figure 44. Chemical Tree - GPC 543 Nylon 6 Resin

Code	Product/Process	Average Daily Prod. x 1000 lb.
2825-03	Nylon 66 resin--From nylon salt	220
0156-01	Acrylic fiber--Suspension poly/wet spinning	1100

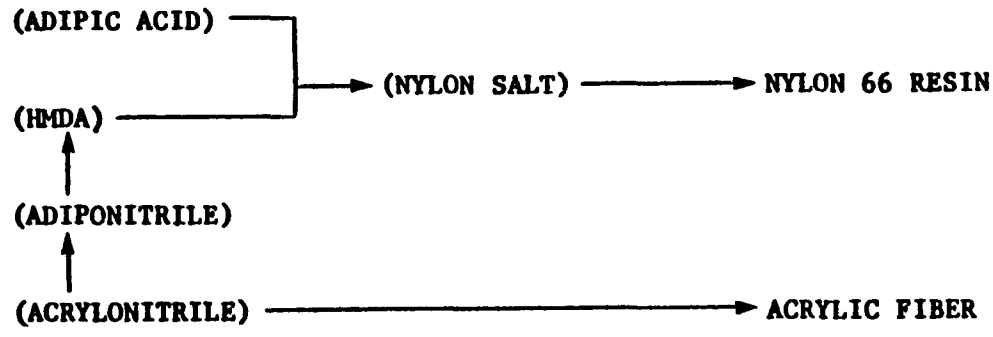


Figure 45. Chemical Tree - GPC 544: Nylon 66 Resin/Acrylic Fiber

Code	Product/Process	Average Daily Prod. x 1000 lb.
3006-21	Polyester resin--From TPA and ethylene glycol	340
3006-31	Polyester fiber--Melt spinning from TPA and E.G. *	340
3145-01	Rayon--Viscose process	275

* No wastewater reported in verification.

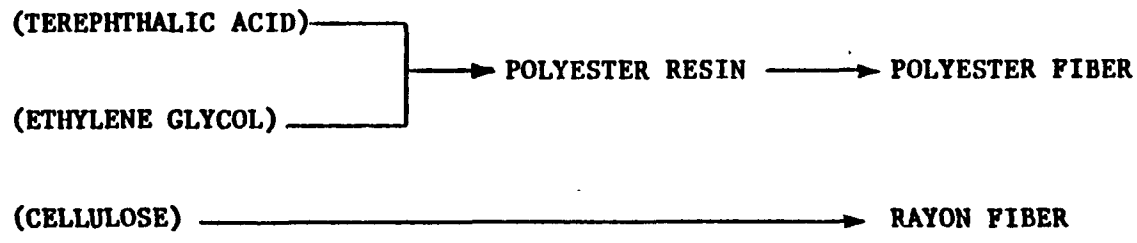


Figure 46. Chemical Tree - GPC 545 Polyester/Rayon Fibers

Code	Product/Process	Average Daily Prod. x 1000 lb.
3008-01	Polyethylene resin--Solution polymerization (HDPE)	300
3008-04	Polyethylene resin--High pressure polymer (LDPE)	650

(ETHYLENE) ⟶ POLYETHYLENE

Figure 47. Chemical Tree - GPC 546 Polyethylene Resin

Code	Product/Process	Average Daily Prod. x 1000 lb.
1656-01	Epoxy resins--Epichlorohydrin + bisphenol-A	8
0155-03	Acrylic resins--Solution polymerization	12
3030-04	Polystyrene--Bulk polymerization without rubber	80
2856-01	Petroleum hydrocarbon resins--Polymerization of cyclopentadiene dimer	140

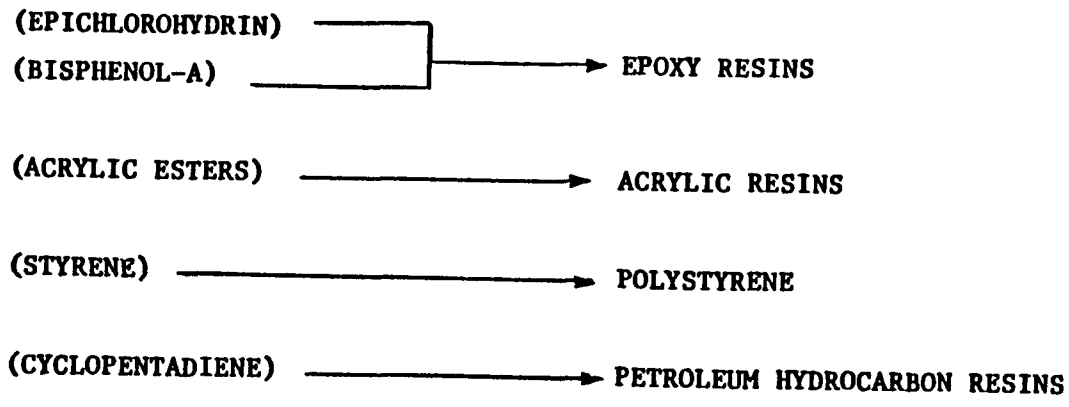


Figure 48. CHEMICAL TREE - GPC 547 - VARIOUS RESINS

Code	Product/Process	Average Daily Prod. x 1000 lb.
3030-02	Polystyrene--Suspension polymerization	320

(STYRENE) ⟶ POLYSTYRENE

Figure 49. Chemical Tree - GPC 548 Polystyrene

Code	Product/Process	Average Daily Prod. x 1000 lb.
3030-04	Polystyrene—Bulk polymerization without rubber	150

(STYRENE) ——————————→ POLYSTYRENE

Figure 50. Chemical Tree - GPC 549 Polystyrene

Code	Product/Process	Average Daily Prod. x 1000 lb.
3020-03	Polypropylene resin--Solution polymerization	450

(PROPYLENE) ⟶ POLYPROPYLENE

Figure 51. CHEMICAL TREE - GPC 550 - POLYPROPYLENE

Code	Product/Process	Average Daily Prod. x 1000 lb.
2905-01	Phenolic resins--Polymerization of phenol with formaldehyde	70

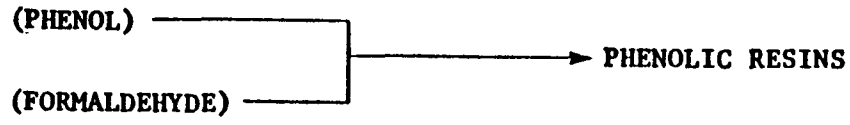


Figure 52. Chemical Tree - GPC 551 Phenolic Resins

Code	Product/Process	Average Daily Prod. x 1000 lb.
2905-01	Phenolic resins--Polymerization of phenol with formaldehyde	110
3506-01	Urea resins--Polymerization of urea with formaldehyde	110
2040-01	Formaldehyde--Dehydrogenation/oxidation of methanol-silver process	110

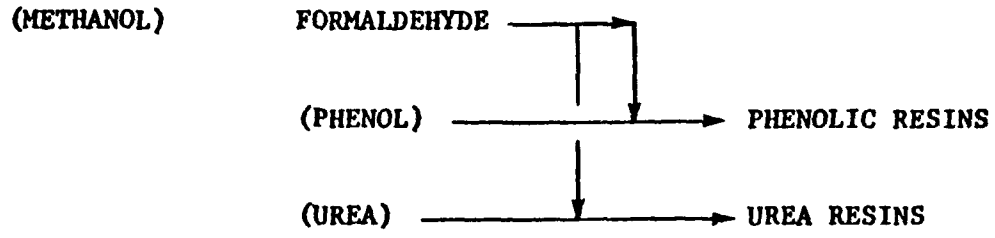


Figure 53. Chemical Tree - GPC 552 Phenolic/Urea Resins

Code	Product/Process	Average Daily Prod. x 1000 lb.
2905-01	Phenolic resins--Polymerization of phenol with formaldehyde	140
2443-01	Melamine resins--Polymerization of melamine with formaldehyde	40
3506-01	Urea resins--Polymerization of urea with formaldehyde	140

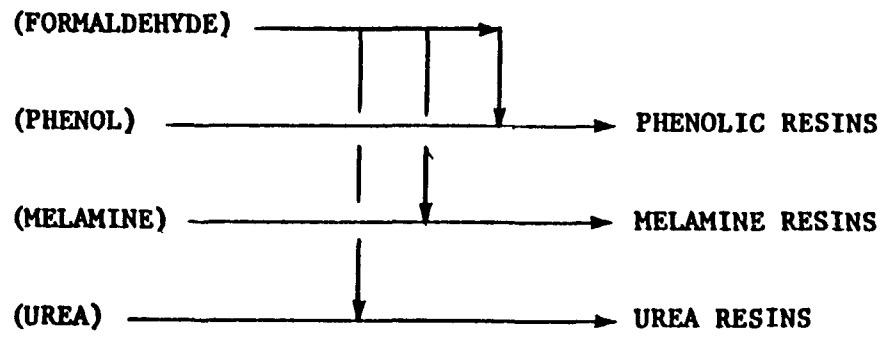


Figure 54. Chemical Tree - GPC 553 Phenolic/Melamine/Urea Resins

Code	Product/Process	Average Daily Prod. x 1000 lb.
2443-01	Melamine resins--Polymerization of melamine with formaldehyde	90
3506-01	Urea resins--Polymerization of urea with formaldehyde	120

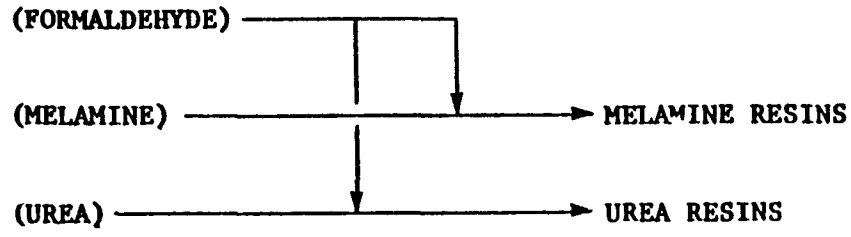


Figure 55. Chemical Tree - GPC 554 Melamine/Urea Resins

APPENDIX H

HEALTH AND ENVIRONMENTAL EFFECTS OF PRIORITY POLLUTANTS

APPENDIX H

HEALTH AND ENVIRONMENTAL EFFECTS OF PRIORITY POLLUTANTS

A. GENERAL

This appendix presents a description of the toxic human health and environmental effects associated with each of the 108 priority pollutants (see Table VI-1) that the Agency is considering regulating with BAT, NSPS or pretreatment standards. The priority pollutants are listed in alphabetical order by the fraction in which they appear -- volatile organic compounds, acid extractable organic compounds, base/neutral extractable organic compounds, metals and cyanide, and polychlorinated biphenyls.

B. PRIORITY POLLUTANTS

1. Volatile Organic Compounds

Acrolein

Acrolein is a potent irritant of the eyes and nose in humans. Effects were observed within 5 minutes at levels of 0.58 mg/cu m (Albin 1962, as reported in USEPA 1980). Vapor concentrations of 23 mg/cu m were reported to be lethal within a short time (Henderson and Haggard 1943, as reported in USEPA 1980). Strong skin irritation was reported to result from dermal exposure to 10% acrolein in ethanol (Lacroix *et al*. 1976, as reported in USEPA 1980). *In vitro* studies have shown that acrolein is a potent inhibitor of the synthesis of human polymorphonuclear leukocytes chemotaxis (Bridges *et al*. 1977, as reported in USEPA 1980). The odor threshold for humans was reported to be 0.48 mg/cu m (Reist and Rex 1977, as reported in USEPA 1980).

Acute inhalation studies in rats, mice, rabbits, and guinea pigs have shown pathological changes in the lungs including edema, hyperemia, hemorrhages, and possible degenerative changes in the bronchial epithelium (Skog 1980, Pattle and Cullumbin 1956, Salem and Cullumbin 1960, as reported in USEPA 1980). Hyperemia and fatty degeneration of the liver and focal inflammatory changes in kidneys have been reported in rats administered lethal subcutaneous doses of acrolein (Skog 1950, as reported in USEPA 1980). Acrolein was reported to cause significant cardiovascular effects, including tachycardia (Basu *et al*. 1972, as reported in USEPA 1980), and bradycardia, and decreased blood pressure (Egle and Hudgins 1974, as reported in USEPA 1980). In acute inhalation studies, acrolein was also found to increase respiratory resistance in guinea pigs exposed to 0.92-2.3 mg/cu m for up to 12 hours (Murphy *et al*. 1963, as reported in USEPA 1980); to be cytotoxic to the airway cells of hamsters exposed to 13.8 mg/cu m for 4 hours (Kilburn and McKenzie 1978, as reported in USEPA 1980); to reduce mucus flow rates in cats

(dosage unspecified; Carson et al. 1966, as reported in USEPA 1980); and to inhibit rabbit phagocytosis, adhesiveness, and calcium dependent ATPase activity in in vitro tests (Low et al. 1977, as reported in USEPA 1980).

In subacute inhalation tests, dogs and monkeys continuously exposed to 2.3-4.1 mg/cu m for 90 days subsequently developed eye and respiratory tract irritation. In the same study, dogs and monkeys exposed repeatedly to 8.5 mg/cu m for eight hours per day, five days per week, for six weeks developed pathological changes in the lungs (squamous metaplasia and basal cell hyperplasia of the trachea) (Lyon et al. 1970, as reported in USEPA 1980). Hamsters, rats, and rabbits exposed by inhalation to 11.3 mg/cu m for 6 hours/day for 13 weeks showed signs of eye irritations, decreased food consumption and decreased weight gain (Feron et al. 1978, as reported in USEPA 1980). Epithelial metaplasia and inflammation of the nasal cavity were observed in a chronic toxicity study in hamsters exposed to 9.2 mg/cu m for 7 hours/day for 52 weeks (Feron and Kruysse 1977, as reported in USEPA 1980). In a subacute oral toxicity study, rats exposed to acrolein in drinking water at concentrations up to 200 mg/liter for 90 days only showed slight weight reduction at the highest level tested. This effect was attributed to unpalatability of drinking water (Albin 1972, as reported in USEPA 1980). Acrolein has been shown to be mutagenic in different short-term bacterial assays (USEPA 1980). The carcinogenic potential of this compound has not yet been established (USEPA 1980).

The acute toxicity of acrolein on freshwater aquatic organisms is reflected by static 96-hour LC50 values of 0.057-0.080 mg/liter in cladoceran (mean acute value of 0.068 mg/liter for the species), 0.09-0.10 mg/liter in the bluegill, and 0.16 mg/liter in the largemouth bass. Aquatic macrophytes were destroyed or badly scorched after one week of exposure to 25 mg/liter. After 24-hour exposure to 10 mg/liter, 98% of adult snails and 100% of snail embryos died. In a total of nine short-term exposures with seven fish species, acute toxicity values ranged from 0.046 to 0.115 mg/liter. Flavor impairment of rainbow trout flesh was reported to occur up to 4 days after a 4-hour exposure to 0.090 mg/liter (USEPA 1980). Toxicity to the saltwater eastern oyster was manifested by a 50% decrease in shell growth at 0.055 mg/liter using a flow through test. Chronic toxicity was observed in cladoceran at 21 µg/liter and the fathead minnow at 21 µg/liter. The effects of acrolein on fresh and saltwater plants have not been studied (USEPA 1980).

EPA has established an ambient water quality criterion of 320 mg/liter for the protection of human health from the toxic properties of acrolein ingested through water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for acrolein.

Acrylonitrile

Acrylonitrile is an acute poison that is a severe skin and eye irritant (NAS 1980). Acute toxicity can cause nasal and respiratory oppressions, vomiting, nausea, weakness, fatigue, headache, and diarrhea (Patterson et

al. 1976, as reported in USEPA 1980). Occupational studies have associated acrylonitrile with diseases of the peripheral nervous system, stomach, duodenum, and skin (Goncharova et al. 1977, Shirshova et al. 1975 and Stamov et al. 1976, as reported in USEPA 1980). Other studies have shown changes in the heart and circulation, blood methemoglobin content, and clinical blood values; lowered blood cell counts; and mild liver injury (Sakarai and Kusimto 1972, Ostrovskaya et al. 1976, Zotova 1975, and Shustov and Mavrina 1975, as reported in USEPA 1980).

Acrylonitrile has been characterized as a serious hazard in inhalation studies (Union Carbide Corporation 1970, as reported in NAS 1980). In one study, all exposed rats died within 5 minutes while breathing saturated air; in another study, all exposed rats died in 4 hours breathing 0.33 mg/liter. Acute oral toxicity values for acrylonitrile range from 27 to 128 mg/kg for mice (Benes and Cerna 1959, Zeller et al. 1969, as reported in NAS 1980) and from 78 to 93 mg/kg for rats (Benes and Cerna 1959, Smyth and Carpenter 1948, as reported in NAS 1980).

A 90-day oral toxicity study, incorporating 0.66 and 0.99 mg/liter of acrylonitrile in the drinking water of rats resulted in the animals' death before the end of the experiment (NRDC 1976, as reported in USEPA 1980).

Rabbits and rats breathing 50 mg/cu m for 6 months developed changes in peripheral blood patterns, functional disorders in the respiratory and cardiovascular systems, and signs of neuronal lesions in the central nervous system (Knobloch et al. 1972, as reported in USEPA 1980).

Chronic inhalation exposures of dogs, cats, rats, guinea pigs, rabbits, and monkeys to concentrations ranging from 0.12 mg/liter to 0.33 mg/liter produced irritation of the eyes and nose, loss of appetite, gastrointestinal disturbances, and incapacitating weakness of the hind legs (Dudley et al. 1942, as reported in NAS 1980). Central nervous system effects also occurred in inhalation studies of rats exposed to 80 ppm for one year (USEPA 1980).

The mutagenicity of acrylonitrile has been demonstrated in the *Salmonella typhimurium* test and in *Escherichia coli* W P2 strains. Fetal malformations and maternal toxicity were observed in rats administered 65 mg/kg/day by gavage on days 6-15 of gestation. Other embryotoxic effects included reduced fetal body weight and crown-rump length and increased incidences of minor skeletal variants.

Acrylonitrile was carcinogenic in rats administered 100 or 300 mg/liter for 12 months in the drinking water. Cancer of the stomach, central nervous system, and inner ear were reported (USEPA 1980).

The toxicity of acrylonitrile to freshwater aquatic organisms was demonstrated in the cladoceran, fathead minnow, guppy, and bluegill, with 96-hour LC50 values ranging from 7.55 to 33.5 mg/liter. The only information on saltwater species is a 24-hour LC50 value of 24.5 mg/liter for the pinfish. No other data including the effects of aquatic plant life are available (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to acrylonitrile through ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at the present time, a level of 0.058 µg/liter, corresponding to an estimated lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for acrylonitrile.

Benzene

Single exposure to benzene at 20,000 ppm has caused death within 5-10 minutes in humans and produced acute toxic effects including nausea, giddiness, headache, unconsciousness, convulsions, and paralysis (Browning 1965 and Eckardt 1973, as reported in NAS 1977). The chronic occupational exposure of benzene to humans has been reported to produce thrombocytopenia, leukopenia, and anemia. In more severe cases of benzene hematoxicity, pancytopenia and acute myeloblastic leukemia have been observed (USEPA 1980). Increased incidence of chromosomal aberrations with aneuploidy and breakage has been observed in nonsymptomatic workers exposed to benzene (NAS 1980).

In chronic animal studies, leukopenia has been reported in rats exposed to 88 ppm, 7 hours per day, for up to 269 days. Below this level, no blood changes were observed in rats, guinea pigs and rabbits (Wolf et al. 1956, as reported in USEPA 1980). Other studies in which leukopenia developed include a study in which rats were given 132 oral doses of 50 mg/kg over 6 months (Wolf et al. 1956, as reported in USEPA 1980) and a study of rats exposed to 44 ppm for 5 hours/day, 4 days/week, for 5 to 7 weeks (Deichmann et al. 1963, as reported in USEPA 1980). Abnormalities of the spleen and lungs were observed in rats exposed to 31-47 ppm, 20 hours per week, for 6-31 weeks (Deichmann et al. 1963, as reported in USEPA 1980). Pregnant mice administered 3 ml/kg gave birth to offspring with malformations and decreased white cell counts (Watanabe and Yoshida 1970, as reported in USEPA 1980). In another study, mice administered 0.3-1.0 ml/kg benzene by gavage during days 6-15 of gestation developed significant maternal lethality and embryonic resorptions (Nawrot and Staples 1979 as reported in USEPA 1980). Inhalation studies in rats conducted at various times before and during pregnancy, at concentrations ranging from 210 mg/cu m to 1,000 mg/cu m produced no malformations but did decrease fetal weight gain (USEPA 1980). Benzene was not mutagenic in the Salmonella/microsome in vitro assay (Lyon 1975, Shahin 1977, Simon et al. 1977, as reported in USEPA 1980). However, it has been reported that chromosomal abnormalities in bone marrow cells have resulted from benzene exposure in a number of species including rats, rabbits, mice, and amphibians (USEPA 1980). Benzene-induced leukemogenesis has not been demonstrated in laboratory animals (USEPA 1980).

A variety of freshwater organisms are sensitive to the affects of benzene. Static 96-hour LC50s have been reported in juvenile rainbow trout (5.3 mg/liter), goldfish (34.42 mg/liter), fathead minnow (33.47 mg/liter), guppy (36.6 mg/liter), mosquitofish (386 mg/liter), bluefish (22.5 mg/liter) and Daphnia magna (203-620 mg/liter). A 50% reduction in the cell numbers

of Chlorella vulgaris was seen after 48 hours of exposure to 530 mg/liter benzene. Most saltwater organisms exposed to benzene gave 96-hour LC50 values between 5 and 100 mg/liter. Striped bass, anchovy, and pacific herring are more sensitive species giving values between 5.8 and 25 mg/liter. Saltwater invertebrates appear to be less sensitive. Female pacific herring exposed to 700 mg/liter for 48 hours exhibited reduced survival of embryos at hatching and reduced survival of larvae through yolk absorption. The 96-hour LC50 values ranged from 27 mg/liter for the grass shrimp to 450 mg/liter for the copepod. Chronic effects for saltwater organisms have not been studied. Benzene concentrations of 20-100 mg/liter inhibited growth in three species of algae (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to benzene through ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at the present time, a criterion of 0.66 µg/liter, corresponding to an incremental lifetime cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for benzene.

Bromoform

Bromoform is regarded as highly toxic to humans by all major routes of exposure (lungs, GI tract, skin). However, information concerning the compound's toxicity in humans is not extensive. Acute inhalation exposures produce irritation of the respiratory tract, pharynx, and larynx, with lacrimation and salivation. Mild poisoning cases may be limited to headache, listlessness, and vertigo. In more severe cases, symptoms may include unconsciousness, loss of reflexes, convulsions, and death resulting from respiratory failure. Histopathological findings include fatty, degenerative and necrotic changes in the liver (USEPA 1980). No information on chronic toxicity in humans is available.

In studies with the mouse, LD50s of 1,820 and 1,400 mg/kg were reported following single subcutaneous and intragastric injections, respectively (USEPA 1980). Single subcutaneous doses of 278 and 1,112 mg/kg bromoform resulted in impaired liver function in the mouse (Kutob and Plaa 1962, as reported in USEPA 1980), and 10 daily injections of 100-200 mg/kg/day produced liver and kidney pathology in the guinea pig (NAS 1978, as reported in USEPA 1980). Reticuloendothelial system function (liver and spleen phagocytic activity) was suppressed in mice given intragastric doses of 0.3 to 125 mg/kg/day bromoform (Munson et al. 1978, as reported in USEPA 1980). Bromoform was shown to be mutagenic in in vitro tests with three strains of Salmonella typhimurium (Clayton and Clayton 1981). Inconclusive evidence for potential carcinogenic activity of bromoform has been reported by Theiss et al. (1977, as reported in USEPA 1980) using the strain A mouse lung tumor assay system. Mice were injected at doses of 4, 48, and 100 mg/kg 3 times/week for 6 to 8 weeks and were sacrificed 24 weeks after the first injection. A significant increase in lung tumors was observed only at the middle dose, and a dose-response relationship was not evident. Epidemiological studies provide some evidence

for a positive association between trihalomethane levels in drinking water supplies and the incidence of cancer. The results of these studies are limited, however, by inability to control for all confounding variables and limited monitoring data (USEPA 1980).

Acute aquatic toxicity for bromoform has been evaluated in two freshwater species. The 96-hour LC50 values reported for Daphnia magna and for the bluegill were 46,500 and 29,300 µg/liter, respectively. In studies with the mysid shrimp and sheepshead minnow, 96-hour LC50s were 24,400 and 17,900 µg/liter, respectively. In an embryo-larval study with sheepshead minnow, chronic effects were observed at 6,400 µg/liter (USEPA 1980). In freshwater and saltwater algae, 96-hour EC50 values of 112,000 and 12,300, respectively, were reported (based on effects in chlorophyll a) (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to bromoform through ingestion of contaminated water and aquatic organisms. However, since a zero level may not be obtainable at the present time, a level of 0.19 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life ambient water quality criterion for bromoform.

Carbon Tetrachloride

Carbon tetrachloride produces acute, subacute, and chronic poisoning with fatalities by ingestion, inhalation, and dermal routes in both humans and animals. In humans, carbon tetrachloride has been shown to cause liver and kidney damage (Dume et al. 1969 and Echardt 1965, as reported in NAS 1977). Signs and symptoms of acute toxicity include dyspnea, cyanosis, proteinuria, hematuria, jaundice, hepatomegaly, optic neuritis, ventricular fibrillation, eye, nose, and throat irritation, headache, dizziness, vomiting, abdominal cramps and diarrhea (NAS 1977). Chronic exposures generally result in gastrointestinal upset, such as nausea and vomiting, and nervous system symptoms, such as headache, drowsiness, and excessive fatigue (Browning 1961, as reported in NAS 1977). Hepatic cirrhosis and necrosis, renal damage, changes in blood enzymes, and increased serum bilirubin also result from chronic exposure (Busuttil et al. 1972, Litchfield and Garland 1974, as reported in NAS 1977).

In a series of studies to evaluate the carcinogenic potential of carbon tetrachloride, liver tumors were observed during lifetime exposures of mice, hamsters, and rats using different routes of exposure: hamsters were orally administered 6.25-12.5 µl/week for 30 weeks (Della-Porta et al. 1961, as reported in NAS 1977); mice were orally administered 0.1 ml, twice a week for 20-26 weeks (Confer and Stenger 1965, as reported in NAS 1977), rats were injected subcutaneously with 0.2-0.3 ml/100 g every two weeks (Kawasaki 1965, as reported in NAS 1977) and rats were administered 1.3 ml/kg orally twice a week for 12 weeks (Ruber and Glover 1967, as reported in NAS 1977). However, carbon tetrachloride was not found to be mutagenic or teratogenic (NAS 1977).

Carbon tetrachloride produces acute and chronic toxic effects on freshwater vertebrates and acute toxic effects on freshwater invertebrates. The 96-hour LC50 values for bluegill were reported as 27.3 and 125 mg/liter. A 48-hour LC50 value of 35.2 mg/liter was reported for Daphnia magna. The 96-hour LC50 values reported for tidewater silversides and Limanda limanda were 150 mg/liter and 50 mg/liter, respectively (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to carbon tetrachloride through the ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at the present time, a level of 0.40 µg/liter, corresponding to an estimated lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for carbon tetrachloride.

Chlorobenzene

Chlorobenzene has been identified as a respiratory irritant and a central nervous system depressant in humans (NAS 1977).

Chlorobenzene has an acute oral LD50 of approximately 3-3.4 mg/kg in rats (Vecerek et al. 1976, as reported in USEPA 1980). The rats died about 7 days after exposure and showed signs of many metabolic disturbances such as elevated levels of serum glutamic-oxaloacetic transaminase (SGOT), lactase dehydrogenase, alkaline phosphatase, blood urea nitrogen, and decreased levels of glycogen phosphorylase and blood sugars.

Chronic feeding studies administered in dogs and rats produced clinical and pathological changes (Knapp et al. 1971, as reported in USEPA 1980). Dogs administered 272.5 mg/kg/day, by gavage, 5 days/per week for a period of 90 days, exhibited gross and/or microscopic pathological changes in the liver, kidneys, gastrointestinal mucosa, and hematopoietic tissues. Clinical symptoms included an increase in immature leukocytes, low blood sugar, elevated serum glutamic-pyruvic transaminase (SGPT) and alkaline phosphatase. In chronic inhalation studies, rats, rabbits, and guinea pigs were exposed to 200, 475, and 1,000 ppm over 44 days (Irish 1963, as reported in USEPA 1980). Histopathological changes in the lungs, liver, and kidneys occurred in the high-dose group. The middle dose group exhibited an increase in liver weight and slight liver histopathology. No effects were reported in the low dose group.

Chlorobenzene was administered orally to rats in daily doses of 14.4-228 mg/kg for a total of 137 doses over 192 days. No blood or bone marrow changes were observed (Irish 1963, as reported in USEPA 1980). No studies have evaluated the carcinogenic potential of chlorobenzene, although an NCI bioassay is in progress.

The acute toxicity of chlorobenzene to various saltwater and freshwater species is reflected by LC50 values for cladoceran of 86 mg/liter, the

goldfish, 51.6 mg/liter, the fathead minnow, 29.1-33.9 mg/liter, the guppy, 45.3 mg/liter, the bluegill, 15.9-24.0 mg/liter, and the sheepshead minnow, 10.5 mg/liter. Chlorobenzene has also demonstrated acute toxic effects to freshwater and saltwater algae with 96-hour EC50 values ranging from 22.4 to 34.1 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 488 µg/liter for the protection of human health from the toxic properties of chlorobenzene through ingestion of water and contaminated aquatic organisms. Using available organoleptic data for controlling undesirable taste and odor of ambient water, the estimated level is 20 µg/liter.

EPA has not yet established an aquatic life water quality criterion for chlorobenzene.

Chlorodibromomethane

No data are available on the toxicity of chlorodibromomethane to humans, animals, or aquatic organisms.

EPA has not yet established ambient water quality criteria for chlorodibromomethane because of the lack of sufficient information.

Chloroethane

Of the chlorinated ethanes, monochloroethane is considered to be one of the least toxic. It is known to disturb cardiac rhythm (Goodman and Gilman 1975, as reported in USEPA 1980) and overdoses can lead to severe contractile failure of the heart (Doering 1975, as reported in USEPA 1980). Exposure to acute concentrations in humans has also been reported to cause neurologic symptoms (including central nervous system depression, headache, dizziness, incoordination, inebriation, and unconsciousness); abdominal cramps; respiratory tract irritation and respiratory failure; and skin and eye irritation. As a halogen-containing hydrocarbon, chloroethane is potentially toxic to the liver (USEPA 1980).

In a series of acute inhalation studies in guinea pigs, exposure to two percent chloroethane in air for 540 minutes produced histopathological changes in liver and kidneys; exposure to four percent chloroethane for the same period resulted in deaths. When guinea pigs were exposed to 23-24 percent chloroethane for five to ten minutes, unconsciousness and some deaths were reported (Sayers et al. 1929, as reported in Clayton and Clayton 1981). Repeated two-hour exposures for 60 days to 5,300 ppm chloroethane was reported to cause a decrease in the phagocytic activity of leukocytes, lowered hippuric acid formation in the liver, and histopathological changes in the liver, brain, and lungs (species tested not specified) (Troshina 1964, as reported in Clayton and Clayton 1981). In a study in which rats and dogs were exposed to chloroethane at concentrations of 0, 1,600, 4,000, or 10,000 ppm for six hours/day, five days/week for two weeks, the only observed effects were slight liver weight increase in the 4,000 and 10,000 ppm male rats and CNS depression

in the high dose animals (Dow Chemical Company, unpublished data, as reported in Clayton and Clayton 1981).

No data on the toxicity of chloroethane to aquatic organisms are available.

EPA has not yet established an ambient water quality criteria for chloroethane because of the lack of sufficient data.

2-Chloroethyl Vinyl Ether

No toxicity data for 2-chloroethyl vinyl ether in humans are available, and only limited acute toxicity information in animals has been published. The acute oral LD50 in the rat has been given as 250 mg/kg, and the acute dermal LD50 in the rabbit as 3,200 mg/kg (Smyth et al. 1949, as reported in USEPA 1980). Toxic effects were observed in rats exposed to 250 ppm 2-chloroethyl vinyl ether vapor for four hours (Carpenter et al. 1949, as reported in USEPA 1980). No chronic studies for this compound have been conducted.

In an acute toxicity study with the bluegill, the 96-hour LC50 was determined to be 354,000 µg/liter (USEPA 1980). No toxicity data are available for saltwater species.

EPA has not yet established ambient water quality criteria for 2-chloroethyl vinyl ether because of the lack of sufficient data.

Chloroform

Chloroform has been reported to cause severe adverse effects on the human body. Acute effects via skin absorption include local irritation, hyperemia, erythemia, and moisture loss (Malten et al. 1968, as reported in USEPA 1980); central nervous system depression and gastrointestinal irritation (Challen et al. 1958, as reported in USEPA 1980) and hepatic and renal damage (Fuhner 1923 and Althausen and Thoenes 1932, as reported in USEPA 1980). Oral doses of 44.6 and 148.3 g have produced severe nonfatal poisonings in humans; a dose of 296.6 g was fatal (Van Oettingen 1964, as reported in NAS 1980). Chronic effects in humans include central nervous system depression, loss of appetite, hallucinations, ataxia, nausea, rheumatic pain, and delirium (NIOSH 1974, as reported in USEPA 1980).

Oral LD50s have been estimated for male rats (0.8 ml/kg), mice (0.33 ml/kg), and dogs (1.0 ml/kg) (Kimura et al. 1971, Hill et al. 1975, Klassen and Plaa 1966, as reported in NAS 1977). Chloroform has been shown to produce liver tumors after oral administration of 138 mg/kg for 78 weeks. In this same study, kidney epithelial tumors were observed when an average dose level of 100 mg/kg was administered to rats by gavage for 78 weeks and sacrificed at 111 weeks (NCI 1976, as reported in NAS 1977). The fetuses from rats exposed to 489 mg/cu m (100 ppm) of chloroform 7 hours/day during the 6th to 15th day of gestation were reported to have an increased incidence in several abnormalities which included acaudia, imperforate anus, subcutaneous edema, missing ribs, and delayed ossification of sternebrae. At an exposure

of 147 mg/cu m (30 ppm) there was an increased incidence of delayed ossification of skull bones and of wavy ribs (Schwetz 1974, as reported in USEPA 1980). Maternal toxicity was observed in pregnant rats given oral chloroform doses of 126 mg/kg/day and greater. Doses of 316 mg/kg/day and greater caused acute toxic nephrosis and hepatitis and death of dams, as well as fetotoxicity. Oral doses of 100 mg/kg/day or higher were toxic to rabbits, dams, and fetuses (Thompson et al. 1974, as reported in USEPA 1980).

Ninety-six hour LC50 values for rainbow trout and bluegill were reported as 43.8-66.8 mg/liter and 100-115 mg/liter, respectively. Daphnia magna had a 48-hour LC50 value of 28.9 mg/liter. Anesthetization or death occurred at concentrations between 97 and 207 mg/liter for stickleback, goldfish, and orangespotted sunfish. Teratogenesis was produced in 40 percent of the embryo of rainbow trout exposed to 10.6 mg/liter for 23 days; 1.24 mg/liter produced 50 percent mortality of the embryo larval stage after a 27 day exposure. A 96-hour LC50 for pink shrimp was reported as 81.5 mg/liter. No other information on marine organisms or on aquatic plant life is available (USEPA 1980).

EPA has established a water quality criterion of zero for maximum protection of human health from the potential carcinogenic effects due to exposure to chloroform through ingestion of contaminated water and contaminated aquatic organisms. However, since zero may not be attainable at the present time, a criterion of 0.19 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, has been recommended.

EPA has not yet established an aquatic life water quality criterion for chloroform.

Dichlorobromomethane

No information is available on human toxicity to dichlorobromomethane. As a halomethane, it is reported that the compound is "probably narcotic in high concentrations" (USEPA 1980).

In mice, the LD50s for males and females administered dichlorobromomethane by gavage is 450 and 900 mg/kg, respectively. At doses between 500 and 4,000 mg/kg, histopathological examinations revealed fatty infiltration in livers, pale kidneys, and hemorrhage in kidneys, adrenal glands, lungs, and brain (Bowman et al. 1978, as reported in USEPA 1980). Cambell (1978, as reported in USEPA 1980) reported reduced water consumption and body weight in mice given 300 mg/liter dichlorobromomethane in drinking water. Cellular and humoral immune responses were suppressed in mice exposed by gavage to 125 mg/kg/day of dichlorobromomethane for 90 days. (Schuller et al. 1978, as reported in USEPA 1980). Suppression of hepatic phagocytic activity has also been reported in mice (USEPA 1980). Limited evidence for tertogenic properties of dichlorobromomethane are presented in a study by Schwetz et al. (1975, as reported in USEPA 1980), in which some fetal anomalies (unspecified) were observed among mice exposed to vapors at 8,375 mg/cu m for 7 hours/day during gestation days 6 to 15. Dichlorobromomethane was reported to be mutagenic in an in vitro test with Salmonella typhimurium (Simmon et al. 1977, as reported in USEPA 1980). Inconclusive evidence for

potential carcinogenic activity of dichlorobromomethane has been reported by Theiss et al. (1977, as reported in USEPA 1980) using the strain A mouse lung tumor assay system. Mice were injected at doses of 20, 40, and 100 mg/kg, three times/week for 6 to 8 weeks and were sacrificed 24 weeks after the first injection. A marginally significant incidence of lung tumors was observed only at the highest dose ($p < 0.067$).

No aquatic toxicity data are available for dichlorobromomethane. Available data for halomethanes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,000 µg/liter (USEPA 1980).

EPA has established ambient water quality criterion of zero for maximum protection of human health from the potential carcinogenic effects due to exposure to dichlorobromomethane through the ingestion of contaminated water and aquatic organisms. However, since the zero level may not be attainable at the present time, a level of 0.19 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for dichlorobromomethane.

1,1-Dichloroethane

1,1-Dichloroethane has been shown to cause marked excitation of the heart, central nervous system depression, respiratory tract irritation, and burning skin in humans. Liver injury was observed in experimental animals exposed to 4,000-17,500 ppm (species, route unspecified) (Sax 1975). The oral LD50 for the rat is 14 g/kg (Sax 1975). Retarded fetal development occurred in rats exposed to 24,250 mg/cu m (Schwetz et al. 1974, as reported in USEPA 1980). No information is available on the toxic effects of 1,1-dichloroethane on aquatic organisms (USEPA 1980).

EPA has not yet established ambient water quality criteria for 1,1-dichloroethane because of the lack of sufficient data.

1,2-Dichloroethane

In humans, 1,2-dichloroethane has been shown to produce central nervous system depression, gastrointestinal upset, and systemic injury to the liver, kidneys, lungs, and adrenals (USEPA 1979, as reported in USEPA 1980). Accidental oral ingestion of a single dose of 0.5-1.0 g/kg resulted in death; autopsy revealed liver necrosis and focal adrenal degeneration and necrosis (Wirtshafter and Schwartz 1939, Yodaiken and Babcock 1973, as reported in USEPA 1980). Acute toxic effects include nausea, vomiting, dizziness, internal bleeding, cyanosis, rapid but weak pulse, and unconsciousness. Chronic exposure to 1,2-dichloroethane has been shown to cause neurological changes, loss of appetite and other gastrointestinal problems, anemia, irritation of the mucous membranes, liver and kidney impairment, and in some cases death (NIOSH 1978, USEPA 1979, as reported in USEPA 1980).

Acute and subacute inhalation studies with dogs, rabbits, guinea pigs, rats, and mice indicate that 1,2-dichloroethane is toxic to the liver, bone marrow, blood, kidneys, myocardium, and sometimes the adrenals (Heppel et al. 1946, Liola et al. 1959, Liola and Fondacaro 1959, as reported in USEPA 1980). Chronic inhalation exposures to 100-400 ppm 1,2-dichloroethane, for 5-32 weeks in the guinea pig, monkey, rabbit, dog, and cat were reported to be toxic to the liver at concentration of 200 ppm and greater (Spencer et al. 1951, Hofman et al. 1971, as reported in USEPA 1980). Increased liver weights were observed in guinea pigs after a 32-week exposure to 100 ppm of 1,2-dichloroethane (Spencer et al. 1951, as reported in USEPA 1980).

Exposure of female rats to 57 mg/cu m (4 hrs/day, 6 days/week) for 6 months before breeding and throughout gestation resulted in a reduction in litter size, number of live births, and fetal weights (vozovaya 1974, as reported in USEPA 1980). 1,2-Dichloroethane was found to be carcinogenic at several sites to both rats and mice administered 50-300 mg/kg for 78 weeks by gavage (NCI 1978, as reported in USEPA 1980).

The acute toxic effects of 1,2-dichloroethane on freshwater organisms is reflected in 96-hour LC50 values for the fathead minnow (118 mg/liter) and the bluegill (431-550 mg/liter). Toxicity to saltwater organisms was demonstrated in the mysid shrimp at 113 mg/liter. Chronic toxicity was observed in the fathead minnow in concentrations ranging from 14 to 29 mg/liter. Toxicity to aquatic plants was observed in saltwater algae at concentrations ranging from more than 126 mg/liter to more than 433 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects from exposure to 1,2-dichloroethane through ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at present, a level of 0.94 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for 1,2-dichloroethane.

1,1-Dichloroethylene

The primary acute effect of 1,1-dichloroethylene is depression of the central nervous system. Animal studies have shown 1,1-dichloroethylene to produce liver and kidney damage in the rat exposed to 189 mg/cu m (Prendergast et al. 1967, as reported in USEPA 1980) and to produce cardiac sensitization at high concentrations (102,000 mg/cu m) for 10 minutes (Siletchnik and Carlson 1974, as reported in USEPA 1980). Pregnant rats exposed by inhalation to 80-160 ppm on days 6 to 15 of gestation showed decreased weight gain, decreased food consumption, and increased water consumption. Increased liver weight was observed at 160 ppm only. In the offspring, there was a significantly increased incidence of skeletal alterations at 80 and 160 ppm. In the same study, rabbits exposed to 160 ppm on days 6 to 18 days of gestation had an increase in resorptions in the dams; in the offspring, a significant increase in several minor skeletal variations was observed (Murray et al. 1979, as reported in USEPA 1980). Kidney adenocarcinomas were

produced in Swiss mice exposed to 100 mg/cu m, 4 hours per day, 4 to 5 days per week for 52 weeks (Maltoni et al. 1977, as reported in USEPA 1980). In another study, a small increase in hepatic hemangiosarcomas was observed in mice exposed to 220 mg/cu m by inhalation 6 hours/day 5 days per week for 7-12 months (Lee et al. 1972, as reported in USEPA 1980). No effect was observed in rats exposed to 200 mg 1,1-dichloroethylene in drinking water for two years or to 100 and 300 mg/cu m by inhalation, 6 hours per day, 5 days per week for 18 months (Rampy et al. 1977, as reported in USEPA 1980). An NCI bioassay is currently in progress (USEPA 1980). 1,1-Dichlorethylene, which has a structure similar to vinyl chloride monomer (a known liver carcinogen in several species including humans), is also suspected of being a human carcinogen.

Acute toxicity of 1,1-dichloroethylene on freshwater aquatic fish life is reflected by 96-hour LC50 values for the fathead minnow of 169 mg/liter and for the bluegill of 73.9 mg/liter. Furthermore, the toxicity of this compound increases with increasing chlorine content. The 48-hour LC50 value for Daphnia magna was reported as 11.6 mg/liter. The 96-hour LC50 values were reported in the sheepshead minnow to be 249 mg/liter, the tidewater silversides 250 mg/liter, and the mysid shrimp 224 mg/liter. No information is available on the chronic effects of 1,1-dichloroethylene on aquatic organisms (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to 1,1-dichloroethylene through ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at present, a level of 0.33 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for 1,1-dichloroethylene.

1,2-Dichloropropane

Acute toxic effects reported in humans include vertigo, lacrimation, irritation of the mucous membrane, and changes in the blood (St. George 1937, as reported in USEPA 1980). Death has been reported after ingestion of a 50-ml solution containing 1,2-dichloropropane (Larcan et al. 1977, as reported in USEPA 1980).

The oral administration of 5,700 mg/kg of 1,2-dichloropropane in dogs produced staggering and loss of coordination in 15 minutes, complete lack of coordination in 90 minutes, and death in 3 hours. Congestion of the lungs, kidney, and bladder was reported along with hemorrhage of the stomach and respiratory tract. Liver and kidney damage was also produced (Wright and Schaffer 1932, as reported in USEPA 1980). Inhalation exposure to 400 ppm for 7 hours/day for 128-140 days was reported to cause slight fatty degeneration of the liver in mice, but no effects were observed in rats similarly exposed (Heppel et al. 1948, as reported in USEPA 1980). 1,2-Dichloropropane is mutagenic in the Salmonella typhimurium assay (DeLorenzo et al. 1977, as reported in USEPA 1980). Chromosomal aberrations in rat bone marrow were

reported (Dragusanu and Goldstein 1975, as reported in USEPA 1980). No information is available on the carcinogenicity of 1,2-dichloropropane in animals (USEPA 1980).

The acute toxicity of 1,2-dichloropropane in freshwater aquatic organisms was demonstrated in cladoceran, fathead minnows, and bluegill; 96-hour LD50 values were 52.5, 139.3, and 280-320 mg/liter, respectively. Tidewater silverside, a saltwater organism, was affected with an LC50 of 240 mg/liter. Growth inhibition of sheepshead minnow was observed after exposure to 164 mg/liter for 33 days. Chronic toxicity was reported in the fathead minnow in concentrations ranging from 6 to 11 mg/liter. No information is available on the toxic effects on aquatic plant life (USEPA 1980).

EPA has not yet established ambient water quality criteria for 1,2-dichloropropane because of the lack of sufficient data.

1,3-Dichloropropylene

No information is available on the toxic effects in humans. Daily oral doses of up to 2.5 mg/kg of 1,3-dichloropropylene for six months caused an increase in trypsin activity, a decrease in trypsin inhibitor, an increase in blood lipase activity, and a decrease in amylase (Strusevich and Ekshtat 1974, as reported in USEPA 1980). Daily oral doses of 2.2 and 55 mg/kg/day for 30 days resulted in changes in the liver function of rats (Kurysheva and Ekshtat 1975, as reported in USEPA 1980). Oral LD50 values for the rat of 140 mg/kg and the mouse of 300 mg/kg and an LC50 for the rat and mouse of 4,530 mg/cu m were reported (Hine *et al*. 1953, as reported in USEPA 1980). 1,3-Dichloropropylene was mutagenic in the *Salmonella typhimurium* assay (DeLorenzo *et al*. 1977, as reported in USEPA 1980). The carcinogenic potential of 1,3-dichloropropylene has not been established, although an NCI bioassay is in progress.

Acute toxicity of 1,3-dichloropropylene in freshwater aquatic organisms is reflected by 96-hour LC50 values in the cladoceran and the bluegill of 6.15 and 6.06 mg/kg, respectively. Acute toxicity to saltwater organisms occurred in the mysid shrimp and sheepshead minnow with LC50 values of 0.79 and 1.77 mg/liter, respectively. Chronic toxicity was observed in the fathead minnow in concentrations ranging from 180 to 330 µg/liter. Acute toxic effects on fresh- and saltwater algae were reported at 4.95 and 1.0 mg/liter, respectively (USEPA 1980).

EPA has not yet established water quality criteria for 1,3-dichloropropylene because of the lack of sufficient data.

Ethylbenzene

Although ethylbenzene has a wide environmental distribution, little information is available on its biological effects (USEPA 1980). It has been shown to persist in man for days after exposure (USEPA 1980). Men exposed to 4.35 mg/liter in the air experienced eye irritation, which diminished in

intensity on continued exposure. At a concentration of 21.75 mg/liter, however, the irritation to eye and nasal membranes was intolerable (Patty 1963).

In a 6-month study on rats, daily single oral doses of ethylbenzene were found to produce histopathological changes in the kidney and liver (Wolf et al. 1956, as reported in USEPA 1980). No data are available on the teratogenicity, mutagenicity, or carcinogenicity of ethylbenzene (USEPA 1980).

Among freshwater animal species, the 96-hour LC50 ranged from 32 mg/liter in the bluegill to 97.1 mg/liter in the guppy. The 48-hour EC50 value in Daphnia magna is 75 mg/liter. The 96-hour LC50s in saltwater invertebrates are 3.7 mg/liter for the bay shrimp, 87.6 mg/liter for the mysid shrimp and 1,030 mg/liter for the pacific oyster. In saltwater fish, the 96-hour LC50 for striped bass was 0.43 mg/liter, but it was 275 mg/liter for the sheepshead minnow. The variability in fish and invertebrate data may be due to difficulties in testing ethylbenzene in saltwater. No adverse effects were observed in aquatic plants exposed to ethylbenzene (USEPA 1980).

EPA has established an ambient water quality criterion of 1.4 mg/liter for the protection of human health from the toxic properties of ethylbenzene ingested through water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for ethylbenzene.

Methyl Bromide

Methyl bromide is a central nervous system depressant and is regarded as highly toxic to humans. Acute fatal intoxication can result from inhalation of vapors at concentrations as low as 1,164 to 1,552 mg/cu m, and harmful effects can occur at 388 mg/cu m (USEPA 1980). Minor poisoning episodes may be limited to mild neurological and GI disturbances, with recovery in a few days. More severe cases may involve visual and speech disturbances, incoordination, tremors developing to convulsions, and psychic disturbances. Neurological disorders may be persistent. Death may result from pulmonary edema or circulatory failure, and pathological changes often include hyperemia, edema, lung and brain inflammation, and degenerative changes in the kidneys, liver, and stomach (Doull et al. 1980, and USEPA 1980). Skin contact with methyl bromide may produce prickling, cold sensation, erythema, vesication, blisters, damage to peripheral nerve tissue, and permanent brain damage (USEPA 1980).

Methyl bromide is also neurotoxic to animals. Exposure to 846 to 997 mg/cu m for 22 to 26 hours was lethal to rats (Irish et al. 1940, as reported in USEPA 1980). A 3-hour exposure to 846 mg/cu m and a 13.5-hour exposure to 1,164 mg/cu m were lethal to rabbits and guinea pigs, respectively (von Oettingen 1964, as reported in USEPA 1980). In rabbits, 128 mg/cu m for 8 hours/day, 5 days/week resulted in lung irritation and paralysis (Irish et al. 1941, as reported in USEPA 1980). Dogs receiving methyl bromide by ingestion (fumigated diet yielding residual bromide at a dose level of 150 mg/kg/day) were adversely affected (Rosenblum et al. 1960, as reported in

USEPA 1980). Subcutaneous administration of methyl bromide (in oil) to rabbits at 20-120 mg/kg caused paralysis, cessation of drinking, and reduced urine excretion (Kakizaki 1967, as reported in USEPA 1980). Cattle fed methyl bromide fumigated hay (resulting in bromide ion concentrations of 6,800 to 8,400 mg/liter) developed signs of CNS toxicity (motor incoordination) at 10 to 12 days of exposure (Knight and Reina-Guerra 1977, as reported in USEPA 1980). Bromomethane was reported to be mutagenic in an *in vitro* test with *Salmonella typhimurium* (Simmon *et al*. 1977, as reported in USEPA 1980). No chronic studies of methyl bromide toxicity are available.

For methyl bromide, the 96-hour LC50 value for the bluegill, a freshwater species, is 11,000 µg/liter and for the tidewater silverside, a saltwater species, is 12,000 µg/liter (USEPA 1980). No chronic toxicity data for aquatic organisms are available.

EPA has established ambient water quality criterion of zero for maximum protection of human health from the potential carcinogenic effects due to exposure to methyl bromide through the ingestion of contaminated water and aquatic organisms. However, since the zero level may not be attainable at the present time, a level of 0.19 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for methyl bromide.

Methyl Chloride

Methyl chloride is not generally regarded as highly toxic in humans, although numerous instances of poisonings have been reported. Serious or prolonged exposures may occur because of methyl chloride's odorless and colorless properties, low irritancy, and characteristic latency of effect (USEPA 1980). Methyl chloride acts principally as a central nervous system depressant. In persons exposed to levels ranging from 52 to more than 20,000 mg/cu m, the following toxic symptoms have been reported: blurred vision, headache, nausea, loss of coordination, and personality changes, lasting from hours to days (USEPA 1980). Severe poisonings are characterized by a latent period followed by serious neurological disorders which may be persistent. Renal and hepatic injury are common. Coma and death may result from cerebral and pulmonary edema and circulatory failure (USEPA 1980). No other chronic effects have been reported, although no epidemiological studies of populations exposed to methyl chloride have been reported.

In acute inhalation studies in animals, methyl chloride has produced severe neurological disturbances. The LC50 value for the mouse was 6,500 mg/cu m following a six-hour inhalation exposure (Davis *et al*. 1977, as reported in USEPA 1980). Permanent muscular dysfunction was reported in mice surviving several weeks of daily six-hour exposures at 1,032 mg/cu m, and paralysis followed exposure to 531 mg/cu m for 20 hours (von Oettinger *et al*. 1964, as reported in USEPA 1980). In dogs and monkeys, signs of poisoning were observed after one six-hour exposure to 1,032 mg/cu m. Daily exposure of dogs to this concentration for 2 to 4 weeks led to some deaths and permanent neuromuscular damage in survivors (von Oettinger 1964, as reported

in USEPA 1980). Under prolonged exposures to less severe levels, methyl chloride increased mucus flow in cats (Weissbecker et al. 1971, as reported in USEPA 1980).

Little aquatic toxicity data are available for methyl chloride. Dawson et al. (1977, as reported in USEPA 1980) reported 96-hour LC50 values for the bluegill (a freshwater species) and the tidewater silverside (a saltwater species) of 550,000 and 270,000 µg/liter, respectively. Data on the general class halomethanes indicate that acute toxicity may occur at levels as low as 11,000 µg/liter (USEPA 1980). No data on toxic effects to aquatic plants are available.

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to methyl chloride through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, a level of 0.19 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for methyl chloride.

Methylene Chloride

The acute toxic effects of methylene chloride on humans include decreased psychomotor performance (Winneke 1974, as reported in USEPA 1980), central nervous system dysfunctions and irritation of the mucous membranes (eyes, respiratory tract, and skin) (NAS 1978, as reported in USEPA 1980). Mild poisoning produces somnolence, lassitude, anorexia, and mild lightheadedness. Fatal poisonings have resulted from cardiac injury and heart failure (NAS 1978, citing Hughes 1954, Stewart and Hake 1976, Collier 1936, Moskowitz and Shapiro 1952, as reported in USEPA 1980). Upon metabolism, methylene chloride will form carbon dioxide, which will increase carboxyhemoglobin levels in the blood and interfere with oxygen transfer and transport (USEPA 1980).

Central nervous system functional disturbances were produced in animal studies in which rats were exposed for 3 hours to 1,740 mg/cu m (Fodor and Roscovanu 1976, as reported in USEPA 1980). Liver changes were reported in mice continuously exposed for up to 2 weeks to 87-347 mg/cu m (NAS 1978 citing Haun et al. 1972, as reported in USEPA 1980). Conjunctivitis, blepharitis, corneal thickening, keratitis, and iritis were observed in rabbits (Ballantyne et al. 1976, as reported in USEPA 1980). Fetotoxicity and embryotoxicity were reported in mice and rats exposed to 4,340 mg/cu m for 7 hours on day 9 of gestation (Schwetz et al. 1975, as reported in USEPA 1980).

Methylene chloride was reported to be mutagenic in the *Salmonella typhimurium* assay. No information on the carcinogenicity of methylene chloride in animals or humans is available (USEPA 1980).

The acute toxic effects of methylene chloride on freshwater aquatic organisms have been studied. 96-Hour LC50 values were determined for *Daphnia magna* (224 mg/liter), fathead minnow (193 mg/liter), and bluegill (224

mg/liter). Among saltwater species, mysid shrimp have reported 96-hour LC50 values of 256 mg/liter; sheepshead minnows, 331 mg/liter; and tidewater silverside, 270 mg/liter. No information on the chronic toxicity of methylene chloride on aquatic organisms is available. Toxicity to both freshwater and saltwater algae was reported to occur in concentrations greater than 662 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to halomethanes including methylene chloride through the ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at the present time, a level of 0.19 µg/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended for all the halomethanes including methylene chloride.

EPA has not yet established an aquatic life water quality criterion for methylene chloride.

1,1,2,2-Tetrachloroethane

A number of cases of human poisonings from occupational exposure to 1,1,2,2-tetrachloroethane have been reported. Frequently noted symptoms include vomiting, nausea, gastric pain, headache and dizziness, and in some cases death resulting from central nervous system (CNS) depression. Chronic exposure can result in hepatotoxic and CNS effects (ACGIH 1981, USEPA 1980). In two occupational studies, hepatic injury and leukopenia were reported in workers exposed to concentrations of 1,1,2,2-tetrachloroethane between 1.5 and 247 ppm for three years, and nervous complaints and gastric symptoms were present in workers exposed to concentrations between 9 and 98 ppm (Jeney et al. 1957, Lobo-Mendoca 1963, as reported in ACGIH 1981). Cases of human deaths have also resulted from accidental or intentional 1,1,2,2-tetrachloroethane ingestion (USEPA 1980).

Acute inhalation exposure of rats and mice to 1,1,2,2-tetrachloroethane produced anesthesia, fatty degeneration of the liver, tissue congestion, and death; exposures in these studies ranged from 5,900 ppm (three hours) to 11,400 ppm (six hours for two days). A three-hour exposure of mice to 600 ppm resulted in increased hepatic triglycerides and total lipids and decreased hepatic energy stores (Tomokuni 1969, as reported in USEPA 1980). Intravenous or intraperitoneal injection of 0.7 ml (total dose administered in five doses over 14 days) in guinea pigs caused weight loss, convulsions, death, and fatty degeneration of the liver and kidney (Muller 1932, as reported in USEPA 1980). Injection of 200 mg/kg was lethal to mice in seven days (Natl. Res. Counc. 1952, as reported in USEPA 1980).

Chronic inhalation exposure of rabbits to 1,1,2,2-tetrachloroethane at 14.6 ppm, 4 hours/day for 11 months induced liver and kidney degeneration (Navrotskiy et al. 1971, as reported in USEPA 1980). White blood cell count, pituitary adrenocorticotropic hormone, and fat content of the liver were affected in rats exposed by inhalation to 1.94 ppm, 4 hours/day for up to 265 days (Deguchi 1972, as reported in USEPA 1980). Monkeys exposed to 1,000 or 4,000 ppm, 2 hours/day for 190 days developed marked vacuolation of the

liver (Horiguchi et al. 1962, as reported in USEPA 1980). A National Cancer Institute bioassay was conducted for 1,1,2,2-tetrachloroethane in which male and female mice received time-weighted average doses of 142 and 282 mg/kg/day by stomach tube for 78 weeks (NCI 1978, as reported in USEPA 1980). The incidence of hepatocellular carcinomas in both male and female mice was positively correlated ($p < 0.001$) with dosage level. In male and female rats given time-weighted average doses of 62 and 108 mg/kg/day (males) and 43 and 76 mg/kg/day (females) for 78 weeks, the incidence of neoplasms was not statistically significant. 1,1,2,2-Tetrachloroethane was shown to be moderately mutagenic in in vitro assays with Salmonella typhimurium and E. coli (USEPA 1980).

In freshwater species, LD50 values of 9,320-23,900 µg/liter have been reported for the cladoceran, 20,300 µg/liter for the fathead minnow, and 19,600-21,300 µg/liter for the bluegill. In an embryo-larval study of fathead minnow, chronic toxicity was observed at 2,400 µg/liter (USEPA 1980). Acute toxicity in saltwater species of 1,1,2,2-tetrachloroethane was reported for mysid shrimp and sheepshead minnow with LD50 values of 9,020 and 12,300 µg/liter, respectively (USEPA 1980). No chronic studies in saltwater species were available. In 96-hour EC50 tests with freshwater and saltwater algae using chlorophyll a and cell number as measured responses, toxic effects were observed at concentrations of 136,000-146,000 and 6,230-6,440, respectively (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to 1,1,2,2-tetrachloroethane through ingestion of contaminated water and aquatic organisms. However, since the zero level may not be attainable at the present time, a level of 0.17 ng/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for 1,1,2,2-tetrachloroethane.

Tetrachloroethylene

The acute effects of tetrachloroethylene on humans are dominated by central nervous system depression. Lassitude, mental fogginess, and exhilaration were observed in human volunteers exposed to 6,258 mg/cu m for 95 and 130 minutes (Carpenter 1937, as reported in USEPA 1980). When this concentration was raised to 10,000 mg/cu m signs of inebriation were observed and, at 13,400 mg/cu m, all volunteers were forced to leave the chamber within 7.5 minutes. Minimal effects on the central nervous system were observed on volunteers exposed to 1,300 mg/cu m (Rowe et al. 1952 and Stewart et al. 1961, as reported in USEPA 1980). Irritation of the mucous membranes have also been reported (NAS 1980). Mild to severe hepatotoxicity was observed in several instances following inhalation of tetrachloroethylene (dose and duration unspecified; Hake and Steward 1977; Salund 1967; Stewart et al. 1961; Stewart 1969; Meckler and Phelps 1966, as reported in NAS 1980). No consistent neurological changes were reported in a study of 12 volunteers exposed to 168 and 670 mg/cu m tetrachloroethylene for 5.5 hours per day for up to 53 days (Stewart et al. 1977, as reported in USEPA 1980).

Occupational exposure to an average concentration of 400 mg/cu m (in one worker, for up to 15 years) resulted in subjective complaints, such as headaches, fatigue, somnolence, dizziness, and a sensation of intoxication (Medek and Kovarik 1977, as reported in USEPA 1980). No other information on the subacute of chronic toxicity of tetrachloroethylene on humans is available.

Few studies have been conducted to evaluate the acute toxic effects of tetrachloroethylene on animals. A single 240-minute exposure to 1,340 mg/cu m (200 ppm) resulted in moderate fatty degeneration of the liver of mice (Kylin et al. 1963, as reported in NAS 1980). Levels of serum glutamic oxaloacetic transaminase were elevated following a single oral dose of 0.75 ml/kg in rats (Moslen et al. 1977, as reported in NAS 1980). Similarly, intraperitoneal administration of 0.3 to 0.5 ml/kg tetrachloroethylene in rats resulted in increased serum enzyme levels (Klassen and Plaa 1967, as reported in NAS 1980). The oral LD50 value for the dog and cat is 4,000 mg/kg, the rabbit, 5,000 mg/kg, and for the mouse, the values range from 195 to 8,100 mg/kg (USDHHS 1980).

Rats, guinea pigs, rabbits, and monkeys exposed repeatedly for 7 hours per day showed no changes in behavior at concentrations up to 2,720 mg/cu m (duration unspecified; Rowe et al. 1952, as reported in USEPA 1980). After a 2-week exposure to 10,999 mg/cu m for 7 hours per day, rats showed signs of marked salivation, restlessness, irritability and loss of equilibrium and coordination (Rowe et al. 1952, as reported in USEPA 1980). Changes in EEG patterns were reported in rats exposed to 100 mg/cu m, 4 hours per day, for 15 to 30 days (Dmitrieva 1966; Dmitrieva and Kuleshov 1972, as reported in USEPA 1980). Increased liver weight and mild to marked central fatty degeneration of the liver were reported in guinea pigs exposed to 670-16,750 mg/cu m, 7 hours per day for up to 158 repeated exposures (Rowe et al. 1952, as reported in USEPA 1980). Congestion and granular swelling in the kidney was observed in rats exposed to 1,540 mg/cu m for 8 hours per day, 5 days per week over a period of 7 months (Carpenter 1937, as reported in USEPA 1980). A very high incidence of nephrotoxicity and a little evidence of hepatotoxicity was observed in a chronic oral study of mice and rats gavaged with doses of tetrachloroethylene ranging from 386 to 1,072 mg/kg/day for 78 weeks (National Cancer Institute 1977, as reported in NAS 1980).

Tetrachloroethylene did not induce mutations in Escherchia coli in the presence of a microsomal activating system (Greim et al. 1975, as reported in NAS 1980). Female rats and mice exposed to 2,000 mg/cu m for 7 hours daily on days 6 through 15 of gestation did not produce teratogenic effects. However, there was a decrease in the fetal body weight of mice, a small but significant increase in fetal resorptions in the rat, subcutaneous edema in mice pups and delayed ossification of skull bones and sternabrae in the mice (Schwetz et al. 1975, as reported in USEPA 1980). Heptocellular carcinomas were reported in mice, but not rats gavaged with daily oral doses of tetrachloroethylene ranging from 386 to 1,072 mg/kg for up to 78 weeks (National Cancer Institute 1977, as reported in NAS 1980).

The acute toxic effects of tetrachloroethylene on aquatic fish life is reflected by 96-hour LC50 values for the cladoceran, 17.7 mg/liter; the midge, 30.8 mg/liter; the rainbow trout, 4.8-5.8 mg/liter; the fathead minnow, 13.5-21.4 mg/liter; the bluegill, 12.9 mg/liter; and the mysid shrimp, 10.2

mg/liter. Chronic toxicity was observed in the fathead minnow in concentrations ranging from 0.5 to 1.4 mg/liter and in the mysid shrimp in concentrations ranging from 0.3 to 0.67 mg/liter. No adverse effects were observed on chlorophyll a or cell number of freshwater algae exposed to concentrations as high as 816 mg/liter. Saltwater algae, however, are more sensitive and have EC50 values ranging from 10.5 to 509 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to tetrachloroethylene through ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at present, a level of 0.80 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for tetrachloroethylene.

Toluene

Acute toxic effects in humans include adverse mental changes, such as altered psychomotor performance, irritability, disorientation, and unconsciousness (NAS 1980). Additionally, toluene abuse has been associated with cardiac arrhythmias and with liver and kidney dysfunction (Hayden et al. 1977, Weisenberger 1977, as reported in NAS 1980). In occupational exposures to solvent mixtures, workers have reported myelotoxicity (NAS 1980), minor blood cell change, and hepatomegaly (Greenburg et al. 1942, as reported in NAS 1980), and immunoincompetence (Lange et al. 1973, as reported in NAS 1980). These affects cannot be attributed to toluene alone.

The minimal lethal concentration of toluene was reported to be 20 mg/liter in mice for a single 8-hour inhalation exposure. The acute oral toxicity of toluene is greater in young rats than in adult animals (Kimura et al. 1971, as reported in NAS 1980). Liver microsomal activity was decreased by acute oral administration of high doses of toluene to rats (Mungikar and Pawar 1976, as reported in USEPA 1980). Rats exposed to 1,000 ppm for 8 hours/day for 1 week had slightly elevated serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic transaminase (SGPT) activities. Chronic studies in two dogs exposed 8 hours/day for 6 months showed nervous system intoxication, incoordination, paralysis of the hind legs, and congestive changes in the lungs, heart, liver, kidney, and spleen (Tahti et al. 1977, as reported in NAS 1980). Chromosome damage of bone marrow cells was reported in rats injected with 1 g/kg of toluene (Lyapkalo 1973, as reported in NAS 1980). Embryotoxicity was observed in rats exposed to toluene vapors at 600 mg/cu m (Hudak et al. 1977, as reported in NAS 1980). The carcinogenic potential of toluene has not been established (USEPA 1980).

The acute toxicity of toluene to freshwater organisms was demonstrated in four species of fish with 96-hour LC50 values ranging from 17.5 top 59.3 mg/liter. In saltwater shrimp and oysters, 96-hour LC50s ranged from 9.5 to 1,050 mg/liter. Striped bass and coho solmon have LC50 values of 6.3 and 10-50 mg/liter, respectively. A range of LC50 values between 17.2 and 38.1

mg/liter were observed in a number of 24-hour tests on the grass shrimp. A chronic toxicity effect was observed on the hatching and survival of sheepshead larvae and embryos. Fresh and saltwater algae were adversely affected at 245 mg/liter and 100 mg/liter, respectively. Photosynthesis and respiration were affected in kelp at 10 mg/liter and in algae at 34-85 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 14.3 mg/liter for the protection of human health from the toxic properties of toluene ingested through water and contaminated organisms.

EPA has not yet established an aquatic life water quality criterion for toluene.

Trans-1,2-Dichloroethylene

Humans have developed nausea, vomiting, weakness, tremor, and cramps following exposure to high concentrations of trans-1,2-dichloroethylene vapor (Sax 1975). The effects are rapidly reversible following removal from exposure. No other information is available on the toxic effects of this compound on humans.

There is limited information available from experimental studies. Repeated inhalation exposures of 800 mg/cu m, 8 hours per day, 5 days per week for 16 weeks of trans-1,2-dichloroethylene produced fatty degeneration of the liver in rats (Freundt *et al*. 1977, as reported in USEPA 1980). In a series of studies using 1,2-dichloroethylene, no measureable effects on growth, mortality, organ and body weights, hematology, clinical chemistry and gross and microscopic pathology were reported in rats, rabbits, guinea pigs, and dogs exposed to levels as high as 4,000 mg/cu m for six months (ACGIH 1977, as reported in USEPA 1980). Trans-1,2-dichloroethylene is not metagenic when assayed with *E. coli* K12 (Greim *et al*. 1975, as reported in USEPA 1980). No information is available on the potential teratogenicity or carcinogenicity of this compound.

The assessment of the toxicity of trans-1,2-dichloroethylene on aquatic life is limited to one 96-hour LC50 value of 135 mg/liter for the bluegill. No other information is available on the toxic effects of this compound on aquatic life (USEPA 1980).

EPA has not yet established ambient water quality criteria for trans-1,2-dichloroethylene because of the lack of sufficient data.

1,1,1-Trichloroethane

1,1,1-Trichloroethane primarily causes central nervous system disorders in humans (USEPA 1980). Symptoms include depression; changes in reaction time, perceptual speed, manual dexterity, and equilibrium; incoordination; and burning and tingling sensation in the hands and feet. Other toxic effects that have been observed in humans include hepatic cellular damage, liver function abnormalities, nausea, vomiting, diarrhea, hypotension, bradycardia,

cardiac arrhythmias, eye irritation, fatigue, and death (NIOSH 1978, as reported in USEPA 1980).

Experimental studies have shown that 1,1,1-trichloroethane induces toxic effects in a wide range of animal species. Cardiac arrhythmia, myocardial depression, tachycardia, tremors and respiratory failure have been reported in the monkey. Cardiac failure, pulmonary congestion, respiratory failure and damage to the central and peripheral nervous systems have been observed in the rat. In the mouse, liver dysfunction, pulmonary congestion, and cardia arrhythmia have been reported. In the guinea pig, lung irritation and liver dysfunction have been observed. Respiratory failure has been induced in the dog and damage to the central and peripheral nervous systems has been reported in the cat (Truhart et al. 1973, Horiguchi and Horiguchi 1971, Tsapko and Rappoport 1972, Beleg et al. 1974, Herd et al. 1974, Torkelson et al. 1958, MacEwen and Vernot 1974, as reported in USEPA 1980). In most studies, high concentrations were used. The lowest concentration producing toxic effects was 73 ppm administered four hours per day for 50-120 days (Tsapko and Rappoport 1972, as reported in USEPA 1980).

The acute toxic effects of 1,1,1-trichloroethane on freshwater aquatic organisms is reflected by 96-hour LC50 values of 52-105 mg/liter for the fathead minnow and 69.7 mg/liter for the bluegill. Toxicity to saltwater organisms has been observed in the mysid shrimp, with a reported LC50 value of 31.2 mg/liter, and in the sheepshead minnow, with an LC50 value of 70.9 mg/liter. Toxicity to plants occurs in freshwater algae at levels greater than 530 mg/liter and in saltwater algae at levels greater than 669 mg/liter. No information is available on the chronic toxicity of 1,1,1-trichloroethane on aquatic organisms (USEPA 1980).

EPA has established an ambient water quality criterion of 18.4 mg/liter for the protection of human health from the toxic properties of 1,1,1-trichloroethane ingested through water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for 1,1,1-trichloroethane.

1,1,2-Trichloroethane

No information is available on the toxic effects of 1,1,2-trichloroethane in humans. Kidney necrosis was reported in the mouse and dog administered single dose intraperitoneal injections of 0.35 ml/kg and 0.45 ml/kg, respectively (Klassen and Plaa 1967, as reported in USEPA 1980). The effective dose that produces kidney necrosis in 50% of the animals is 0.17 ml/kg in mice and 0.4 ml/kg in the dog, both administered by intraperitoneal injection. Centrilobular necrosis of the liver was observed in dogs treated with a single dose of 0.35 ml/kg by intraperitoneal injection (Klassen and Plaa 1967, as reported in USEPA 1980). 1,1,2-Trichloroethane has been found to cause liver and adrenal cancer in mice administered 195 and 390 mg/kg/day by gavage for 78 weeks (NCI 1978, as reported in USEPA 1980).

The acute toxic effects of 1,1,2-trichloroethane on freshwater organisms is reflected by 96-hour LC50 values for Daphnia magna of 18-43 mg/liter, for the fathead minnow of 81.7 mg/liter, and for the bluegill of 40.2 mg/liter. Chronic toxicity has been observed in the fathead minnow in concentrations ranging from 6 to 14.8 mg/liter. No information is available on the toxicity to saltwater organisms or the effects of 1,1,2-trichloroethane to aquatic plants (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to 1,1,2-trichloroethane through ingestion of contaminated water and contaminated fish. However, since the zero level may not be attainable at present, a level of 0.6 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for 1,1,2-trichloroethane.

Trichloroethylene

The acute toxic effects of trichloroethylene in humans include nervous system depression, incoordination and unconsciousness (NAS 1977). Clinical signs and symptoms are principally those of gastrointestinal upset, narcosis, and occasional cardiac abnormalities (NAS 1980). Controlled human clinical studies have shown that inhalation of 100 to 200 ppm (duration unspecified) caused complaints of the eye, throat irritation, and fatigue in several exposed volunteers (Gamberale et al. 1976, Nomiyama and Nomiyama 1977, Vernon and Ferguson 1969, as reported in NAS 1980). In a separate study, two patients who drank 350 and 500 ml trichloroethylene were rendered unconscious for four and eight days, respectively. Hypotension and cardiac arrhythmias were delayed in onset, but were quite serious in nature (Dhuner et al. 1957, as reported in NAS 1980).

In an epidemiology study, evidence of increased nervous system disorders was found in workers exposed for 5 to 15 years to concentrations less than the threshold limit value (50 ppm). (Grandjean et al. 1955, as reported in USEPA 1980).

Insomnia, tremors, severe neurasthenic syndromes coupled with anxiety states and progressive bradycardia have been reported in workers exposed to concentrations of trichloroethylene ranging from 30 to 632 ppm (duration unspecified) with disturbances of the nervous system continuing for up to 1 year following exposure (Bardodej and Vyskoch 1956, as reported in USEPA 1980). Headaches have been reported in workers exposed to concentrations as low as 27 ppm (Nomiyama and Nomiyama 1977, as reported in USEPA 1980). Each of these accounts of the chronic effects of trichloroethylene suffer from a lack of accurate exposure levels and the inability to distinguish trichloroethylene induced effects from those caused by other factors.

Behavioral studies have generally confirmed that the CNS depressant activity of trichloroethylene observed in humans also occurs in rats following roughly equivalent exposures (Khorvat and Formanek 1959 and Goldberg et al.

1964, as reported in USEPA 1980). The acute oral LD50 of trichloroethylene in rats if 4,920 mg/kg (NIOSH 1980); and the acute intraperitoneal LD50 for the mouse is 2.2 ml/kg (Klaasen and Plaa 1967, as reported in NAS 1980). A single intraperitoneal injection of 0.6 ml/kg caused a loss of muscle tone, depression of reflexes, and slowing of heart rate in guinea pigs (Mikiskova and Mikiska 1966, as reported in NAS 1980).

In subacute toxicity studies, rabbits injected intramuscularly with 4.38 grams per animal three times a week for four weeks developed a loss of Purkinje cells with associated basket cells in the cerebellum and other less specific damage to the telencephalic cortex, basal gaglia and brain stem nuclei (Bartonicek and Brun 1970, as reported in USEPA 1980). Similar effects have been reported in rabbits exposed by inhalation to 1,889 ppm for 20 to 30 days (Bernardi et al. 1956, as reported in USEPA 1980) and in dogs exposed to 297-502 ppm (duration unspecified; Baker 1958, as reported in USEPA 1980).

The chronic toxicity from long term exposure to trichloroethylene is not considered to differ significantly from the observed acute toxic effects (NAS 1980). Maximum no-effect levels have been reported in monkeys, 400 ppm, rabbits and rats, 200 ppm and guinea pigs, 100 ppm, exposed to trichloroethylene vapor 7 hours per day, 5 days per week for six months (Adams et al. 1951, as reported in NAS 1980).

In another study, rats, guinea pigs, monkeys, rabbits, and dogs exposed by inhalation to either 730 ppm, 8 hours per day, 5 days per week, for 6 weeks or 35 ppm continuously for 90 days showed no evidence of adverse effects (Prendergast 1967, as reported in NAS 1980).

Trichloroethylene has been found to be mutagenic in a number of microsomally activated in vitro screening systems, including Salmonella typhimurium (NAS 1980), Excherichia coli (Greim et al. 1975, as reported in USEPA 1980) and Saccharomyces cerevisiae (Shahin and von Barstel 1977, as reported in USEPA 1980). No evidence of teratogenicity was observed in mice and rats exposed to 297 ppm on days 6 through 15 of gestation for 7 hours per day (Schwetz et al. 1975, as reported in NAS 1977).

Trichloroethylene has been found to be carcinogenic in the liver of mice orally administered time weighted average doses of 1,169 and 2,339 mg/kg for males and 869 and 1,739 mg/kg for females five days per week for 78 weeks. Some evidence of metastasis of hepatocellular carcinoma to the lungs was also observed in the mice. No increase in the incidence of tumors was observed in parallel experiments with rats (NCI 1976, as reported in USEPA 1980). Two other long-term bioassays, one in rats and the other using a different strain of mice, yielded negative results (Maltoni 1979 and Van Duuren et al. 1979, as reported in USEPA 1980). This combined with questions about the design of the NCI study have raised questions about the true carcinogenic potential of trichloroethylene (NAS 1980).

The acute toxicity of trichloroethylene on aquatic fishlife is reflected by 96-hour LC50 values for cladoceran, 39-100 mg/liter, fathead minnow, 40.7-66.8 mg/liter, and the bluegill 44.7 mg/liter. Intoxication characterized by erratic swimming, uncontrolled movement and loss of equilibrium was observed in grass shrimp and sheepshead minnow after several

minutes of exposure to 2.0 mg/liter and 20 mg/liter, respectively. A loss of equilibrium was reported in the fathead minnow exposed to 21.9 mg/liter for 96 hours. A 50 percent decrease in 14C uptake during photosynthesis was observed in saltwater algae exposed to 8 mg/liter trichloroethylene. No information is available one the chronic toxicity of trichloroethylene to aquatic fishlife.

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to trichloroethylene through the ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at the present time, a criterion of 2.7 µg/liter at an incremental lifetime cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for trichloroethylene.

Vinyl Chloride

This compound in highly flammable and volatile with high explosive characteristics. The toxicity of this compound has been clearly described in animals and in humans (NAS 1977 and USEPA 1980).

In humans, vinyl chloride has been shown to produce central nervous system dysfunction, sympathetic-sensory polyneuritic and organic disorders of the brain (Smirnova and Granik 1970, as reported in NAS 1977) In occupational studies, vinyl chloride has been associated with scleroma-like skin alterations, Raynaud's syndrome, acroosteolysis, thrombocytopenia, portal fibrosis, and hepatic and pulmonary dysfunction (Juehe and Lange 1972, Juene et al. 1974, Berk et al. 1975, Martsteller et al. 1975, as reported in NAS 1977). In addition, hepatic angiosarcoma, one of the rarest human malignant neoplasms, has been observed in vinyl chloride workers (Anon 1974, Makk et al. 1976, as reported in NAS 1977 and Creech and Johnson 1974, as reported in USEPA 1980). Lesions of the skin, bone, liver, spleen, and lungs have also been reported after chronic exposure to this compound (Popper and Thomas 1975, Gedigk et al. 1975, Thomas and Pepper 1975, as reported in NAS 1977).

In acute and subchronic inhalation studies, vinyl chloride has been shown to produce lung congestion, hemorrhaging, blood-clotting difficulties, and congestion of liver and kidneys (species unspecified; Mastromatteo et al. 1960, as reported in NAS 1977). Numerous studies have reported its carcinogenic effects in rats, mice, hamsters, and rabbits by both inhalation and oral administration (USEPA 1980).

No acute or chronic data are available on the effects of vinyl chloride on freshwater of saltwater organisms (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to vinyl chloride through the ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be

attainable at the present time, a level of 2.0 μg/liter, corresponding to a lifetime incremental increase of cancer risk of 0.000001, was recommended.[1]

EPA has not yet established an aquatic life water quality criterion for vinyl chloride.

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[1]In the Carcinogen Assessment Group's summary and conclusions regarding carcinogenicity of vinyl chloride (July 14, 1978) an individual lifetime risk of 0.00001 was associated with an intake of 1.054 mg/kg/day (0.53 mg/liter). A risk of 0.000001 would be associated with an intake of 0.1054 mg/kg/day (0.053 mg/liter).

2. Acid Extractable Organic Compounds

4-Chloro-m-cresol

The only reported toxic effect of 4-chloro-m-cresol in humans is vesicular dermatitis. In sensitive individuals, a 1.5 percent aqueous solution caused a pruritic vesicular dermatitis within four hours of exposure which regressed within a week (Guy and Jacobs 1941, as reported in USEPA 1980).

In animals, acute exposure to 4-chloro-m-cresol produces severe muscle tremors, damage to renal tubules, and death in a few hours (Wein 1939, as reported in USEPA 1980). For the mouse, Wein (1939, as reported in USEPA 1980) reported LD50s by subcutaneous injection and by intravenous injection of 360 and 70 mg/kg. For the rat, the subcutaneous LD50 was given as 400 mg/kg. An oral LD50 of 1,330 mg/kg has also been reported for the mouse (Schrotter et al. 1977, as reported in USEPA 1980). In subchronic studies in which rats were injected subcutaneously with 80 mg/kg/day for 14 days and rabbits were injected with approximately 6.5 mg/kg/day for four weeks, the only observed effect was mild inflammation at the site of injection in the rats (Wein 1939, as reported in USEPA 1980). No chronic toxicity data for 4-chloro-m-cresol are available.

Limited aquatic toxicity data is available for 4-chloro-m-cresol. The 96-hour LD50 for the fathead minnow is 30 µg/liter (USEPA 1980). No data are available for saltwater species.

EPA has not yet established ambient water quality criteria for protection of human health and aquatic life from exposure to 4-chloro-m-cresol because of the lack of sufficient information. However, using available organoleptic data for control of undesirable taste and odor quality of ambient water, the recommended criterion is 3,000 µg/liter.

2-Chlorophenol

Very little information is available on the toxic effects of 2-chlorophenol on humans. Acute toxicity has been characterized as being "likely" to be corrosive and irritating to the eyes and skin (Doedens 1963, as reported in USEPA 1980). 2-Chlorophenol is considered to be a weak uncoupler of oxidative phosphorylation (Mitsuda et al. 1963, as reported in USEPA 1980) and a convulsant poison (Farquharson et al. 1958 and Angel and Rogers 1972, as reported in USEPA 1980).

Relatively few acute toxicological studies are available on laboratory animals. Acute LD50 values have been reported as follows: the rat, 670 mg/kg-oral and 900 mg/kg-subcutaneous (Diechmann 1943, as reported in USEPA 1980); the mouse, 670 mg/kgoral (Bubnov et al. 1969, as reported in USEPA 1980); and the blue fox, 440 mg/kg-oral (Bubnov et al. 1969, as reported in USEPA 1980). Subcutaneous LD50 values have been reported for the rabbit, 950 mg/kg (Christensen and Luginbyhl 1975, as reported in USEPA 1980). Minimum lethal dose values have also been reported for the rabbit, 120 mg/kg, (intravenous administration; Kuroda 1926, as reported in USEPA 1980); for the guinea pig 800 mg/kg, (subcutaneous administration; Christensen and Luginbyhl

1975, as reported in USEPA); and for the albino rat, 230 mg/kg (intraperitoneal administration; Farquharson et al. 1958, as reported in USEPA 1980). Symptoms of acute toxicity in rats, regardless of the route of administration, include restlessness and increased rate of respiration, followed by the development of motor weakness, tremors and convulsions. Eventually, dyspnea and the appearance of coma result and continue until death (Farquharson et al. 1958, as reported in USEPA 1980). Marked kidney injury including red blood cells casts in the tubules, fatty infiltration of the liver, and hemorrhages in the intestine were observed in rats following fatal poisoning (dose unspecified; Patty 1963). Similar pathological effects were reported for the blue fox and the mouse (Bubnor et al. 1969 as reported in USEPA 1980). A rapid onset of convulsions was observed in mice administered 2-chlorophenol intraperitoneally (dose unspecified; Angel and Rogers 1972, as reported in USEPA 1980).

No information is available on the subacute or chronic toxicity, nor on the mutagenic or teratogenic potential of 2-chlorophenol. Topical application of 0.3% dimethylbenzanthracene in benzene as an initiator followed by 20% 2-chlorophenol twice weekly for 20 weeks, promoted papillomas in mice (Boutwell and Bosch 1959, as reported in USEPA 1980).

The acute toxicity of 2-chlorophenol on aquatic fish life is reflected by 96-hour LC50 values for the cladoceran, 2.6-7.4 mg/liter, the goldfish, 12.4 mg/liter, the fathead minnow, 11.6-14.5 mg/liter, the guppy, 20.2 mg/liter, and the bluegill, 6.6-10 mg/liter. No effect was observed in fathead minnow exposed to 3.9 mg/liter in a chronic test using the embryo-larval method. A reduction in chlorophyll was observed in an alga exposed to 500 mg/liter for 72 hours indicating that plants may be less sensitive to 2-chlorophenol than fish life. 2-Chlorophenol was found to impair the flavor of fish at concentrations as low as 2 mg/liter (USEPA 1980).

EPA has not yet established ambient water quality criteria for 2-chlorophenol because of the lack of sufficient data.

2,4-Dichlorophenol

No information is available on the toxic effects of 2,4-dichlorophenol on humans. In vitro studies, however, indicate that 2,4-dichlorophenol is an uncoupler of oxidative phosphorylation (Mitsuda et al. 1963, as reported in USEPA 1980).

Relatively few studies are available on the acute or subacute toxicity of 2,4-dichlorophenal in animals. Oral LD50 values have been reported for the rat, 580 and 4,000 mg/kg (Derchman 1943 and Kobayashi et al. 1972, as reported in USEPA 1980) and for the mouse, 1,600 mg/kg. (Kobayashi et al. 1972, as reported in USEPA 1980). Acute subcutaneous and intraperitoneal LD50 values for the rat have been reported to be 1,730 mg/kg and 430 mg/kg, respectively (Deichman 1943 and Farquharson et al. 1958 as reported in USEPA).

In a subacute study, all mice survived when 2,4-dichlorophenol was administered orally for 10 days at a dose of 667 mg/kg body weight (Kobayashi

et al. 1972, as reported in USEPA 1980). In a six-month feeding study by the same authors, no observed adverse changes in growth rate, hematology, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), and behavior were reported for male mice fed dietary levels as high as 230 mg/kg/day (Kobayashi et al. 1972, as reported in USEPA 1980). However, at the 230 mg/kg/day dosage levels, slight abnormalities in liver histopathology were observed. These authors concluded that 100 mg/kg/day was a maximum no-effect level in mice.

No information is available on the chronic toxicity, mutagenicity or teratogenicity of 2,4-dichlorophenol. Topical application of 0.3% dimethylbenzanthracene in benzene as an initiator followed by 20% (312 mg/kg) 2,4-dichlorophenol twice weekly for 39 weeks promoted papillomas and carcinomas in mice (Boutwell and Bosch 1959, as reported in NAS 1980).

The acute toxic effects of 2,4-dichlorophenol on aquatic fish life is reflected by 96-hour LC50 values of 2.6 mg/liter for cladoceran, 2.02 mg/liter for the bluegill and 8.23 mg/liter for the juvenile fathead minnow. Chronic toxicity was observed in the fathead minnow exposed to concentrations ranging from 290 to 460 µg/liter. The toxicity of 2,4-dichlorophenol to aquatic plants occurs at much higher concentrations, ranging from 50 to 100 mg/liter depending on the species. Flavor impairment studies showed that fish flavor became tainted in 2,4-dichlorophenol concentrations ranging from 0.4 µg/liter for the largemouth bass to 14 µg/liter for the bluegill (USEPA 1980).

EPA has established an ambient water quality criterion of 3.09 mg/liter for the protection of human health from the toxic properties of 2,4-dichlorophenol.

EPA has not yet established an aquatic life water quality criterion for 2,4-dichlorophenol.

2,4-Dimethylphenol

No information on the toxicity of 2,4-dimethylphenol in humans is available (NAS 1977). Acute toxicity has been observed in rats, mice, and rabbits (NAS 1977). Irritation of mucous membranes, enlargement of blood vessels of the ears and extremities, and excitability followed by lethargy were observed in rats and mice exposed by inhalation to 2,4-dimethylphenol. In the same studies, oral LD50 values for rats and mice were reported as 3,200 mg/kg and 809 mg/kg, respectively; a dermal LD50 of 1,040 mg/kg was reported for rats (Uzhdovini et al. 1974, as reported in USEPA 1980). Topical papillomas have been reported in mice treated with 2,4-dimethylphenol twice weekly for 28 weeks (Boutwell and Bosch 1959, as reported in NAS 1977).

The acute toxicity of 2,4-dimethylphenol on aquatic organisms is reflected in the 96-hour LC50 values of 2.12 mg/liter for cladoceran, 16.75 mg/liter for the fathead minnow, and 7.75 mg/liter for the bluegill. Chronic toxicity was observed in the fathead minnow in concentrations ranging from 1.5 to 3.2 mg/liter. Chronic tests with invertibrate species, which appear to be most sensitive to 2,4-dimethylphenol, have not been performed (USEPA 1980). Algae

and duckweed were affected by concentrations of 500 mg/liter and 292.8 mg/liter, respectively. No information is available on the toxic effects of 2,4-dimethylphenol on saltwater organisms (USEPA 1980).

EPA has not yet established an ambient water quality criterion for 2,4-dimethylphenol because of the lack of sufficient information. However, using available organoleptic data for control of undesirable taste and odor quality of ambient water, the estimated level is 400 µg/liter.

4,6-Dinitro-o-cresol

A number of human poisonings by 4,6-dinitro-o-cresol have been reported. 4,6-Dinitro-o-cresol may be absorbed in acutely toxic amounts through the respiratory and gastrointestinal tracts and through the skin. Signs and symptoms of both acute and chronic poisoning include profuse sweating, malaise, thirst, lassitude, loss of weight, headache, sensation of heat, and yellow staining of the skin, hair, sclera, and conjunctiva. Other effects occasionally reported include kidney damage, diarrhea, disorders of the gastrointestinal tract, cardiovascular system, and peripheral vascular and central nervous systems (Doull et al. 1980, and USEPA 1980). It has been estimated that 5 mg/kg may prove fatal to humans (Fairchild 1977, as reported in USEPA 1980). A study was conducted in which five male volunteers were given oral doses of 75 mg/day of 4,6-dinitro-o-cresol for five consecutive days (Harvey et al. 1951, as reported in USEPA 1980). At blood levels of 20 mg/kg, an exaggerated sense of well-being was experienced. At blood levels of 40 to 48 mg/kg, headache, lassitude, and malaise were reported. In patients who received 4,6-dinitro-o-cresol for the treatment of obesity during the 1930s, poisonings, deaths, and the development of cataracts were reported. Signs of poisoning occurred in three people who had taken as little as 0.35 to 1.5 mg/kg/day (NIOSH 1978, as reported in USEPA 1980). In a Russian study of agricultural workers, exposure to 0.7 to 0.9 mg/cu m produced unspecified changes in the blood, cardiovascular, and autonomic and central nervous systems and in the gastrointestinal tract (Burkatskaya 1965, as reported in USEPA 1980).

Acute oral LD50 values have been reported for the rat (30-85 mg/kg), mouse (16.4-47 mg/kg), and rabbit (24.8 mg/kg) (USEPA 1980). LD50 values for subcutaneous administration in the rat, mouse, and goat range from 20-50 mg/kg. 4,6-Dinitro-o-cresol is less toxic by the dermal route, with LD50s for the mouse and rabbit of 187 and 1000 mg/kg, respectively (USEPA 1980). In a feeding study with rats, no adverse effects were observed among rats on diets containing 100 mg 4,6-dinitro-o-cresol/kg food. At 1000 mg/kg, observed effects included weight loss, emaciation, unkempt appearance, and minor histopathological effects on the liver, kidneys, and spleen (Spencer et al. 1948, as reported in USEPA 1980). Ambrose (1942, as reported in USEPA 1980) reported no observable effects in rats fed diets containing 63 mg 4,6-dinitro-o-cresol/kg food for 105 days. At dietary levels of 125 mg/kg food, 60 percent of the animals died. Cats exposed to airborne 4,6-dinitro-o-cresol at 0.2 mg/cu m for 2-3 months had slightly increased body temperatures and leukocyte counts and decreased hemoglobin concentrations, erythrocyte counts, and catalase and peroxidase activities (Burkatskaya 1965, as reported in USEPA 1980).

No aquatic toxicity data are available for 4,6-dinitro-o-cresol.

EPA has established an ambient water quality criterion of 13.4 µg/liter for protection of human health from the toxic properties of 4,6-dinitro-o-cresol through ingestion of contaminated water and aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for 4,6-dinitro-o-cresol.

2,4-Dinitrophenol

2,4-Dinitrophenol is a potent uncoupler of oxidative phosphorylation. This prevents the utilization of energy provided by cellular respiration and glycolysis by inhibiting the formation of high energy phosphate bonds. 2,4-Dinitrophenol may also act directly on the cell membrane causing toxic effects on cells not dependent on oxidative phosphorylation for their energy requirements (USEPA 1980). Acute poisoning in humans from 2,4-dinitrophenol results in sudden onset of pallor, burning thirst, agitation, dyspnea, profuse sweating, and hyperpyrexia (Horner 1942, as reported in USEPA 1980). Therapeutic doses of 2,4-dinitrophenol have resulted in skin rashes with intense itching and considerable swelling (Tainter et al. 1933, as reported in USEPA 1980). A loss of taste for salt and sweets has been reported in some patients treated therapeutically with 2,4-dinitrophenol (Tainter et al. 1933, as reported in USEPA 1980). The development of cataracts in humans has been clearly demonstrated after use of 2,4-dinitrophenol (USEPA 1980). Bone marrow effects (agranulocytosis) and neuritis have also been reported in humans (Horner 1942, as reported in USEPA 1980).

In animals, an increase in the percentage of stillborn young and neonatal deaths has been reported in a study of rats treated with 20 mg/kg 2,4-dinitrophenol, 8 days prior to mating (Wulff et al. 1935, as reported in USEPA 1980).

The acute toxicity of 2,4-dinitrophenol on freshwater aquatic organisms is reflected in LC50 values of 4.09 mg/liter for Daphnia magna, 16.7 mg/liter for the juvenile fathead minnow, and 0.62 mg/liter for the bluegill. Increased respiration was observed in the tadpoles of Southern bullfrogs exposed to 5.52 mg/liter for 7 hours. A 96-hour lethal threshold value of 0.7 mg/liter has been reported for juvenile Atlantic salmon (USEPA 1980).

Saltwater organisms are also affected by 2,4-dinitrophenol. Mysid shrimp had a 96-hour LC50 of 4.85 mg/liter; the embryo of herring, a 96-hour LC50 of 5.5 mg/liter; and sheepshead minnow, a 96-hour LC50 of 29.4 mg/liter. Respiration and motility were reported to be inhibited in the sperm of sea urchin exposed to 92.0 mg/liter for more than 1 hour. Abnormal cleavage in the embryo of the sea urchin was also reported to occur at 46 mg/liter for 2 hours. At levels of 10 mg/liter, complete mortality of Lymnaeid snails was reported within 24 hours. The chronic effects of 2,4-dinitrophenol on hatching and survival in an early life test with the sheepshead minnow resulted in limits of 7.9 mg/liter (USEPA 1980).

Freshwater plants also exhibited a toxicity to 2,4-dinitrophenol. Chlorophyll synthesis in algae was inhibited after 3 days exposure to 50 mg/liter. A 50% reduction in growth was observed in duckweed exposed to 1.47 mg/liter. Eight-day toxicity thresholds for different species of algae varied from 16-33 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 70 µg/liter for the protection of human health from the toxic properties of total dinitrophenols through ingested water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for 2,4-dinitrophenol.

2-Nitrophenol

The acute toxic effects of 2-nitrophenol include kidney and liver damage and methemoglobin formation in experimental animals (Sax 1975). Experimental studies have shown 2-nitrophenol to inhibit chloride transport in red blood cells in rats (Motais et al. 1978, as reported in USEPA 1980) and to increase the platelet count in rats administered 1 mg/kg by intraperitoneal injection (Gabor et al. 1960, as reported in USEPA 1980). Oral LD50 values are reported for the rat and mouse as 2,830 mg/kg and 1,300 mg/kg, respectively (Vernot et al. 1977, as reported in USEPA 1980). No other information on the toxicity of 2-nitrophenol in human or nonhuman mammals is available.

The 24-hour LC50s of 2-nitrophenol in freshwater organisms are 210 mg/liter for Daphnia magna and 66.9 mg/liter for juvenile bluegill. Eight-hour exposures of gold fish to 33.3 mg/liter resulted in 38% mortality. Shrimp showed 96-hour lethal threshold values of 32.9 mg/liter. No chronic toxicity information is available on any aquatic organisms (USEPA 1980).

Freshwater plants were reported to be more susceptible to the effects of 2-nitrophenol than aquatic fishlife. Inhibition of chlorophyll synthesis was observed in algae treated with 35 mg/liter for 3 days and a 50% reduction in growth was reported in duckweed exposed to 62.5 mg/liter (USEPA 1980).

EPA has not yet established ambient water quality criteria for 2-nitrophenol because of the lack of sufficient information.

4-Nitrophenol

No information is available on the acute or chronic toxicity of 4-nitrophenol to humans (USEPA 1980). The known effects of 4-nitrophenol demonstrated by animal studies include methemoglobinemia and shortness of breath (Von Oettingen 1949, as reported in USEPA 1980). 4-Nitrophenol inhibited chloride transport in rat red blood cells (Motais et al. 1978, as reported in USEPA 1980) and increased respiratory volume in rats administered 7-12 mg by stomach tube (Grant 1959, as reported in USEPA 1980). Oral LD50s for the rat and mouse are reported as 350 and 470 mg/kg, respectively (Fairchild 1977, Vernot et al. 1977, as reported in USEPA 1980).

Acute toxicity of 4-nitrophenol has been demonstrated in freshwater aquatic fishlife. 96-Hour LC50 values reported for 4-nitrophenol for bluegills and fathead minnows are 8.28 mg/liter and 60.5 mg/liter, respectively. The toxicity of this compound to daphnids shows a wide variation with values ranging from 8.4 to 21.9 mg/liter. Mortality of 42% in goldfish was reported after 8 hours exposure to 8.0 mg/liter. 4-Nitrophenol produced 96-hour LC50s of 7.17 mg/liter in mysid shrimp and 27.1 mg/liter in the sheepshead minnow. 96-Hour lethal threshold values for shrimp and soft-shell clams were reported to be 26.4 and 29.4 mg/liter, respectively (USEPA 1980).

Chronic studies on hatching and survival in the early life stage test showed toxic effects in concentrations ranging from 10 to 16 mg/liter for sheepshead minnows. No other information is available on the chronic effects on aquatic organisms (USEPA 1980).

Inhibition of chlorophyll synthesis was reported in alga exposed to 25 mg/liter after 3 days. Growth inhibition of 50% was also reported in alga exposed to 6.95 mg/liter for 80 hours and in duckweed exposed to 9.45 mg/liter (USEPA 1980).

EPA has not yet established ambient water quality water criteria for 4-nitrophenol because of the lack of sufficient information.

Pentachlorophenol

Exposure of pentachlorophenol in humans has been reported to cause loss of appetite, eye irritation, respiratory difficulties, anesthesia, hyperpyrexia, sweating, dyspnea, skin irritation, and rapidly progressive coma (Menon 1958, as reported in NAS 1977). The minimum lethal dose for humans is estimated to be 29 mg/kg (Toxic Substance List 1974, as reported in NAS 1977). Chronic exposure to pentachlorophenol has been associated with the development of chloracne, a type of acneform dermatitis (Baader and Bauer 1951 and Nomura 1953, as reported in USEPA 1980). Nonfatal chronic exposures can produce muscle weakness, headache, anorexia, abdominal pain, and weight loss, in addition to skin, eye, and respiratory irritation (USEPA 1980).

Acute symptoms in animals include vomiting, hyperpyrexia, elevated blood pressure, increased respiration rate, and tachycardia (NAS 1980). The acute oral LD50s for pentachlorophenol are reported in the ranges of 120-140 mg/kg for the mouse, 27-100 mg/kg for the rat, 100 mg/kg for the guinea pig, 100-130 mg/kg for the rabbit, and 150-200 mg/kg for the dog (Christensen et al. 1974, Deichmann et al. 1942, Knudsen et al. 1974, McGavack et al. 1941, Stohlman 1951, as reported in NAS 1977). Teratogenic effects have been reported in rats orally administered amounts up to 50 mg/kg/day during day 6-15 of gestation (Schwetz et al. 1974, as reported in NAS 1977). No evidence of mutagenicity was found in several short-term bioassays (Anderson et al. 1972, Fahrig et al. 1978, Vogel and Chandler 1974, Buselmaier et al. 1973, as reported in USEPA 1980).

Dermal application of a 20% solution of pentachlorophenol dissolved in benzene did not increase the rate of papillomas in mice pretreated with

dimethylbenzanthracene (Boutwell and Bosch 1959, as reported in USEPA 1980). Mice dosed with commercial pentachlorophenol at 46.4 mg/kg from 7-28 days of age and then fed 130 ppm in the diet for the remainder of their life did not have a significant increase in tumors (Innes et al. 1969, as reported in USEPA, 1980). No effect was also observed in rats fed amounts up to 30 mg/kg for 22-24 months (Schwetz et al. 1978, as reported in USEPA 1980).

Pentachlorophenol has been shown to be acutely toxic to a wide variety of freshwater fish. No chronic test data are available. For nine fish species tested, the 96-hour LC50 values ranged from 37 to 340 µg/liter. Toxicity tests gave 96-hour LC50 values ranging from 50-130 µg/liter in sockeye salmon, from 60-77 µg/liter in the bluegill, and 340 µg/liter in fathead minnows (USEPA 1980).

Saltwater marine life are also affected by pentachlorophenol. Adjusted 96-hour LC50 values for sheepshead minnows, pinfish, and striped mullet ranged from 21 to 442 µg/liter. Pentachlorophenol appears to be most toxic to molluscs; shrimp are less sensitive and oysters more sensitive than fish. Oyster embryos develop abnormally when exposed to 55 µg/liter for 48 hours. Lugworm feeding activity was significantly inhibited by concentrations of 80 µg/liter pentachlorophenol during a 144-hour exposure. Chronic studies of saltwater organisms show that 195 µg/liter significantly reduced hatching of embryos spawned by exposed parental fish and reduced survival of second generation sheepshead minnows in a 151-day life cycle exposure (USEPA 1980).

In plant tests, pentachlorophenol caused complete destruction of chlorophyll in algae in 72 hours at 7.5 µg/liter, and in kelp in 4 days at 2.66 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 1.01 mg/liter for protection of public health from the toxic properties of pentachlorophenol ingested through water and contaminated fish.

EPA has not yet established an aquatic life water quality criterion for pentachlorophenol.

Phenol

The predominant effect of phenol on humans is on the central nervous system leading to sudden collapse and unconsciousness (USEPA 1980). Numerous cases of phenol toxicity resulting from occupational exposures have reported symptoms including shock, collapse, coma, convulsions, cyanosis, and death (StajduhavCaric 1968, Noury 1940, Johstone and Miller 1960, Cronin and Brauer 1949, Duvernevil and Ravier 1962, Abraham 1972, Light 1931, as reported in USEPA 1980).

People who had consumed estimated daily doses of 10-240 mg phenol in well water for approximately 1 month developed burning of the mouth, mouth sores, diarrhea, headaches, skin rashes, abdominal pain, dizziness, and dark urine (Baker et al. 1978, as reported in USEPA 1980).

The toxic effects observed in animals are quite similar to those in humans. The pathological changes produced by phenol in animals vary with the route of absorption, vehicle employed, concentration, and duration of exposure. Local damages to the skin include eczema, inflammation, discoloration, papillomas, necrosis, sloughing, and gangrene. Following oral ingestion, the mucous membranes of the throat and esophagus may show swelling, corrosions, and necroses, with hemorrhage and serious infiltration of the surrounding areas. In a severe intoxication, the lungs may show hyperemia, infarcts, bronchopneumonia, purulent bronchitis, and hyperplasia of the peribronchial tissues. There can be myocardial degeneration and necrosis. The hepatic cells may be enlarged, pale, and coarsely granular with swollen, fragmented, and pyknotic nuclei. Prolonged administration of phenol may cause parenchymatous nephritis, hyperemia of the glomerular and cortical regions, cloudy swelling, edema of the convoluted tubules, and degenerative changes of the glomeruli. Blood cells become hyaline, vacuolated, or filled with granules. Muscle fibers show marked striation (Deichman and Keplinger 1963, as reported in USEPA 1980).

The acute toxicity of phenols in mammals ranges from an oral LD50 of 100 mg/kg in the cat (Macht 1915, as reported in USEPA 1980) to 620 mg/kg in the rabbit (Clark and Brown 1906, as reported in USEPA 1980). Pathological changes reported in mammals include intense congestion of the peritoneum, abdominal viscera, kidney and adrenals, and marked degenerative changes in the kidney. In rats fed 8,000 mg/liter in their drinking water over two generations, there were reduced growth rates in the young with many deaths (Heller and Pursell 1938, as reported in USEPA 1980). In another study, no effects were observed in rats fed for 12 months at concentrations as high as 2,400 mg/liter in the drinking water (Diechmann and Oesper 1940, as reported in USEPA 1980). Slight kidney and liver effects were reported in rats administered 20 daily doses of 0.1 g/kg by gavage (Unpublished report of Dow Chemical 1976, as reported in USEPA 1980). Skin painting studies of mice exposed to 20% phenol concentrations have produced skin ulcerations, exhibited strong promoting action on tumor development, and exhibited a weak carcinogenic response (Salaman and Glendenning 1957, as reported in USEPA 1980).

The acute toxicity of phenol to freshwater vertebrates ranges from 44.5 mg/liter at 96 hours for the goldfish to 10.2 mg/liter for rainbow trout. Rainbow trout was the most sensitive species tested with a 24-hour LC50 of 5.0 mg/liter for embryos. Juvenile rainbow trout were killed at 6.5 mg/liter phenol in 2 hours. At these concentrations, there was rapid damage to gills and severe pathology of other tissues. Typical gross pathological changes have also been reported and include internal hemorrhages, deterioration of gill membranes, degradation of the liver, and brain damage. Phenol appears to act as a nerve poison causing too much blood to flow to the gills and to the heart cavity of the fish. Pathological changes in the gills and in fish tissue have been found at concentrations of 20-70 µg/liter. Phenol is acutely toxic to freshwater invertebrates, although it appears to be less toxic to fish food organisms and lower aquatic life than to fish. The 48-hour LC50 for the freshwater flea is 9.6 mg/liter. A concentration of 2.0 mg/liter inhibited egg development in oysters and reduced oxygen consumption approximately 50% in the freshwater snail. Phenols inhibit chlorophyll

synthesis, and cause complete destruction of algae at concentrations of 50 and 1,500 mg/liter, respectively (USEPA 1980).

EPA has established an ambient water quality criterion of 3.5 mg/liter for the protection of human health from the toxic properties of phenol through ingestion of contaminated water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for phenol.

2,4,6-Trichlorophenol

No information is available on the toxic effects of 2,4,6-trichlorophenol on humans. In animal studies, a number of different adverse effects have been observed. Convulsions were produced in rats injected intraperitoneally with 2,4,6-trichlorophenol during an acute toxicity test. The LD50 was estimated at 276 mg/kg in this test (Farquharson et al. 1958, as reported in USEPA 1980). 2,4,6-Trichlorophenol was reported to cause inhibition of lactate dehydrogenase and hexokinase, in vitro, in concentrations ranging from 0.0028-0.005 Molar (species unspecified; Stockdale and Selwyn 1971, as reported in USEPA 1980). 2,4,6-Trichlorophenol was shown to penetrate the rabbit eye with the highest amounts concentrating in the cornea and conjunctiva (Ismail et al. 1975, as reported in USEPA 1980). However, the significance of this finding has not yet been established.

No information is available on the subacute or chronic effects of 2,4,6-trichlorophenol. In a mutagenicity study using a strain of Saccharomyces cerevisiae, 400 mg of 2,4,6-trichlorophenol increased the mutation rate (Fahrig et al. 1978, as reported in USEPA 1980). Evidence of a genetic change in the offspring of mothers administered 50 or 100 mg/kg of 2,4,6-trichlorophenol on day 10 of gestation was reported in a mouse spot test (Fahrig et al. 1978, as reported in USEPA 1980). 2,4,6-Trichlorophenol was negative in the Salmonella-mammalian microsome Ames test (Rasanen et al. 1977, as reported in USEPA 1980). In a lifetime feeding study, 2,4,6-trichlorophenol was shown to be carcinogenic in male rats, including lymphomas or leukemias, and in both male and female mice, including hepatocellular carcinomas or adenomas. Both tests were conducted with dosage levels of approximately 5,000 ppm in the diet (NCI 1979, as reported in USEPA 1980). The topical application of a 20% solution of 2,4,6-trichlorophenol in benzene did not increase the incidence of papillomas in mice pretreated with dimethylbenzanthracene (Boutwell and Bosch 1958, as reported in USEPA 1980).

The acute toxicity of 2,4,6-trichlorophenol on aquatic life is reflected by the following 96-hour LC50 values for the cladoceran, 6 mg/liter, the fathead minnow, 0.6 mg/liter, the juvenile fathead minnow, 9 mg/liter, and the bluegill, 0.3 mg/liter. Chronic toxicity was observed in an early life stage test using fathead minnow exposed to concentrations ranging from 0.53-0.97 mg/liter. One hundred percent mortality was observed in lymnaeid snails exposed to 5 mg/liter for 24 hours. The highest estimated concentration (ETC) of 2,4,6-trichlorophenol that will not impair the flavor of fish is 52 µg/liter for a 48-hour exposure of rainbow trout. Complete destruction of

chlorophyll was observed in an alga exposed to 10 mg/liter and chlorosis was reported in duckweed exposed to 5.9 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to 2,4,6-trichlorophenol through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 1.2 µg/liter, corresponding to a lifetime incremental cancer risk of 10-6, was recommended.

EPA has not yet established an aquatic life water quality criterion for 2,4,6-trichlorophenol.

3. Base/Neutral Extractable Organic Compounds

Acenaphthene

Very little information on the toxic effects of acenaphthene in humans is available. It is irritating to skin and mucous membranes, and may cause vomiting if swallowed in large quantities (Sax 1975). In animals, oral administration of 2 g/kg daily for 32 days was reported to cause loss of body weight, changes in peripheral blood, heightened amino transferase levels in blood serum, and mild morphological damage to both liver and kidneys in rats. In the same studies, oral LD50s for rats and mice were 10 g/kg and 2.7 g/kg, respectively (Knoblock et al. 1969, as reported in USEPA 1980). Chronic inhalation studies showed toxic effects to the blood, lungs, and glandular constituents in rats exposed to 12 mg/cu m, 4 hours per day, six days per week for 5 months (Reshetyuk et al. 1970, as reported in USEPA 1980).

No information is available on the teratogenicity of acenaphthene and the only data on mutagenicity, using microoganisms as the indicator system, were negative (USEPA 1980). Very little work has been done to determine whether acenaphthene may have carcinogenic potential. Negative results were obtained in the newt with acenaphthene injected subcutaneously (dosage not reported, USEPA 1980). The only other carcinogenicity studies available involve acenaphthene as a component of a complex mixture of polycyclic aromatic hydrocarbons (USEPA 1980).

In one study, isolated polycyclic hydrocarbon-rich fractions from the neutral portion of cigarette smoke condensate were applied (dose unspecified) to the dorsal skin of female mice, five times a week for 13 months. The acenaphthene containing extracts were applied five weeks after the animals were painted once with 125 µg of 7,12-dimethylbenz(a)anthracene. No significant tumor-promoting activity over controls were observed (Akin et al., 1976 as reported in USEPA 1980). In a second study benzene extracts, of gasoline exhaust condensates containing an unspecified concentration of acenapthene, were reported to be carcinogenic in mouse skin painting studies (details unspecified; Hoffman and Wynder, 1962 as reported in USEPA 1980).

The most thoroughly investigated effect of acenaphthene is its ability to produce nuclear and cytological changes in microbial and plant species. Most of these changes, such as an increase in cell size and DNA content, are

associated with disruption of the spindle mechanisms during mitosis and the resulting induction of polyploidy (USEPA 1980).

The acute toxicity of acenaphthene to aquatic organisms is reflected in a static 48-hour LC50 of 41.2 mg/liter for Daphnia magna; and static 96-hour LC50 values of 1.7 mg/liter in the bluegill, 0.97 mg/liter in the mysid shrimp and 2.23 mg/liter in the sheepshead minnow. Chronic toxicity by the embryo-larval test was observed in the sheepshead minnow. Algae were affected at 500-530 µg/liter. Bluegill were reported to accumulate acenaphthalene during a 28-day exposure and a bioconcentration factor of 387 was reported for this same species (USEPA 1980).

Sufficient data were not available for EPA to establish ambient water quality criteria that would protect human health against the potential toxicity of acenaphthene. However, a level of 0.02 mg/liter was recommended based on available organoleptic data, for controlling undesirable taste and odor quality of ambient water.

EPA has not yet established an aquatic life water quality criterion for acenaphthene.

Acenaphthylene

No information is available on the toxic effect of acenaphthylene on humans, animals, or aquatic life. EPA has not yet established ambient water quality criteria for acenaphthylene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include acenaphthylene, through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 2.8 ng/liter, corresponding to a lifetime incremental cancer risk of 10-6, was recommended.

Anthracene

No information is available on the toxicity of pure anthracene to humans. However, anthracene oils can cause headaches, nausea, loss of appetite, skin rashes, and irritation of the mucous membranes (Encyclopedia of Occupational Health and Safety 1971). Various oils of anthracene and substances containing anthracene have also been associated with carcinogenesis in animals (Searle 1976). Anthracene exhibits relatively low toxicity to animals. The acute oral toxicity in rats is greater than 3,200 mg/kg. Large oral doses in rats caused reaction of the abdominal wall, rough coat, and diarrhea (Patty 1963). Anthracene applied to the backs of guinea pigs caused a slight skin irritation on the pig skin (Patty 1963). No information is available on the carcinogenic effects of pure anthracene.

There is limited information on the toxicity of anthracene to aquatic organisms. A 90% lethal photodynamic response was observed in protozon

exposed to 0.1 µg/liter for 60 minutes. Bioconcentration factors ranged from 200 in the Daphnia magna to 3,500 in the may fly (USEPA 1980).

EPA has not yet established ambient water quality criteria for anthracene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include anthracene, through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 2.8 ng/liter, corresponding to a lifetime incremental cancer risk of 10-6, was recommended.

Benz(a)anthracene

No information is available on the toxic effects of benz(a)- anthracene on humans. Most of the studies in animals have been conducted to evaluate the carcinogenic potential of benz(a)anthracene. This is especially true of the derivatives of benz(a)anthracene. Benz(a)anthracene has been shown to be carcinogenic in the mouse by several routes of administration (IARC 1973). Oral administration of 1.5 mg as a 3% solution in "methocelaerosol OF" by stomach tube, for 15 treatments over a 5-week period, resulted in the development of hepatomas and lung adenomas (Klein 1963, as reported in IARC 1973). Skin tumors developed when benz(a)anthracene was applied topically 3 times a week, to mice, in a 1% concentration in toluene and a 0.002% concentration in n-dodecane (Bingham and Falk 1969, as reported in IARC 1973). Sarcomas were also produced in mice following subcutaneous injections with as low as 50 µg administered as a single dose (Steiner and Edgecomb 1952, as reported in IARC 1973). No information is available on the mutagenicity or teratogenicity of benz(a)anthracene (USEPA 1980).

The only information available on the toxic effects of benz(a)anthracene on aquatic life is a 6-month study of bluegill exposed to 1.0 mg/liter. In this study, 87% mortality was observed (USEPA 1980).

EPA has not yet established ambient water quality criteria for benz(a)anthracene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include benz(a)anthracene, through ingestion of contaminated water and contaminated aquatic organisms. However, since the zero level may not be attainable at present, a level of 2.8 ng/liter, corresponding to a lifetime incremental cancer risk of 10-6, was recommended.

Benzidine

Epidemiological studies show that occupational exposure to benzidine is strongly associated with bladder cancer. A high incidence of bladder tumors was reported in workmen exposed to benzidine or benzidine and aniline in British chemical factories. Thirty cases of bladder tumors were reported, with a mean latent period of ten years (Case et al. 1954, as reported in

USEPA 1980). In a retrospective study of a single factory, 17 of 76 workmen exposed to benzidine alone developed bladder tumors (Goldwater et al. 1965, as reported in IARC 1972). Bladder cancer has been reported in several studies of Italian dyestuff workers. In a study by Barsotti and Vigliani (1952, as reported in USEPA 1980), 13 of 83 workers developed bladder carcinomas from exposure to benzidine during the period 1931 to 1948. Dermatitis and increased urinary β-glucuronidase activity have also been reported in workers exposed to benzidine (USEPA 1980).

Benzidine is also carcinogenic to experimental animals. Cholangiomas and liver-cell tumors were reported in lifetime feeding studies with rats fed 0.017% benzidine in the diet and hamsters fed 0.1% in the diet (Boyland et al. 1954, Saffiotti et al. 1967, as reported in IARC 1972). Three of seven dogs given 200-300 mg/day, 6 days/week, for 5 years developed bladder carcinomas seven to nine years after the start of treatment (Spitz et al. 1950, as reported in IARC 1972). Benzidine administered subcutaneously to rats at a dose of 15 mg/week for their lifespan produced liver injury, cirrhosis, hepatomas, sebaceous gland carcinomas, and adenocarcinomas of the rectum (Spritz et al. 1950, as reported in USEPA 1980). A cumulative dose of 0.75 mg/kg of benzidine given subcutaneously for 15 days produced tumors in 20 of 22 rats, including hepatomas, cholangiomas, intestinal tumors, and sebaceous gland carcinomas (Holland et al. 1974, as reported in USEPA 1980). Hepatomas have been reported in mice given subcutaneous injections of benzidine (USEPA 1980).

In addition to its carcinogenic activity, benzidine also causes a reduction in catalase and peroxidase activity, a reduction in erythrocytes and thrombocytes, and an increase in leukocytes when injected in rats (Soloimskaya 1968, as reported in USEPA 1980). Neish (1967, as reported in USEPA 1980) reported that an intraperitoneal dose of 12.7 mg/kg in rats increased liver glutathione levels. Benzidine is mutagenic in the Ames assay with *Salmonella typhimurium* and gave positive results in a DNA synthesis inhibition test with HeLa cells (USEPA 1980).

Limited toxicity data are available for aquatic organisms. The 96-hour LC50 for rainbow trout and lake trout, red shiner, and the flagfish range from 2,500 to 16,200 µg/liter. Chronic toxicity data for freshwater organisms are not available. No saltwater organisms have been tested with benzidine (USEPA 1980).

EPA has established an ambient water quality criterion of zero for maximum protection of human health from the potential carcinogenic effects due to exposure to benzidine through the ingestion of contaminated water and aquatic organisms. However, since the zero level may not be attainable at the present time, a level of 0.00012 µg/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended.

EPA has not yet established an aquatic life water quality criterion for benzidine.

3,4-Benzofluoranthene

3,4-Benzofluoranthene is a member of the class of polynuclear aromatic hydrocarbons (PAHs). Many members of this class of chemicals are carcinogenic. 3,4-Benzofluoranthene has produced skin tumors in mice following repeated skin painting. Three groups of mice were painted with 0.01, 0.1, or 0.5% solutions of 3,4-benzofluoranthene in acetone three times per week. After 8-12 months, the incidence of carcinomas in the survivors was 0%, 85%, and 90% (Wynder and Hoffman 1958, as reported in IARC 1973). In a later study, a single dermal application of 1 mg in acetone produced no tumors in mice after 63 weeks; the same procedure followed by repeated paintings with croton resin produced carcinomas in 5 of 20 mice (Van Duuren et al. 1966, as reported by IARC 1973). 3,4-Benzofluoranthene also produced sarcomas in mice at the site of injection after 3 subcutaneous injections of 0.6 mg of the compound over a two month period (Lacassagne et al. 1963, as reported in IARC 1973).

No human case studies or epidemiological studies have been conducted which establish 3,4-benzofluoranthene as a human carcinogen. Indirect evidence for the compound's carcinogenicity comes from air pollution studies which indicate an excess of lung cancer mortality among workers exposed to high concentrations of PAH-containing material such as coal gas, tars, soot, and coke oven emissions (IARC 1973, USEPA 1980).

No data are available on the aquatic toxicity of 3,4-benzofluoran- thene.

EPA has not yet established ambient water quality criteria for 3,4-benzofluoranthene itself because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include 3,4-benzofluoranthene, through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at present, a level of 2.8 ng/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended.

Benzo(k)fluoranthene

No information is available on the toxic effects of benzo(k)fluoranthene in humans, animals, or aquatic organisms.

EPA has not yet established ambient water quality criteria for benzo(k)fluoranthene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include benzo(k)fluoranthene, through ingestion of contaminated water and aquatic organisms. Since a zero level may not be obtainable at present, a level of 2.8 ng/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

Benzo(g,h,i)perylene

Benzo(g,h,i)perylene is a member of the class of polynuclear aromatic hydrocarbons (PAHs), many of which are known for their ability to induce cancer.

No data are available on the aquatic or mammalian toxicity of benzo(g,h,i)perylene. This compound has not been identified as a carcinogen; however, benzo(g,h,i)perylene acted as a cocarcinogen when applied with benzo(a)pyrene, a known carcinogen, to the skin of mice at a dose of 2 mg, 3 times per week, for 52 weeks (Van Duuren et al. 1973 and 1976, as reported in USEPA 1980).

EPA has not yet established ambient water quality criteria for benzo(g,h,i)perylene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons through ingestion of contaminated water and contaminated aquatic organisms. Since a zero level may not be attainable at present, a level of 2.8 ng/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended.

Benzo(a)pyrene

Benzo(a)pyrene is a member of the class of polynuclear aromatic hydrocarbons (PAHs) and is a well-known animal carcinogen. Administration of 50-250 ppm in the diet to mice for 122 to 197 days resulted in a greater than 70 percent incidence of stomach tumors (Neal and Ridgon 1976, as reported in IARC 1973). Leukemias, lung adenomas, and stomach tumors were produced in mice by dietary administration of 250 ppm benzo(a)pyrene for 140 days (Rigdon and Neal, 1969, as reported in IARC 1973). In hamsters and rats, oral administration of benzo(a)pyrene produced tumors of the esophagus, forestomach, and intestine (IARC 1973).

Benzo(a)pyrene is also carcinogenic when administered by intratracheal instillation. Instillation of 3.25 to 52 mg once weekly for 52 weeks in Syrian golden hamsters produced a respiratory tract tumor incidence of 10 to 93 percent (Feron et al. 1973, as reported in USEPA 1980).

Many experiments have been conducted involving repeated application to the skin or subcutaneous and intramuscular injection. In skin painting studies, the threshold dose to induce tumors is dependent on the species and strain of animal tested and the vehicle used. Thrice weekly applications of benzo(a)pyrene in acetone to CAF1 mice produced papillomas and carcinomas at a concentration as low as 0.001% benzo(a)pyrene (Wynder et al. 1957, as reported in IARC 1973). In a subcutaneous carcinogenicity study using C57 mice, injection of benzo(a)pyrene in oil at doses of 0.00004, 0.0004, 0.004, and 0.04 mg produced sarcomas in, respectively, 0, 1, 5, and 23 mice of groups of 50 (Hieger 1959, as reported in IARC 1973).

Little information on other potential toxic effects of benzo(a)pyrene is available. Benzo(a)pyrene has been shown to have little effect on fertility

or on the developing embryo in several mammalian and nonmammalian studies (USEPA 1980). Benzo(a)pyrene induced damage to the bronchial epithelium of Syrian golden hamsters in animals treated intratracheally with 0.63 mg benzo(a)pyrene (total dose) dispersed in various vehicles once weekly for life (Reznik-Schuller and Mohr 1974, as reported in USEPA 1980).

No human case studies or epidemiologic studies have been conducted which establish benzo(a)pyrene as a human carcinogen. Indirect evidence for the compound's carcinogenicity comes from air pollution studies which indicate an excess of lung cancer mortality among workers exposed to high concentrations of PAH-containing material such as coal gas, tars, soot, and coke-oven emissions (IARC 1973, USEPA 1980).

No data are available on the aquatic toxicity of benzo(a)pyrene.

EPA has not yet established ambient water quality criteria for benzo(a)pyrene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at present, a level of 2.8 ng/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended.

Bis(2-chloroethoxy) methane

No toxicity data for bis(2-chloroethoxy) methane in humans are available, and only limited acute toxicity information in animals has been published. Acute LD50 values for the rat by oral administration and for the guinea pig by dermal administration have been given as 65 mg/kg and 170 mg/kg, respectively (NIOSH 1980). Unspecified toxic effects were observed in rats exposed to 62 ppm bis(2-chloroethoxy) methane vapors for four hours (NIOSH 1980). No chronic studies for this compound have been conducted.

No aquatic toxicity data are available for bis(2-chloroethoxy) methane. Available data for the class of chloroalkyl ethers indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 238 mg/liter (USEPA 1980).

EPA has not yet established ambient water quality criteria for bis(2-chloroethoxy) methane because of the lack of sufficient data.

Bis(2-chloroethyl) Ether

Bis(2-chloroethyl) ether is moderately persistent and insoluble in water. Toxic effects of exposure to bis(2-chloroethyl) ether have been observed in humans. Exposure to 550 ppm was intolerable and caused irritation of the eyes and nasal passages. Bis(2-chloroethyl) ether can also affect the kidney and liver (Sax 1975).

A wide variety of acute effects has been observed in animals. Guinea pigs exposed to 500-1,000 ppm by inhalation immediately developed severe eye and nasal irritation and in 3 hours developed respiratory disturbances and death with pulmonary lesions. Autopsy revealed congestion of the lungs and upper respiratory tract, pulmonary edema, and congestion of the liver, brain, and kidney (Schrenk et al. 1933, as reported in USEPA 1980). Oral LD50s have been reported as 75-150 mg/kg for rats, 136 mg/kg for mice, and 126 mg/kg for rabbits. Only mild physiological stress has been observed in animals exposed chronically to bis(2-chloroethyl) ether. Bis(2-chloroethyl) ether at 300 mg/kg administered orally or by intraperitoneal injection for 80 weeks produced an increased incidence of liver tumors in mice (Innes et al. 1969, as reported in USEPA 1980).

No information is available on the toxic effects of bis(2- chloroethyl) ether on aquatic fish or plant life (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to bis(2-chloroethyl) ether through ingestion of contaminated water and contaminated aquatic organisms. Since the zero level may not be attainable at the present time, a level of 0.030 µg/liter, corresponding to a lifetime incremental risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for bis(2-chloroethyl) ether.

Bis(2-chloroisopropyl) Ether

No evidence of human toxicity from exposure to bis(2-chloro- isopropyl) ether is available. Acute toxicity of bis(2-chloroisopropyl) ether has been reported in rats exposed by inhalation to 350 ppm for eight, 5-hour exposures. Toxic effects included respiratory difficulty, lethargy, retarded weight gain, and congestion of the liver and kidneys. Lethargy and retarded weight gain were also observed in rats exposed to 70 ppm for 20, 6-hour exposures (Gage 1979, as reported in USEPA 1980). An oral LD50 for the rat was established as 240 mg/kg and a dermal LD50 for the rabbit is reported as 3,000 mg/kg (Smyth et al. 1951, as reported in USEPA 1980).

In a chronic oral toxicity study, the major toxic effects observed in the rat exposed to 200 mg/kg/day for a total of 728 days were on the lungs, where congestion, pneumonia, and aspiration were noted. Centrilobular necrosis of the liver, hyperkeratosis of the esophagus, and agiectasis of the adrenal cortex were also reported. In this same experiment, mice exposed to 10 mg/kg/day also developed centrilobular necrosis of the liver (NCI, unpublished as reported in USEPA 1980). Bis(2-chloroisopropyl) ether is mutagenic in in vitro assays (USEPA 1980). Tumors were induced by bis(2-chloroisopropyl) ether in rats and mice fed 200 and 25 mg/kg/day respectively for 5 days per week for two years. However, the significance of this result has not been fully determined because of high tumor incidence in controls (NCI, unpublished as reported in USEPA 1980).

No information is available on the toxicity of bis(2-chloroisopropyl) ether on aquatic organisms (USEPA 1980).

EPA has established a limit of 3.47 µg/liter for the protection of human health against the toxic properties of bis(2-chloroisopropyl) ether through ingestion of water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for bis(2-chloroisopropyl) ether.

Bis-2-ethylhexyl Phthalate

Toxic effects of bis-2-ethylhexyl phthalate in humans have not been reported. In chronic animal studies, liver and kidney weights increased in both the parental (P1) generation of rats fed 0.4% bis-2-ethylhexyl phthalate for a maximum of 2 years and in their offspring also fed 0.4% bis-2-ethylhexyl phthalate for 1 year; and in a second study, at 0.5% for up to 2 years (Carpenter 1953 and Harris et al. 1956, as reported in USEPA 1980). Liver damage was reported in monkeys exposed to bis-2-ethylhexyl phthalate solubilized in blood (administered as transfusions) in concentrations ranging from 6.6 to 33 mg/kg for periods ranging from 6 months to 1 year (Kevy et al. 1978, as reported in USEPA 1980). Tubular atrophy and degeneration in the testes were observed in rats administered bis-2-ethylhexyl phthalate in the diet at 1.5 and 3.0% for 90 days (Shaffer et al. 1945, as reported in USEPA 1980). Oral LD50 values have been reported in the rat as 26.0 mg/kg, and in the rabbit as 34.0 mg/kg. A dermal LD50 has been reported in the guinea pig as 10.0 mg/kg (Autian 1973, as reported in USEPA 1980). Dose-related skeletal abnormalities and reduced fetal weight were reported in rats administered 5 ml/kg bis-2-ethylhexyl phthalate by intraperitoneal injection during days 5, 10, and 15 of gestation (Singh et al. 1972, as reported in USEPA 1980).

In a recent National Cancer Institute/National Toxicology Program bioassay (NTP 1982), bis-2-ethylhexyl phthalate was carcinogenic to both rats and mice fed diets containing the test chemical at concentrations of 6,000 or 12,000 ppm (rats) and 3,000 or 6,000 ppm (mice) for 103 weeks. An increased incidence of hepatocellular carcinomas was observed in high dose female rats and male mice and in both groups of female mice. In addition, degeneration of the seminiferous tubules was observed in the high-dose male rats and mice, and hypertrophy of the cells in the anterior pituitary was found in the high-dose male rats.

In freshwater aquatic life 48-hour LC50 values ranging from 1 to 5 mg/liter, for Daphnia magna and greater than 18 mg/liter for the midge have been reported. The 96-hour LC50 values were reported as greater than 32 mg/liter for the scud and greater than 770 mg/liter for the bluegill. Significant increase in total body protein catabolism was reported in rainbow trout exposed to 14-54 µg/liter for 24 days. Bis-2-ethylhexyl phthalate at 100 µg/g in the diet of the guppy caused an increase in abortions. Significant reproductive impairment in Daphnia magna was reported after chronic exposure to bis-2-ethylhexyl phthalate at 3 µg/liter. No other

information was available on the chronic toxic effects of bis-2-ethylhexyl phthalate or on its toxic effects in aquatic plantlife (USEPA 1980).

EPA has established an ambient water quality criterion of 15 mg/liter for the protection of human health from the toxic properties of bis-2-ethylhexyl phthalate ingested through water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for bis-2-ethylhexyl phthalate.

4-Bromophenyl Phenyl Ether

No human or mammalian toxicity data are available for 4-bromophenyl phenyl ether.

Limited aquatic toxicity data are available for 4-bromophenyl phenyl ether. For *Daphnia magna*, the 48-hour EC50 is 360 µg/liter, and the 96-hour LC50 for the bluegill is 4,940 µg/liter. In an embryo-larval test with the fathead minnow, adverse effects on survival and growth were observed at 122 µg/liter (USEPA 1980).

EPA has not yet established ambient water quality criteria for 4-bromophenyl phenyl ether because of the lack of sufficient data.

Butyl Benzyl Phthalate

Butyl benzyl phthalate is not known to be toxic to humans. A Russian report suggests that certain phthalate esters such as dibutyl phthalate and butyl benzyl phthalate have caused polyneuritis in industrial workers (Milkov *et al*. 1973, as reported in Doull *et al*. 1980); however, similar observations have not been reported in this country (Doull *et al*. 1980).

Phthalate esters are considered as having a low order of acute toxicity in animal studies. The only reported LD50 value for butyl benzyl phthalate is that reported for the mouse by intraperitoneal injection of 3.16 g/kg (Autian 1973, as reported in USEPA 1980). When single doses of butyl benzyl phthalate were administered to groups of rats either orally (1.8 g/kg) and intraperitoneally (4 g/kg), the animals died after four to eight days (Mallette and Von Hamm 1952, as reported in USEPA 1980). Histopathological studies demonstrated toxic splenitis and degeneration of central nervous system tissue with congestive encephalopathy. Myelin and glial proliferation were also reported. Most phthalate esters are precluded from presenting an acute toxic response by inhalation because of their low volatility (USEPA 1980). No chronic or subchronic toxicity data are available for butyl benzyl phthalate.

The LC50 values for butyl benzyl phthalate have been reported for three fish and one invertebrate species ranging from 1,700 µg/liter for the bluegill to 92,300 µg/liter for *Daphnia magna* (USEPA 1980). For two saltwater species, acute toxic values were reported for the mysid shrimp of

900 and 9,630 µg/liter and for the sheepshead minnow of 3,000 and 445,000 µg/liter. The lower values were obtained using a solvent in which the chemical is more soluble and thus presumably more available (USEPA 1980). Chronic studies have been conducted with two freshwater species. In a life-cycle study of Daphnia magna, effects were observed at 440 µg/liter, and in an early life stage test with the fathead minnow, an acute value of 220 was reported (USEPA 1980). EC50 values for freshwater algae showed wide variation in toxicity, with a range of 110 to 1,000,000 µg/liter (USEPA 1980).

EPA has not yet established ambient water quality criteria for butyl benzyl phthalate because of the lack of sufficient data.

2-Chloronaphthalene

Little toxicity information is available on 2-chloronaphthalene. As a class, chlorinated naphthalenes have been associated with the development of chloracne in humans, and, in some cases, fatal liver disease. It has been demonstrated, however, that monochloronaphthalenes did not produce chloracne in a test with dermal application to the inner side of the rabbit ear (Adams et al. 1941, as reported in Clayton and Clayton 1981).

EPA has not yet established ambient water quality criteria for 2-chloronaphthalene because of the lack of sufficient information.

4-Chlorophenyl Phenyl Ether

The only available toxicity data on 4-chlorophenyl phenyl ether is that reported for experimental animals by Hake and Rowe (1963, as reported in USEPA 1980) for various chlorinated phenyl ethers. For an unspecified monochloro phenyl ether, the lethal oral dose within four days of dosing in guinea pigs was given as 700 mg/kg. The "survival dose" was reported as 200 mg/kg. Within 30 days of the single oral dosing, the "lethal" and "survival" doses were reported to be 600 and 100 mg/kg, respectively. Hake and Rowe also reported that 19 daily oral doses of 100 mg/kg/day of a monochloro phenyl ether over a 29-day period produced no effects in rabbits. Because of inadequate experimental detail, these results are difficult to interpret (USEPA 1980).

No aquatic toxicity information is available for 4-chlorophenyl phenyl ether. Available data for haloethers indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 360 and 122 µg/liter , respectively (USEPA 1980).

EPA has not yet established ambient water quality criteria for 4-chlorophenyl phenyl ether because of the lack of sufficient data.

Chrysene

No information is available on the toxicity of chrysene in humans. In one experimental study, Swiss mice painted three times weekly with a 1% solution of chrysene in acetone developed skin cancer. This effect was not repeatable in other studies however, and the carcinogenicity of chrysene has not been clearly determined (IARC 1973).

No information is available on the toxic effects of chrysene on aquatic organisms. However, chrysene was reported to bioconcentrate 8.2 times in clams over 24 hours (USEPA 1980).

EPA has not yet established ambient water quality criteria for chrysene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include chrysene, through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 2.8 ng/liter, corresponding to a lifetime incremental cancer risk of 10-6, was recommended.

Dibenzo(a,h)anthracene

Dibenzo(a,h)anthracene (DB(a,h)A) is a member of the class of polynuclear aromatic hydrocarbons (PAHs) and was the first pure chemical compound shown to be carcinogenic (IARC 1973). When DB(a,h)A was administered to mice as an aqueous-oil emulsion in the place of drinking water at an average dose of 0.76-0.85 mg/day, all surviving mice at 200 days had respiratory tract tumors and 12 of 13 females had mammary carcinomas (Snell and Stewart 1962, as reported in IARC 1973). A single dose of 1.5 mg DB(a,h)A in polyethylene glycol produced forestomach papillomas in 2 of 42 male mice within 30 weeks (Berenblum and Haran 1955, as reported in IARC 1973).

Many studies have reported the induction of skin tumors from application of DB(a,h)A. Thrice weekly paintings with solutions containing 0.001, 0.01, and 0.1% DB(a,h)A produced skin carcinomas in one of 30 mice, 43 of 50 mice, and 39 of 40 mice, respectively (Van Duuren *et al*. 1967, as reported in IARC 1973). In a subcutaneous injection study, single injections of DB(a,h)A at doses ranging from 0.00019 to 8 mg produced incidences of local sarcomas of 2.5% to 100% (IARC 1973). In *in vitro* hamster embryo cell transformation studies, DB(a,h)A at concentrations of 2.5 to 10 µg/ml did not produce any compound-related increase in cell transformations; however, the 5,6-epoxide of DB(a,h)A did produce a dose-related increase in cell transformations (Grover *et al*. 1971, Huberman *et al*. 1972, as reported in USEPA 1980). High doses of DB(a,h)A have been reported to produce an immunosuppressive effect in mice (Malmgren *et al*. 1952, as reported in USEPA 1980).

No human case studies or epidemiological studies have been conducted which establish DB(a,h)A as a human carcinogen. Indirect evidence for the compound's carcinogenicity comes from air pollution studies which indicate an excess of lung cancer mortality among workers exposed to high concentrations

of PAH-containing material such as coal gas, tars, soot, and coke-oven emissions (IARC 1973, USEPA 1980).

No data are available on the aquatic toxicity of DB(a,h)A.

EPA has not yet established ambient water quality criteria for DB(a,h)A because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include DB(a,h)A, through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at present, a level of 2.8 ng/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended.

Di-n-butyl Phthalate

Occupational exposure to di-n-butyl phthalate has been associated with toxic polyneuritis (Milkov et al. 1973, as reported in USEPA 1980). Abnormal encephalographic responses were observed in three workers exposed to 0.12-0.15 mg/cu m (Men'shikova 1971, as reported in USEPA 1980).

In animal studies, testicular atrophy has been reported in rats administered 2,000 mg/kg orally "for a period of time." Significant weight loss of the testes was observed after 14 days. Zinc metabolism was also affected as evidenced by increased zinc levels in the urine (Carter et al. 1977, as reported in USEPA 1980). In an inhalation experiment, a dose-related increase in gamma globulin was observed in rats exposed to concentrations ranging from 0.098-0.98 mg/cu m, continuously for 93 days (Men'shikova 1971, as reported in USEPA 1980). In a feeding study, 50% of rats fed 1.25% di-n-butyl phthalate in the diet died in the first week of administration following one year exposure to levels up to 0.25% (Smith 1953, as reported in USEPA 1980). A dermal LD50 for rabbits has been reported as 20 ml/kg (Autian 1973, as reported in USEPA 1980). Dose-related skeletal abnormalities and reduced fetal weight were reported in the offspring of rats administered 1/10, 1/5, and 1/3 the LD50 value of 3.05 ml/kg di-n-butyl phthalate, by intraperitoneal injection during days 5, 10, and 15 of gestation (Singh et al. 1972, as reported in USEPA 1980).

The acute toxicity of di-n-butyl phthalate on fresh water aquatic organisms is reflected by 96-hour LC50 values for scud (2.1 mg/liter), midge (4.0 mg/liter), rainbow trout (6.47 mg/liter), fathead minnow (1.3 mg/liter), and bluegill (0.73-1.2 mg/liter). Crayfish were less sensitive, having an LC50 greater than 10 mg/liter. No information is available on the toxic effects in saltwater organisms or the chronic effects in any aquatic organisms. Di-n-butyl phthalate was reported to be extremely toxic to plant life, causing toxic effects in algae in concentrations as low as 3.4 µg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 34 mg/liter for the protection of human health from the toxic properties of di-n-butyl phthalate ingested through contaminated water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for di-n-butyl phthalate.

1,2-Dichlorobenzene

The acute toxic effects on humans following exposure to 1,2-dichlorobenzene include eye and upper respiratory tract irritation when inhaled (Dupont 1938, as reported in USEPA 1980), and skin irritation and burning when absorbed through the skin (Reidel 1941, as reported in USEPA 1980). Chronic exposure to 1,2-dichlorobenzene produced weakness, fatigue, nausea, headaches, peripheral lymphadenopathy, chronic lymphoid leukemia, acute myeloblastic leukemia, acute hemolytic anemia, leukocytosis, and bone marrow hyperplasia under different conditions of exposure (Girard et al. 1969 and Gadrat et al. 1962, as reported in USEPA 1980). Chronic skin contact resulted in contact eczematoid dermatitis, erythema, and edema (Dowing 1939, as reported in U.S. EPA 1980).

Acute toxic effects were observed in rats exposed to a 4,800 mg/cu m concentration of 1,2-dichlorobenzene (Cameron et al. 1937 in USEPA 1980). In this study, nasal and ocular irritation, drowsiness, massive liver necrosis, coma, and death occurred after exposure by inhalation for 11-50 hours. Rats (2,138 mg/kg), mice (2,000 mg/kg), rabbits (1,875 mg/kg), and guinea pigs (3,375 mg/kg) exposed to different levels of 1,2-dichlorobenzene in single doses by stomach tube developed acute poisoning manifestations including hyperemia of the mucous membranes, adynamia, ataxia, paraparesis, paraplegia, dyspnea, and death due to central respiratory paralysis (Varshavskaya 1967, as reported in USEPA 1980). On necropsy, enlarged necrotic livers, and brain, stomach, and kidney edema were observed in these animals. Chronic exposure to 1,2-dichlorobenzene at levels of 0.0010.1 mg/kg/day predominantly affected the hematopoietic system after 5 months (preliminary report, Varshavskaya 1967, as reported in USEPA 1980). In this report, rats fed 0.1 mg/kg developed disturbances of higher cortical function in the central nervous system, inhibition of erythropoiesis, thrombocytosis, neutropenia, and inhibition of bone marrow mitotic activity. Hepatic prophyria was induced in rats fed 455 mg/kg 1,2-dichlorobenzene by stomach tube daily for 15 days. Severe liver damage with intense necrosis and fatty changes were observed (Rimington and Ziegler 1963, as reported in USEPA 1980). 1,2-Dichlorobenzene was not mutagenic in the Salmonella typhimurium assay (Anderson et al. 1972, as reported in USEPA 1980). No information is available on the teratogenicity of 1,2-dichlorobenzene, and the carcinogenic potential of 1,2-dichlorobenzene has not been established (USEPA 1980).

The acute toxicity of 1,2-dichlorobenzene to freshwater aquatic organisms has been demonstrated in the bluegill and cladoceran with 96-hour LC50 values of 5.59-27 and 2.44 mg/liter, respectively. Chronic toxicity was observed in fathead minnows in concentrations ranging from 1.6 to 2.5 mg/liter. Plant toxicity was demonstrated in freshwater algae at 91.6-98 mg/liter. Saltwater organisms affected by 1,2-dichlorobenzene include tidewater silverside, sheepshead minnows, and mysid shrimp. LD50 values are 7.3, 9.66, and 1.97 mg/liter, respectively. Emergence from parasitized oysters was observed in the polychaete worm at concentrations of 100 mg/liter. Saltwater algae were affected at 44.1 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 400 µg/liter for the protection of human health from the toxic properties of total dichlorobenzenes including 1,2-dichlorobenzene ingested through water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for 1,2-dichlorobenzene.

1,3-Dichlorobenzene

No information is available on the human or animal effects which result from exposure to 1,3-dichlorobenzene.

The acute toxicity of 1,3-dichlorobenzene to aquatic organisms is reflected in the 96-hour LC50 values of 5.02 mg/liter for the bluegill, 7.79 mg/liter for the fathead minnow, 28.1 mg/liter for the cladoceran, 2.85 mg/liter for the mysid shrimp, and 7.77 mg/liter for the sheepshead minnow. Chronic toxicity has been observed in the fathead minnow in concentrations ranging from 1 to 2.27 mg/liter. Algae are affected by concentrations ranging from 46.9 to 179 mg/liter, depending on the species (USEPA 1980).

EPA has established an ambient water quality criterion of 400 µg/liter for the protection of human health from the toxic properties of total dichlorobenzenes, including 1,3-dichlorobenzene, through ingestion of contaminated water and aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for 1,3-dichlorobenzene.

1,4-Dichlorobenzene

1,4-Dichlorobenzene is toxic to mammals, birds, and aquatic organisms and imparts an offensive taste and odor to water. Human exposure to 1,4-dichlorobenzene is associated with leukemia and other blood dyscrasias, liver necrosis, and eye irritation. Symptoms of exposure include headaches, weakness, nausea, jaundice, and anemia (Sumers et al. 1952, Cotter 1953, Perrin 1941, Hallowell 1959, Campbell and Davidson 1970, Nalbandian and Pierce 1965, as reported in USEPA 1980).

Acute inhalation studies in rabbits exposed to 100 gm/cu m for 30 minutes daily caused central nervous system depression, and occular and nasal irritation; rats similarly exposed developed irritation and narcosis while guinea pigs exposed under the same conditions developed irritation, central nervous system depression and deaths (Domenjoz 1946, as reported in USEPA 1980). Rats and guinea pigs administered 500 mg/kg by stomach tube developed centrilobular hepatic necrosis and marked cloudy swelling of renal tubular epithelium; no effects were observed at 10 and 100 mg/kg (Hollingsworth et al. 1956, as reported in USEPA 1980).

In subchronic studies, rabbits subjected by inhalation to 1,4-dichlorobenzene at approximately 800 ppm for 8-hour periods, 5 days per

week for as long as 12 weeks, developed tremors, weakness, nystagmus, and reversible nonspecific eye changes (Pike 1944, as reported in NAS 1977). Rabbits fed 1,000 mg/kg for 5 days per week developed similar toxicity symptoms after several months (Pike 1944, as reported in NAS 1977).

Rats and guinea pigs exposed at 2,050 mg/cu m, 5 hours per day, 5 days per week for 6 months displayed growth depression (guinea pigs); liver pathology including cloudy swelling, fatty degeneration, focal necrosis, and cirrhosis; and increased liver and kidney weights (rats only) (Hollingsworth et al. 1956, as reported in USEPA 1980).

The acute toxicity of 1,4-dichlorobenzene on aquatic organisms is reflected in 96-hour LC50 values of 11 mg/liter for the cladoceran, 13 mg/liter for the midge, 1.12 mg/liter for the rainbow trout, 4 mg/liter for the fathead minnow, 4.28 mg/liter for the bluegill, 1.99 mg/liter for the mysid shrimp, and 7.4 mg/liter for the sheepshead minnow. Chronic toxicity was observed in the fathead minnow exposed to concentrations ranging from 5.6 to 1.04 mg/liter in the embryo-larval test. Algae were affected by concentrations ranging from 54.8-98.1 mg/liter, depending on the species (USEPA 1980).

EPA has established an ambient water quality criterion of 400 µg/liter for the protection of human health from the toxic properties of total dichlorobenzenes including 1,4-dichlorobenzene through ingestion of contaminated water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for 1,4-dichlorobenzene.

3,3'-Dichlorobenzidine

3,3'-Dichlorobenzidine has not been shown to be toxic in humans. Three epidemiological studies of industrial exposure to 3,3'-dichlorobenzidine have been conducted by Gerarde and Gerarde (1974) in the United States, by MacIntyre (1975) and Gadian (1975) in Great Britain, and by Akiyama (1970) in Japan (all as reported in USEPA 1980). The studies do not provide any evidence that 3,3'-dichlorobenzidine induces bladder cancer, the characteristic lesion induced by carcinogenic aromatic amines such as benzidine used in the dye and pigment industry. The evidence is not, however, conclusive since the populations studied have been small, tumors may not have appeared at the time of the study because of a long latent period, and the focus of the studies has been solely on bladder cancer as the lesion of interest (USEPA 1980).

In rats, the oral LD50 for 3,3'-dichlorobenzidine is 488 for females and 676 for males (Gaines and Nelson 1977, as reported in USEPA 1980). Pliss (1959, as reported in USEPA 1980) injected rats subcutaneously with 120 mg of 3,3'-dichlorobenzidine and observed a state of excitation and short-lived convulsions. Exposure of rats to a concentrated atmospheric dust of 3,3'-dichlorobenzidine dihydrochloride for 14 days results in slight to moderate pulmonary congestion; the actual concentration of the compound was not measured (Gerarde and Gerarde 1974, as reported in USEPA 1980). However

HCl may have caused or contributed to the observed pulmonary effects. Freeman et al. (1973, as reported in USEPA 1980) reported that 3,3'-dichlorobenzidine at concentrations of 5 ppm or greater was cytotoxic to embryonic rat cells in culture.

3,3'-Dichlorobenzidine has not been shown to be teratogenic. However, the compound has been demonstrated to increase significantly the incidence of leukemia in the offspring of pregnant female mice given doses of 8 to 10 mg by subcutaneous injection during the last week of gestation (Golub et al. 1974, as reported in USEPA 1980). When tested for mutagenicity in the Ames assay using several strains of bacteria, 3,3'-dichlorobenzidine caused frameshift and base-pair substitution mutations (USEPA 1980). 3,3'-Dichlorobenzidine is carcinogenic in experimental animals. Male and female rats fed the chemical at 1,000 mg/kg diet for up to 488 days developed significantly increased incidences ($p < 0.05$) of mammary adenocarcinomas, granulocytic leukemia (males only), and Zymbal's gland carcinomas (males only) (Stula et al. 1975, as reported in USEPA 1980). 3,3'-Dichlorobenzidine added to the feed of rats for 12 months (total dose of 4.63 g/rat) resulted in a wide variety of tumors in 22 of 29 surviving animals (Pliss 1959, as reported in USEPA 1980). Papillary transitional cell carcinomas of the urinary bladder and hepatic carcinomas were induced in female beagle dogs given oral doses of 100 mg of 3,3'-dichlorobenzidine, three times per week for six weeks, then five times per week for up to 7.1 years. Tumors of these types were not found in controls (Stula et al. 1978, as reported in USEPA 1980).

No aquatic toxicity data are available for 3,3'-dichlorobenzidine.

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to 3,3'-dichlorobenzidine through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, a level of 0.010 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for 3,3'-dichlorobenzidine.

Diethyl Phthalate

Diethyl phthalate produces irritation of the mucous membranes of the nasal passages and the upper respiratory tract in humans when vaporized by heat (USEPA 1980).

In animals, diethyl phthalate produced small but significant decreases in the growth rate of rats exposed for 2 years to 5% diethyl phthalate in the diet, but no other effects were reported at dosages of 0.5 and 2.5%. No adverse effects were reported in dogs fed levels up to 2.5% for 1 year (Food Research Laboratories, Inc. 1955, as reported in USEPA 1980). An oral LD50 for rabbits has been reported as 1.0 g/kg (Autian 1973, as reported in USEPA 1980). Dose-related skeletal abnormalities and reduced fetal weight were reported in rats administered 1/10, 1/5, and 1/3 of the LD50 value of 5.06 ml/kg diethyl phthalate, by intraperitoneal injection on days 5, 10, and 15 of gestation (Singh et al. 1972, as reported in USEPA 1980).

The acute toxicity of diethyl phthalate on freshwater aquatic organisms is reflected by 96-hour LC50 values of 52.1 mg/liter for Daphnia magna and 98.2 mg/liter for bluegill. The saltwater species, mysid shrimp, gave a 96-hour LC50 value of 7.59 mg/liter. Sheepshead minnows were also affected at an LC50 of 29.6 mg/liter (USEPA 1980).

No information is available on the chronic effects of diethyl phthalate on aquatic organisms. Diethyl phthalate is toxic to algae in concentrations ranging from 3.0 to 90.3 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 350 mg/liter for the protection of human health from the toxic properties of diethyl phthalate ingested through water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for diethyl phthalate.

Dimethyl Phthalate

Few toxic effects have been observed in humans exposed to dimethyl phthalate (USEPA 1980). In animal studies, rats fed for 2 years at levels of 2% and 8% in the diet developed minor growth retardation. At 8%, some indication of nephritic damage was observed. A 90-day LD50 value of 4 ml/kg was obtained when dimethyl phthalate was applied to the skin of rabbits (Draize et al. 1948, as reported in USEPA 1980). Oral LD50s have been reported for the mouse (7.2 g/kg), rat (2.4 g/kg), and guinea pig (2-4 g/kg) (Autian 1973, as reported in USEPA 1980). Dose-related skeletal abnormalities and reduced fetal weight were reported in the offspring of rats administered 1/10, 1/5, and 1/3 the LD50 value of 3.38 ml/kg by intraperitoneal injection during days 5, 10, and 15 of gestation (Singh et al. 1972, as reported in USEPA 1980).

The acute toxicity of dimethyl phthalate on freshwater aquatic organisms is reflected by 96-hour LC50 values for Daphnia magna (33 mg/liter) and bluegill (49.5 mg/liter). The saltwater species, mysid shrimp, gave a 96-hour LC50 value of 73.7 mg/liter, and sheepshead minnow showed an LC50 of 58 mg/liter. Significant decreases in survival were reported in the larvae of grass shrimp exposed to 100 mg/liter during active larvae development. No information on the chronic effects of dimethyl phthalate on aquatic organisms is available. The toxic effect of dimethyl phthalate on plant life occurs at levels similar to those causing toxic effects on fishlife. Toxicity occurred in a wide variety of algae in concentrations ranging from 26.1 to 185 mg/liter (USEPA 1980).

EPA has established a water quality criterion of 313 mg/liter for the protection of human health from the toxic properties of dimethyl phthalate ingested through contaminated water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for dimethyl phthalate.

2,4-Dinitrotoluene

The acute toxic effects of 2,4-dinitrotoluene in humans include methemoglobinemia followed by cyanosis (USEPA 1980). The symptoms of methemoglobinemia following absorption by inhalation, ingestion, or dermal absorption of 2,4-dinitrotoluene include headache, vertigo, fatigue, nausea, dyspnea, tremor, dizziness, loss of weight and appetite, paralysis, chest pain, shortness of breath, and palpitations. Many of these symptoms were observed in workers exposed to 2,4-dinitrotoluene (USEPA 1980). Acute oral LD50s for 2,4-dinitrotoluene are 268 and 1,625 mg/kg for rats and mice, respectively (USEPA 1980).

The 13 week subacute toxicity of 2,4-dinitrotoluene by oral administration was studied in dogs, rats, and mice (Ellis et al. 1976 in USEPA 1980). The dogs were fed daily doses of 1, 5, and 25 mg/kg, and the rats and mice were fed dietary concentrations of 0.07, 0.2, and 0.7%. Toxic effects in dogs and rats included inhibited muscle coordination in the hind legs, decreased appetite, and weight loss. The latter two symptoms were also observed in mice. Other symptoms observed in the test species were methemoglobinemia, anemia with reticulocytosis, lesions in the spleen and liver, demyelination in the brain, and aspermatogenesis. The highest dose was lethal to some animals in all three species, and the lowest dose produced no toxic effect.

Chronic exposure to 2,4-dinitrotoluene produced benign tumors in rats (National Cancer Institute 1978, as reported in USEPA 1980). Rats were fed average doses of 17.6 and 440 mg/kg/day, and mice were fed doses of 16.3 and 81.5 mg/kg/day for 78 weeks. Treated male rats at both dose levels developed a significantly higher incidence of fibroma of the skin and subcutaneous tissue (a benign tumor) than their controls. An increased incidence of mammary gland fibroadenoma was observed in the high dose female rats. Certain rare neoplasms occurred at low incidence in high dose rats, but the study concluded that these tumors were not compound related. No tumors were observed at a statistically significant incidence in mice. Data available in the literature on the mutagenicity of 2,4-dinitrotoluene are "limited and rather confusing" (USEPA 1980).

The 48-hour EC50 value for 2,4-dinitrotoluene to *Daphnia magna* is 35 mg/liter and is 31 mg/liter for the fathead minnow. No data are available that describe the toxic effect of 2,4-dinitrotoluene to aquatic plants (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to 2,4-dinitrotoluene. Since the zero level may not be attainable at the present time, a level of 0.11 µg/liter, corresponding to an estimated lifetime incremental cancer risk of 10-6, was recommended.

EPA has not yet established an aquatic life water quality criterion for 2,4-dinitrotoluene.

2,6-Dinitrotoluene

The only toxicity information available on 2,6-dinitrotoluene are oral LD50 values for the rat of 177 mg/kg and for the mouse of 1,000 mg/kg (USEPA 1980).

EPA has not yet established ambient water quality criteria for 2,6-dinitrotoluene because of lack of sufficient data.

Di-n-octyl Phthalate

Di-n-octyl phthalate is not known to be toxic to humans. A Russian report suggests that certain phthalate esters such as dibutyl phthalate and butyl benzyl phthalate have caused polyneuritis in industrial workers (Milkov et al. 1973, as reported in Doull et al. 1980); however, similar observations have not been reported in this country (Doull et al. 1980).

Phthalate esters are considered as having a relatively low order of acute toxicity. A reported oral LD50 for di-n-octyl phthalate in the mouse is 13 g/kg. LD50 values for the rat by intraperitoneal administration and the guinea pig by dermal exposure are reported to be 50 ml/kg and 5 ml/kg, respectively (Autian 1973, as reported in USEPA 1980). Most phthalate esters are precluded from presenting an acute toxic response by inhalation because of their low volatility (USEPA 1980). In a teratogenicity study, Singh et al. (1975, as reported in USEPA 1980) administered eight phthalic acid esters, including dioctyl phthalate, to rats by intraperitoneal injection on days 5, 10, and 15 of gestation. Dioctyl phthalate was administered at doses of 5 and 10 ml/kg. All of the esters were reported to produce dose-related gross or skeletal abnormalities and reduced fetal weight, although dioctyl phthalate had the least adverse effects on embryo-fetal development. No other chronic or subchronic data are available for di-n-octyl phthalate.

Little aquatic toxicity data are available for di-n-octyl phthalate. In a 26-day early life stage study with rainbow trout, the LC50 was reported to be 139,500 µg/liter. In 7 to 8-day early life stage studies with redear sunfish, channel catfish, and the largemouth bass, LC50 values of 6,180, 690, and 32,900 µg/liter were reported (USEPA 1980). Available data for the general class of phthalate esters indicate that acute and chronic toxicity to freshwater aquatic life can occur at concentrations as low as 940 and 3 µg/liter, respectively.

EPA has not yet established ambient water quality criteria for di-n-octyl phthalate because of the lack of sufficient data.

1,2-Diphenylhydrazine

No information on the toxicity of 1,2-diphenylhydrazine to humans is available.

The oral LD50 of 1,2-diphenylhydrazine in rats is 301 mg/kg (Mason Research Institute Report 1971, as reported in NAS 1977). Studies in rats and

mice have shown that 1,2-diphenylhydrazine produces both benign and malignant tumors when administered subcutaneously or orally. Pliss (1974, as reported in NAS 1977 and USEPA) administered 1,2-diphenylhydrazine by subcutaneous injection (40 mg/week/rat and 5 mg/week/mouse) and in the diet (30 mg/mouse, five times per week) for 588 days, and observed an increased incidence of rhabdomyosarcomas, pulmonary adenomas, leukemia, and liver tumors in the mouse, and tumors of the uterus, mammary glands, Zymbal's gland, liver, and spleen, and lymphoid leukemias in rats. In a study by the National Cancer Institute (NCI 1978, as reported in USEPA 1980), mice were fed diets containing 1,2-diphenylhydrazine at concentrations of 0.008 and 0.04 percent for males and 0.004 and 0.04 percent for females for 78 weeks. Male rats were fed diets containing 0.008 and 0.03 percent 1,2-diphenylhydrazine and females 0.004 and 0.01 percent. In rats, 1,2-diphenylhydrazine produced a significantly increased incidence of hepatocellular carcinomas or neoplastic nodules in males at both dose levels and females at the high dose; Zymbal's gland squamous cell carcinomas in high dose males; adrenal tumors in high dose males; and mammary carcinomas in high dose females. 1,2-Diphenylhydrazine produced hepatocellular carcinomas in high dose female mice, but not in male mice.

In a 48-hour EC50 test with Daphnia magna and a 96-hour LC50 test with the bluegill, toxic values of 4,100 µg/liter and 270 µg/liter, respectively, were reported (USEPA 1980). No data are available for any saltwater species.

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to 1,2-diphenylhydrazine through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, a level of 42 ng/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for 1,2-diphenylhydrazine.

Fluoranthene

No information is available on the toxicity of fluoranthene in humans. In acute toxicity studies in animals, this compound was found to present a relatively low degree of toxicity (USEPA 1980). Fluoranthene does not appear to be a direct acting carcinogen or mutagen, but it is a potent cocarcinogen in animal studies (USEPA 1980). In skin painting studies, fluoranthene increased the tumor incidence and reduced the time-to-tumor of mice treated with benzo(a)pyrene (Van Duuren and Goldschmidt 1976, as reported in USEPA 1980).

Fluoranthene is acutely toxic to various freshwater and marine organisms. Bluegill were found to have a 96-hour LC50 value of 3.98 mg/liter. Daphnia magna were more resistant, with a 48-hour LC50 of 325 mg/liter. Fluoranthene has a 96-hour LC50 value of 40 µg/liter and chronic value of 16 µg/liter in mysid shrimp. No data are available on the chronic toxicity of fluoranthene on freshwater organisms (USEPA 1980).

EPA has established an ambient water quality criterion of 42 µg/liter for the protection of human health from the toxic properties of fluoranthene ingested through water and contaminated organisms.

EPA has not yet established an aquatic life water quality criterion for fluoranthene.

Fluorene

No information is available on the toxic effects of fluorene on humans, and very little information is available on the effects on animals. The carcinogenic potential of fluorene was tested by skin painting and subcutaneous administration in mice and by feeding and subcutaneous administration in rats. Carcinogenicity was not established in any of these tests (dose, duration unspecified; Hueper and Conway 1964).

Only one study is available on the toxic effects of fluorene on aquatic life. A crude oil extract of fluorene was used in a 96-hour LC50 test on a polychaete worm. The LC50 value determined in this test is 1.0 mg/liter (USEPA 1980).

EPA has not yet established ambient water quality criteria for fluorene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include fluorene, through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 2.8 ng/liter, corresponding to a lifetime incremental cancer risk of 10-6, was recommended.

Hexachlorobenzene

An outbreak of hexachlorobenzene-induced porphyria cutanea tarda occurred in Turkey between 1955 and 1959 as a result of human consumption of seed grain that had been treated with hexachlorobenzene (IARC 1979, USEPA 1980). More than 3,000 people were affected by the condition, characterized by blistering and epidermolysis of the exposed skin, loss of hair, and skin atrophy. A mortality rate of 14% was reported within several years, and among breast-fed infants of mothers whose milk contained hexachlorobenzene, the infant mortality rate was greater than 95% (Cam 1960, Peters 1976, as reported in IARC 1979 and USEPA 1980). There was no evidence of porphyria in Louisiana residents living near an hexachlorobenzene manufacturing plant and whose average plasma hexachlorobenzene levels were 3.6 µg/liter; however, plasma coproporphyrin levels were abnormally high (Burns and Miller 1975, as reported in IARC 1979 and USEPA 1980).

In experimental animals, the acute toxicity of hexachlorobenzene is relatively low. The oral LD50 in rats varies from 3,500 to 10,000 mg/kg (Booth and McDowell 1975, as reported in IARC 1979). With prolonged moderate exposure, hexachlorobenzene exhibits a wide range of biological effects. In rats given 500 mg/kg hexachlorobenzene in their diet for 4 months,

hepatocellular hypertrophy and necrosis, spleen enlargement, prophyria, and death were observed (Kimbrough and Linder 1974, as reported in IARC 1979). Immunosuppression was observed in mice fed 167 mg/kg diet for 6 weeks (Loose et al. 1977, as reported in IARC 1979). Pigs were administered hexachlorobenzene in the diet at doses of 0.05-50 mg/kg/day for 90 days. Porphyria and death occurred at the highest dose level, histopathological changes of the liver at 5 mg/kg/day, and increased excretion of coproporphyrin in the groups receiving 0.5 and 5 mg/kg/day (den Tonkelaar et al. 1978, as reported in IARC 1979). Porphyrinuria has also been observed in rabbits, Japanese quail, guinea pigs, and mice (USEPA 1980). Hexachlorobenzene is fetotoxic and produces some teratogenic effects. Mice administered 100 mg/kg/day orally on gestation days 7-16 gave birth to offspring with an increased incidence of cleft palates and kidney malformations (Courtney et al. 1976, as reported in IARC 1979 and USEPA 1980). In a four-generation study with rats treated with hexachlorobenzene at doses ranging from 0 to 640 mg/kg diet, the neonatal survival rate and neonatal body weight were reduced at the 80 mg/kg level; birth weights were reduced at the 160 mg/kg level. No gross abnormalities were found in this study (Grant et al. 1977, as reported in USEPA 1980).

Hexachlorobenzene was shown to be carcinogenic in two studies. Groups of male and female rats were fed diets containing 0, 50, 100 or 200 mg hexachlorobenzene/kg diet for 120 days, and 300 mg/kg diet for 15 weeks. An increased incidence of liver cell tumors was observed in the 100, 200 and 300 mg/kg diet groups (Cabral et al. 1978 and 1979, as reported in IARC 1979). In a lifetime study with Syrian golden hamsters given dietary concentrations of 0, 50, 100, and 200 mg hexachlorobenzene/kg diet (equivalent to 0, 4, 8, and 16 mg/kg bw/day), a significantly increased incidence of hepatomas was reported at all doses tested. Liver hemangioendotheliomas and alveolar thyroid adenomas were found at the high dose level (Cabral et al. 1977, as reported in IARC 1979).

Few data are available on the toxicity of hexachlorobenzene to aquatic organisms. In 10 to 15 day studies with the largemouth bass, no effects were observed at concentrations of 26 and 10 µg/liter (Laska et al. 1978, as reported in USEPA 1980). In a study of saltwater protozoa, a 10-day exposure to 1 µg/liter of hexachlorobenzene caused decreased growth (Geike and Prasher 1976, as reported in USEPA 1980). Ninety-six hour exposure of pink shrimp to 25 µg/liter resulted in 33 percent mortality (Parrish et al. 1974, as reported in USEPA 1980).

EPA has established an ambient water quality criterion of 0.0072 µg/liter for the protection of human health from the toxic properties of hexachlorobenzene ingested through contaminated water and aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for hexachlorobenzene.

Hexachlorobutadiene

The acute oral toxicity of hexachlorobutadiene is moderate to high. Schwetz et al. (1977, as reported in USEPA 1980) reported oral LD50 values

of 200-400 mg/kg for female rats, 580 mg/kg for male rats, and 64 mg/kg for weanling rats. Oral LD50 values reported by Gradiski et al. (1975, as reported in USEPA 1980) for male rats and mice were 250 and 80 mg/kg; hepatic and renal disorders and effects on the central nervous system were noted.

In chronic and subchronic studies of hexachlorobutadiene, the kidney appears to be the most sensitive organ. In a 30-day feeding study, renal tubular epithelial degeneration, necrosis, and an increase in the kidney-to-body weight ratio occurred in female rats receiving 30, 65, or 100 mg/kg/day hexachlorobutadiene in their diets, but not at 3 mg/kg/day (Kociba et al. 1971, as reported in USEPA 1980). In a two-year chronic study with rats given 0, 0.2, 2, or 20 mg/kg/day hexachlorobutadiene in their diet, a significant increase in renal tubular adenomas and carcinomas was observed in both male and female rats at the 20 mg/kg/day level (Kociba et al. 1977, as reported in USEPA 1980). Slight renal tubular epithelial hyperplasia was noted at the 2 mg/kg/day level. A statistically significant increase in urinary coproporphyrin was observed in male rats ingesting 20 mg/kg/day hexachlorobutadiene and in females ingesting 2 mg/kg/day. Schwetz et al. (1977, as reported in USEPA 1980) reported renal tubular dilation, degeneration, and regeneration in kidneys from male and female rats given hexachlorobutadiene in their diet at a dose level of 20 mg/kg/day for 143 days; a mottled cortex was noted at the 2 mg/kg/day level. Kidney damage has also been induced by inhalation of hexachlorobutadiene; injury to the tubular epithelium was reported after 15 daily six-hour exposures to 25 ppm hexachlorobutadiene (Gage 1970, as reported in USEPA 1980). Evidence for the teratogenic potential of hexachlorobutadiene is inconclusive. Poteryaeva (1966, as reported in USEPA 1980) reported increased neonatal mortality, decreased birthweight, kidney damage, and degenerative changes in red blood cells in offspring of female rats given single subcutaneous injections of 20 mg/kg hexachlorobutadiene before breeding. Schwetz et al. (1977, as reported in USEPA 1980) observed no effects in offspring of male and female rats given hexachlorobutadiene in the diet at dose levels of 0.2 and 2.0 mg/kg/day for 90 days before mating, during mating, and throughout gestation and lactation; at the 20 mg/kg/day level, a slight decrease in weanling body weight was the only observed effect.

Acute toxicity data have been reported for four species of freshwater fish, with LC50 values ranging from 90 to 326 µg/liter (USEPA 1980). In static tests conducted with saltwater mysid shrimp, grass shrimp, pinfish, and sheepshead minnow, the 96-hour LC50 values were 59, 32, 399, and 557 µg/liter, respectively. In an embryo-larval test with fathead minnow, a chronic toxicity value of 9.3 µg/liter was reported (USEPA 1980).

EPA has established a water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to hexachlorobutadiene through ingestion of contaminated water and contaminated aquatic organisms. Since the zero level may not be attainable at the present time, a criterion of 0.45 µg/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended.

EPA has not yet established aquatic life ambient water quality criteria for hexachlorobutadiene.

Hexachloroethane

The acute toxic effects of hexachloroethane in humans include extensive eye irritation including inability to close the eyelid, tearing of the eyes, inflammation of the delicate membrane lining of the eye, and visual intolerance to light (NIOSH 1978, as reported in USEPA 1980).

Liver degeneration and tubular nephrosis of the kidney were observed in rabbits administered daily oral doses of 320 or 1,000 mg/kg for 12 days (Weeks et al. 1979, as reported in USEPA 1980). Inhalation exposure of dogs, guinea pigs, and rats to 260 ppm for 6 hours/day, 5 days per week, for 6 weeks produced central nervous system toxicity in dogs and rats, and increased liver size in guinea pigs and rats (Weeks et al. 1978, as reported in USEPA 1980). Rats administered 500 mg/kg/day orally from day 6 through 16 of gestation gave birth to a significantly lower number of live fetuses (Weeks et al. 1979, as reported in USEPA 1980). Liver cancer was produced in mice administered 590 mg/kg/day hexachloroethane by stomach tube for 78 weeks (National Cancer Institute 1978, as reported in USEPA 1980).

The toxicity of hexachloroethane to freshwater organisms was demonstrated in Daphnia magna at 8.07 mg/liter, midge at 1.7 mg/liter, rainbow trout at 0.98 mg/liter, fathead minnow at 1.53 mg/liter, and the bluegill at 0.98 mg/liter. Toxicity to saltwater organisms was observed in the mysid shrimp at 0.94 mg/liter and the sheepshead minnow at 2.4 mg/liter. Chronic toxicity has been observed in the fathead minnow in concentrations ranging from 0.41 to 0.7 mg/liter. Toxicity to aquatic plants was observed in freshwater algae at 87-93.2 mg/liter and in saltwater algae at 7.75-8.57 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects from exposure to hexachloroethane through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 1.9 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for hexachloroethane.

Hexachlorocyclopentadiene

Little human toxicity data is available on hexachlorocyclopenta- diene. Occupational experience has shown that hexachlorocyclopentadiene is highly irritating (USEPA 1980, ACGIH 1980). In a recent incidence in which approximately 200 sewage treatment plant workers were exposed to acutely toxic levels of hexachlorocyclopentadiene from illegal disposal of the compound, workers reported severe irritation of the eyes, nose, throat, and lungs; headache; nausea; and respiratory difficulty (Carter 1977 and Singal 1978, as reported in USEPA 1980).

The acute oral toxicity of hexachlorocyclopentadiene was investigated by Treon et al. (1955, as reported in USEPA 1980). The LD50 for rats and rabbits was determined to be about 500 mg/kg. IRDC (1972) and Naishstein and

Lisovskaya (1965, both as reported in USEPA 1980) have reported similar LD50s for rats of 584 mg/kg and 600 mg/kg, respectively. Hexachlorocyclopentadiene appears to be as acutely toxic by dermal exposure as by oral exposure (USEPA 1980). A 7-hour inhalation exposure to 1.5 ppm hexachlorocyclopentadiene was lethal to rabbits; five 7-hour exposures to 1.0 ppm or two 7-hour exposures to 3.2 ppm was lethal to rats (Treon et al. 1955, as reported in USEPA 1980).

To date, no adequate subchronic or chronic oral toxicity studies of hexachlorocyclopentadiene have been performed. Hexachlorocyclopentadiene was given orally to rats at levels ranging from 0.00002 to 20 mg/kg/day for six months (Naishstein and Lisovskaya 1965, as reported in USEPA 1980). At the high dose level, 2 of 10 animals died; at 0.002 mg/kg/day, the only reported effects were neutropenia and a tendency toward lymphocytosis. Rats, rabbits, guinea pigs, and mice exposed to vapors of hexachlorocyclopentadiene showed irritation of the eyes and mucous membranes (Treon et al. 1955, as reported in ACGIH 1980). At the lowest exposure level (0.15 ppm for 7 hours on each of 150 days over a 216-day period) degenerative changes in the liver and kidneys of all species and pulmonary irritation in mice were observed. Hexachlorocyclopentadiene has been tested for mutagenicity and reported to be nonmutagenic in both short-term in vitro mutagenicity assays and in a mouse dominant lethal study (USEPA 1980).

Hexachlorocyclopentadiene is highly toxic to freshwater fish. EC50 levels for Daphnia magna of 39 and 52 µg/liter have been reported. Investigators have reported 96-hour LC50 values for the fathead minnow ranging from 7 to 180 µg/liter. LC50 values for the channel catfish and bluegill have been reported to be 97 and 130 µg/liter, respectively. The chronic toxicity value for the fathead minnow in an embryo-larval test is 5.2 µg/liter. Ninety-six-hour LC50 values for three saltwater invertebrate and three saltwater fish species ranged from 7 to 48 µg/liter for all species except the polychaete for which the LC50 value was 371 µg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 206 µg/liter for the protection of human health from the toxic properties of hexachlorocyclopentadiene through ingestion of contaminated water and aquatic organisms.

EPA has not yet established an aquatic life ambient water quality criterion for hexachlorocyclopentadiene.

Indeno(1,2,3-cd)pyrene

Indeno(1,2,3-cd)pyrene is a member of the class of polynuclear aromatic hydrocarbons (PAHs) many of which are known for their ability to induce cancer.

Limited data are available on the carcinogenicity of indeno(1,2,3-cd)pyrene. When groups of 20 female mice were painted three times weekly with indeno(1,2,3-cd)pyrene in acetone at concentrations of 0.01, 0.05, 0.1 and 0.5%, no tumors were produced at the 2 lower doses, 6 papillomas and 3 carcinomas were produced at the 0.1% dose level, and 7 papillomas and 5 carcinomas were produced at the 0.5% dose level (Hoffman and Wynder 1966, as

reported in IARC 1973). Indeno(1,2,3-cd)pyrene also produced tumors by subcutaneous administration. Three injections of 0.6 mg indeno(1,2,3-cd)-pyrene in olive oil at one month intervals to male and female mice resulted in sarcomas in 10 of 14 male mice after 265 days and in only 1 of 14 females after 145 days (Lacassagne et al. 1965, as reported in IARC 1973).

No human case studies or epidemiologic studies have been conducted which establish indeno(1,2,3-cd)pyrene as a human carcinogen. Indirect evidence for the compound's carcinogenicity comes from air pollution studies which indicate an excess of lung cancer mortality among workers exposed to high concentrations of PAH-containing material such as coal gas, tars, soot, and coke oven emissions (IARC 1973, USEPA 1980).

No data are available on the aquatic toxicity of indeno(1,2,3-cd)- pyrene.

EPA has not yet established ambient water quality criteria for indeno(1,2,3-cd)pyrene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection to human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at present, a level of 2.8 ng/liter corresponding to a lifetime incremental cancer risk of 0.000001 was recommended.

Isophorone

Human exposure to isophorone vapor causes irritation of the eyes, nose, and throat of unacclimatized workers at concentrations of 25 ppm. Workers exposed to 5 to 8 ppm complained of fatigue and malaise (Silverman et al. 1946, as reported in Clayton and Clayton 1981 and USEPA 1980). Inhalation of 200 and 400 ppm isophorone resulted in nausea, headache, dizziness, faintness, inebriation, and a feeling of suffocation (Smyth and Seaton 1940, as reported in USEPA 1980).

In rats, exposure to isophorone vapors at 750 ppm for "several" hours produced no symptoms other than slight eye and nose irritation; "some" deaths were reported after four hours at 1,840 ppm, usually due to paralysis of the respiratory system or lung irritation (Smyth and Seaton 1940, as reported in USEPA 1980). Rats and guinea pigs exposed to 100 to 500 ppm isophorone 8 hours/day, 5 days/week, for 6 weeks showed weight loss and evidence of pathologic changes in the lung, spleen, and kidney. In rats at 200 ppm and guinea pigs at 500 ppm, conjunctivitis and nasal irritation were observed. Exposure of rats to 50 ppm produced evidence of pathologic changes in the lung and kidney (Smyth 1941, as reported in USEPA 1980). It has been suggested that the material used in these two inhalation studies was an impure commercial product containing appreciable amounts of other volative materials. Therefore, the reported results are of uncertain reliability (USEPA 1980).

Oral LD50s have been reported for rats and mice ranging from 1.87 to 2.37 g/kg. The LD50 reported for rabbits following acute dermal exposure to isophorone is 1.39 g/kg (USEPA 1980) . Isophorone is weakly irritating to the

skin of rabbits, and induces reversible irritation of the conjunctiva and corneal opacity when applied to rabbit eyes (Truhaut et al. 1972, as reported in USEPA 1980). In a 90-day feeding study in rats and dogs given 750 to 3,000 ppm in the diet (rats) or 35 to 150 mg/kg/day in gelatin capsules (dogs), no significant differences between treated and control groups regarding hematology, blood chemistry, urinalysis, or pathologic lesions were observed (Parkin 1972, as reported in USEPA 1980).

For Daphnia magna and the bluegill, EC50 concentrations for isophorone of 117,000 and 224,000 µg/liter, respectively, have been reported (USEPA 1980). For a saltwater species, the mysid shrimp, the 96-hour LC50 is 12,900 µg/liter. Chronic effects were observed in an embryo-larval test with the sheepshead minnow, a saltwater species, at a concentration of 110,000 µg/liter. Because this value is greater than the 96-hour LC50 for mysid shrimp, the sheepshead minnow was not considered to be a sensitive species (USEPA 1980). Cell number production and chlorophyll a content in a freshwater alga were affected in a 96-hour test at concentrations of 122,000 to 126,000 µg/liter. In a saltwater alga, these effects were observed at 110,000 and 105,000 µg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 5.2 mg/liter for protection of human health from the toxic properties of isophorone through ingestion of contaminated water and aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for isophorone.

Naphthalene

Systemic toxicity to naphthalene in humans includes nausea, headache, diaphoresis, hematuria, fever, anemia, liver damage, convulsions, and coma (Sax 1975). Toxic effects have been reported in infants when the only exposure was to the mother during pregnancy (Zinkham and Childs 1958, Anziulewicz et al. 1959, as reported in USEPA 1980). Occupational studies have associated laryngeal cancer with worker exposure in a coaltar naphthalene production facility (Wolf 1976, as reported in USEPA 1980).

Acute toxicity in animals is reflected by the following toxicity values. In the rat, oral LD50s range from 1.78 to 9.43 gm/kg. In the same species, an inhalation LC50 of 100 ppm for 8 hours and a dermal LD50 of 2.5 gm/kg were reported. An LD50 of 5.1 gm/kg was reported in mice injected subcutaneously with naphthalene (Ime et al. 1973, Gaines 1969, NIOSH 1977, Union Carbide Corporation 1968, as reported in USEPA 1980). In subacute and chronic studies, rats fed 2% naphthalene in the diet for at least 60 days developed early cataracts in both groups (Fitzhugh and Buschke 1949, as reported in USEPA 1980). Lens changes and early cataracts were also observed in rabbits fed 1,000 mg/kg naphthalene by gavage for various lengths of time up to 28 days (Van Heyningen and Pirie 1976, as reported in USEPA 1980). Bronchial epithelial changes have been observed in mice treated by intraperitoneal injection with 67.4 mg/kg and sacrificed at different times up to 7 days following treatment (Mahvi et al. 1977, as reported in USEPA 1980).

The offspring of rabbits administered a metabolite of naphthalene, 2-naphthol, were born with cataracts and evidence of retinal damage (Van der Hoeve 1913, as reported in USEPA 1980). However, naphthalene was not mutagenic in several microsomal/bacterial assay systems (McCann et al. 1975, Kraemer et al. 1974, as reported in USEPA 1980).

Lymphatic leukemia was observed in mice treated (skin painting) with a solution of 0.5% coal tar naphthalene in benzene, 5 days per week for life (Knake 1956, as reported in USEPA 1980). Rats, subcutaneously injected with 500 mg/kg of coal tar naphthalene every 2 weeks for a total of seven treatments, developed metaplastic lymphosarcoma (Knake 1956, as reported in USEPA 1980). These studies are difficult to interpret, however, because of the nature of the purity of the naphthalene administered. Several skin painting studies, using pure naphthalene on mice (Kennaway 1930, Kennaway and Hieger 1930, as reported in USEPA 1980) and rabbits (Bogdat'eva and Bid 1955, as reported in USEPA 1980) showed no carcinogenic effect. The possible carcinogenicity of pure naphthalene consequently has not yet been established.

The acute toxicity of naphthalene to aquatic organisms was demonstrated in six different species in concentrations ranging from 2.3 to 8.9 mg/liter. Mosquitofish and pacific oysters were more resistant, having 96-hour LC50 values of 150 and 199 mg/liter, respectively. Chronic toxicity has been demonstrated in the fathead minnow in concentrations ranging from 0.45 to 0.85 mg/liter. Algae were affected at 33 mg/liter (USEPA 1980).

EPA has not yet established ambient water quality criteria for naphthalene because of the lack of sufficient information.

Nitrobenzene

The most characteristic acute symptom in humans, following exposure to nitrobenzene, is cyanosis as a result of methemoglobin formation. Anemia may develop 1 or 2 weeks after a poisoning or in more severe, cases may lead to coma and death. The symptoms of chronic occupational exposure to nitrobenzene include cyanosis, methemoglobinemia jaundice, anemia, sulfhemoglobinemia, persistence of Heinz bodies in the erythrocytes, and dark-colored urine. There have been reports of menstrual disturbances in women chronically exposed to nitrobenzene, and changes have been observed in the tissues of the chorion and placenta from pregnant women occupationally exposed to nitrobenzene (USEPA 1980).

Nitrobenzene has induced methemoglobin formation in dogs, cats, and rats, but not in guinea pigs or rabbits (Levin 1927, as reported in USEPA 1980). An early study found that nitrobenzene fumes caused cerebellar disturbances in dogs and birds (Dresbach and Chandler 1918, as reported in USEPA 1980). Little information is available on the teratogenic effects of nitrobenzene. In one study on rats, however, a subcutaneous dosage of 125 mg/kg during the preimplantation and placentation periods was associated with delay of embryogenesis, alteration of normal placentation, and fetal abnormalities (Kazanina 1968, as reported in USEPA 1980). No studies are available that

show nitrobenzene to be mutagenic or carcinogenic. Nitrobenzene is, however, structurally related to known carcinogens (USEPA 1980).

Among freshwater animal species, the 48-hour EC50 is 27 mg/liter for Daphnia magna, and the 96-hour LC50 is 42.6 mg/liter for the bluegill. A freshwater alga, Selenastrum capricornuum, has an EC50 value for chlorophyll a of 44.1 mg/liter. In saltwater animal species, the 96-hour LC50s are 6.68 mg/liter for the mysid shrimp and 58.6 mg/liter for the sheepshead minnow. The saltwater alga Skeletonema costatum had an EC50 value for chlorophyll a of 10.3 mg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 19.8 mg/liter for the maximum protection of human health from the toxic properties of nitrobenzene through the ingestion of water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for nitrobenzene.

N-Nitrosodimethylamine

Dialkyl N-nitrosoamines are characteristically hepatotoxic, producing hemorrhagic centrilobular necrosis. A few cases of human poisoning from exposure to N-nitrosodimethylamine have been reported. Symptoms included abdominal pains, exhaustion, headaches, and distended abdomens. Clinical examination showed signs of liver damage and bronchopneumonia in one case, and autopsies have revealed cirrhotic livers (USEPA 1980).

Acute exposure of experimental animals to N-nitrosodimethylamine produces liver lesions in 24 to 48 hours, peritoneal and sometimes pleural exudate, and in some cases kidney lesions (USEPA 1980). An acute oral LD50 of 40 mg/kg has been reported for the rat (USEPA 1980). N-nitrosodimethylamine has been reported to induce forward and reverse mutations in several bacterial species, gene recombination and conversion in Saccharomyces cerevisiae, recessive lethal mutation in Drosophila melanogaster, and chromosome aberrations in mammalian cells (Montesano and Bartsch 1976, as reported in USEPA 1980). N-nitrosodimethylamine is carcinogenic in all species tested: mice, rats, hamsters, guinea pigs, rabbits, ducks, mastomys, various fish, newts, and frogs (IARC 1978). In mice, chronic administration in the drinking water at a concentration corresponding to a dose of 0.4 mg/kg/day produced lung adenomas and hemangiocellular tumors in 13/17 and 2/10 mice, respectively (Clapp and Toya 1970, as reported in IARC 1978). Numerous studies in the rat have shown that administration of 50-100 mg/kg in the diet or drinking water (or 4 mg/kg/day) leads to the development of high incidences of hepatocellular carcinomas and cholangiocellular tumors. Kidney tumors have been produced by short-term or single-dose treatment with high oral doses (up to 30 mg/kg body weight) (IARC 1978). Lung adenocarcinomas and squamous-cell carcinomas have also been observed after oral N-nitrosodimethylamine treatment (IARC 1980). Druckrey et al. (1967, as reported in IARC 1978) reported tumors of the nasal cavity in rats exposed by inhalation twice weekly for 30 minutes at a concentration corresponding to 2 mg/kg body weight. Single subcutaneous

administration of N-nitrosodimethylamine in mice at doses of 1, 2, 4, and 8 mg/kg produced lung tumor incidences of 29%, 35%, 39%, and 67%, respectively (Cardesa et al. 1974, as reported in IARC 1978). A thorough review of the available carcinogenicity data for N-nitrosodimethylamine is presented in IARC (1978) and USEPA (1980).

No acute aquatic toxicity information is available for N-nitrosodimethylamine. However, available data for nitrosamines indicates that acute toxicity to freshwater aquatic life occurs at concentrations as low as 5,850 µg/liter (USEPA 1980). In a 52-week feeding study with rainbow trout, a dose-related response of hepatocellular carcinoma occurred in trout fed 200 to 800 mg N-nitrosodimethylamine (Grieco et al. 1978, as reported in USEPA 1980). In a study by Harshbarger et al. 1971 (as reported in USEPA 1980), crayfish exposed for six months to N-nitrosodimethylamine developed extensive degeneration of the antennal gland at 200,000 µg/liter and hyperplasia of the hepatopancreas at 100,000 µg/liter. No toxicity data are available for saltwater species.

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to N-nitrosodimethylamine through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, a level of 1.4 ng/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for N-nitrosodimethylamine.

N-Nitrosodiphenylamine

The dialkyl N-nitrosamines are characteristically hepatotoxic, producing hermorrhagic centrilobular necrosis. Acute oral LD50 values in the rat have been given as 1,650 and 3,000 mg/kg (USEPA 1980). The class of N-nitroso compounds includes some of the most powerful chemical mutagens known. However, N-nitrosodiphenylamine is reported to give a negative response in both *Salmonella typhimurium* and *E. coli* mutagenicity assays after activation with a rat liver microsomal preparation (USEPA 1980). In a recent National Cancer Institute bioassay (Cardy et al. 1979, as reported in USEPA 1980) rats developed neoplastic and non-neoplastic urinary bladder lesions after two years of dietary administration of N-nitrosodiphenylamine at a dose-level of 200 mg/kg/day.

Acute toxicity from exposure to N-nitrosodiphenylamine has been reported for *Daphnia magna* and the bluegill at concentrations of 7,760 and 5,850 µg/liter, respectively. A 96-hour LC50 for the mummichog, a saltwater species, of 3,300 mg/liter has been reported (USEPA 1980). No chronic aquatic studies are available for N-nitrosodiphenylamine.

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to N-nitrosodiphenylamine through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the

present time, a level of 4.9 µg/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for N-nitrosodiphenylamine.

N-Nitrosodi-n-propylamine

No information on the toxicity of N-nitrosodi-n-propylamine in humans is available.

The dialkyl N-nitrosamines are characteristically hepatotoxic, producing hemorrhagic centrilobular necrosis. An acute oral LD50 in the rat is reported as 480 mg/kg. The subcutaneous LD50 for the rat is given as 487 mg/kg and for the Syrian golden hamster as 600 mg/kg (IARC 1978). N-nitrosodi-n-propylamine is carcinogenic in experimental animals. Administration in drinking water at doses of 4, 8, 15, or 30 mg/kg/day produced liver carcinomas in 45 of the 48 rats tested after induction times ranging from 120 to 300 days (Druckrey et al. 1967, as reported in IARC 1978). Male and female rats injected subcutaneously with 24.4, 48.7, or 97.4 mg/kg N-nitrosodi-n-propylamine once weekly for life developed a high incidence of neoplasms of the nasal or paranasal cavities (45 of 58 treated rats). In addition, 13 liver tumors, 11 lung cancers, and 11 squamous-cell papillomas of the esophagus were seen (Althoff et al. 1973, as reported in IARC 1978). Syrian golden hamsters were treated subcutaneously with 1.2% N-nitrosodi-n-propylamine in olive oil once weekly for life at doses of 3.75, 7.5, 15, 30, or 60 mg/kg. A high incidence of tumors of the nasal and paranasal cavities, laryngobronchial tract, and lung were observed in treated guinea pigs but not in controls (Althoff et al. 1973, as reported in IARC 1978). N-nitrosodi-n-propylamine is mutagenic in in vitro assays with the bacteria Salmonella typhimurium and E. coli and Chinese hamster V79 cells (IARC 1978).

No aquatic toxicity studies are available for N-nitrosodi-n- propylamine. However, available data for nitrosamines indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 5,850 µg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to N-nitrosodi-n-propylamine through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, a level of 0.8 ng/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for N-nitrosodi-n-propylamine.

Phenanthrene

Phenanthrene is a polynuclear aromatic hydrocarbon that causes skin photosensitization in humans and has produced cancer in animals (Sax 1975). The oral LD50 in mice is 700 mg/kg (USDHHS 1980).

The information on the toxicity of phenanthrene to aquatic organisms is limited to one study of a crude oil fraction of phenanthrene that produced a 96-hour LC50 value of 600 µg/liter in the polychaete worm (USEPA 1980).

EPA has not yet established ambient water quality criteria for phenanthrene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include phenanthrene, through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 2.8 ng/liter, corresponding to a lifetime incremental cancer risk of 10-6, was recommended.

Pyrene

No information is available on the toxicity of pyrene in humans, animals, or aquatic life. In an *in vitro* assay, pyrene exhibited toxicity to transplanted rat respiratory epithelium and the submucosa of the trachea (Topping *et al*. 1978). Derivatives of pyrene, however, such as benzo¢a!pyrene, are highly potent animal carcinogens.

EPA has not yet established ambient water quality criteria for pyrene because of the lack of sufficient data. However, the agency has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to polynuclear aromatic hydrocarbons, which include pyrene, through ingestion of contaminated water and contaminated aquatic organisms. However, since a zero level may not be attainable at present, a level of 2.8 ng/liter, corresponding to a life time incremental cancer risk of 10-6, was recommended.

2,3,7,8-Tetrachlorodibenzo-p-dioxin

2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is one of the most toxic substances known. It produces a delayed biological response in many species and is highly toxic at low doses to aquatic organisms and mammals, including humans. Many human exposures to TCDD result from occupational exposure to 2,4,5-trichlorophenol (2,4,5-TCP) or 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), in which TCDD is a contaminant.

TCDD has been identified as the cause of numerous outbreaks of chloracne in humans. In addition, investigators have reported muscular weakness, loss of appetite and weight, sleep disturbances, hypotension, abdominal pain, liver

impairment, peripheral neuropathy, psychopathological changes, hyperlipaemia and hypercholesterolaemia, and porphyria cutanea tarda among exposed persons (USEPA 1979).

TCDD is extremely toxic in all animal species tested, with oral LD50 values for several species ranging between 0.6 and 115 µg/kg (NAS 1977). Rats given single doses of 25 and 50 µg/kg of TCDD showed moderate to severe thymic atrophy and liver damage; rats that received 100 µg/kg showed 43% mortality, severe liver damage, thymic atrophy, and icterus (Gupta et al. 1973, as reported in NAS 1977). Mortality following acute oral doses may be delayed. In female rats given single oral doses up to 300 µg/kg, delayed mortality was observed over a 90-day period (Greig et al. 1973, as reported in NAS 1977). McConnell et al. (1978, as reported in USEPA 1979) observed weight loss, blepharitis, facial alopecia with acneform eruptions, and anemia in rhesus monkeys given single oral doses of 70 to 350 µg/kg TCDD. Deaths were also reported.

Damage to the liver and thymus are the predominant effects of subchronic administration of TCDD. In a 13-week study conducted by Kociba et al. (1976, as reported in NAS 1977), degenerative changes in the liver and thymus, porphyria, altered serum enzyme concentrations, and loss of body weight were reported in rats given 0.1 µg/kg TCDD five days a week. Young female rhesus monkeys given a diet containing 500 ppt TCDD for up to nine months showed loss of facial hair and eyelashes, edema, accentuated hair follicles, and dry scaly skin (Allen et al. 1977, as reported in USEPA 1979). Five of the eight monkeys died from severe pancytopenia.

TCDD is fetotoxic and teratogenic in various animal species. Fetuses from rats that had received 0.125 µg/kg/day showed reduced body weight and a slight increase in intestinal hemorrhage and edema. At 0.5 µg/kg/day, a reduction in fetal number and increase in fetal deaths were reported (Sparschu et al. 1971, as reported in NAS 1977). Courtney and Moore (1971, as reported in NAS 1977) reported an increase in fetal kidney malformations in rats that received TCDD subcutaneously at 0.5 µg/kg/day on days 9, 10 or 13 and 14 of gestation. Increased incidences of cleft palate and kidney abnormalities were reported in mice that received 1.0 and 3.0 µg/kg/day on days 6 to 16 of gestation (Smith et al. 1976, as reported in USEPA 1979). The Advisory Committee on 2,4,5-T (1971, as reported in NAS 1977) reported gastrointestinal hemorrhage in the fetuses of hamsters that received 0.5 µg/kg/day TCDD on days 6-10 of gestation.

TCDD is a potent carcinogen. Ingestion by rats of 2.2 ppb or 0.1 µg/kg/day induced squamous cancer of the respiratory tract and oral cavity in males and females and liver cancer in females only (Kociba et al. 1978). Van Miller et al. (1977, as reported in USEPA 1979) fed rats diets containing 0.001 to 1,000 µg TCDD/kg diet. An increased incidence of liver tumors was reported in groups of rats receiving TCDD at levels of 0.005 to 5 µg/kg of diet; animals in the higher dose groups died between the second and fourth weeks of treatment.

TCDD has been shown to be mutagenic in three bacterial systems (Hussain 1972, as reported in USEPA 1979). However, this finding has not been confirmed by other researchers.

The data on aquatic toxicity of TCDD is not extensive. Miller et al. (1973, as reported in USEPA 1979) exposed coho salmon to 0.000056 µg/liter of TCDD for 96 hours under static conditions and then transferred the fish to control water. Sixty days after exposure, the mortality among the exposed fish was 12 percent compared to 2 percent among controls. An exposure of 0.056 µg/liter for 24 hours was lethal to all salmon within 40 days. Reduced reproduction was reported in the snail, Physa sp. and worm, Paranis sp., exposed to 0.2 µg/liter for approximately 1,175 hours (Miller et al. 1973, as reported in USEPA 1979).

No data on the toxicity of TCDD to saltwater organisms or to aquatic plants are available.

EPA has proposed an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to TCDD through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, the Agency considered establishing an interim criterion of 4.55×10^{-8} µg/liter, corresponding to a lifetime risk of 0.000001. A final criterion will be established upon completion of a review of the carcinogenic potential of TCDD by the Agency.

EPA has not yet established an aquatic life water quality criterion for TCDD.

1,2,4-Trichlorobenzene

Human exposure to 1,2,4-trichlorobenzene vapor at 3 and 5 ppm causes minor eye and respiratory irritation (Rowe 1975 in USEPA 1980). No apparent "serious" illness, change in liver function, or alteration of blood components were observed over a period of 4 years in workers employed in a plant where benzene was chlorinated. One worker inhaled a "massive" amount of trichlorobenzene and experienced lung hemorrhaging (NAS 1977).

The single dose acute oral LD50 for 1,2,4-trichlorobenzene is 756 mg/kg in rats and 766 mg/kg in mice (Brown et al. 1969 in USEPA 1980). The rats' deaths occured within 5 days of exposure, and the mice died within 3 days of exposure. For both species, decreased signs of activity were observed at low doses, and convulsions occurred at higher doses. 1,2,4-Trichloro- benzene was not irritating in subchronic skin irritation studies with rabbits and guinea pigs (Brown et al. 1969 in USEPA 1980). However, skin inflammation in rabbits was noted after 3 weeks of exposure. 1,2,4-Trichlorobenzene applied to rabbit ears for 13 weeks produced some dermal irritation.

Rats, rabbits, and monkeys were administered 1,2,4-trichlorobenzene by inhalation at concentrations of 25, 50, and 100 ppm for up to 26 weeks (Coate et al. 1977 in USEPA 1980). No "exposure-related" ophthalmologic, hematologic, pulmonary, or metabolic (blood urea nitrogen, bilirubin, serum glutamic oxaloacetic transaminase, serum glutamic-pyruvic transaminase, lactic dehydrogenase, and alkaline phosphatase) changes were observed. Histologic changes were noted in the livers and kidneys of rats necropsied at 4 weeks. The changes were dose-related and were observed in animals from each treatment

group. However, after 20 weeks, no compound-related histopathological changes were noted in rabbits or monkeys. Mice exposed to 600 ppm of 1,2,4-trichlorobenzene for 6 months did not develop hepatomas. No other information is available on the carcinogenicity, mutagenicity, or teratogenicity of 1,2,4-trichlorobenzene (USEPA 1980).

The acute toxicity of 1,2,4-trichlorobenzene to various saltwater and freshwater species is reflected by the 96-hour LC50 values in cladoceran of 50.2 mg/liter, rainbow trout of 1.5 mg/liter, fathead minnow of 2.9 mg/liter, mysid shrimp of 0.45 mg/liter, and sheepshead minnow of 21.4 mg/liter. The chronic toxicity value for 1,2,4-trichlorobenzene to fathead minnow ranges from 0.28 to 0.71 mg/liter. 1,2,4-Trichlorobenzene has also demonstrated acute toxic effects to freshwater and saltwater algae with 96-hour EC50 values ranging from 8.7 to 36.7 mg/liter. The whole-body, 28-day bioconcentration factor of 1,2,4-trichlorobenzene in the bluegill is 182 (USEPA 1980).

EPA has not yet established ambient water quality criteria for 1,2,4-trichlorobenzene because of the lack of sufficient information.

4. Metals and Cyanide

Antimony

The major toxic symptoms that are associated with antimonial compounds in humans involve the gastrointestinal tract, heart, respiratory tract, skin, and liver. The most serious effects of these compounds have been observed during antimonial therapy and during industrial exposure (National Institute for Occupational Safety and Health 1978, as reported in NAS 1980). Symptoms include cardiac alterations, bradycardia, and fluctuations in blood pressure (Brieger et al. 1954, NIOSH 1978, as reported in NAS 1980). Respiratory changes include irritation of the mucous membranes, upper respiratory tract irritation, and, more seriously, pneumonoconiosis. Pneumonia has also been cited as a side effect to the therapeutic use of antimony. Gastrointestinal symptoms include cramps, nausea, pain, anorexia, diarrhea, and vomiting (NAS 1980). Chromosomal damage in human leukocytes studied in vitro occurred with exposure to as little as 280 mg/liter of antimony (Paton and Allison 1972, as reported in USEPA 1980 and NAS 1980). A greater incidence of spontaneous abortion, premature deaths, and gynecological problems were reported in antimony workers at a metallurgical plant (Belyayeva 1967, as reported in NAS 1980).

Acute poisoning of antimonial compounds in animals produce labored breathing, general weakness, and other signs of cardiovascular insufficiency leading to death among many animals within several days after exposure. Oral LD50 values are 300 mg/kg (tartar emetic) for the rat and 804 mg/kg (antimony trifluoride) for the mouse (Bradley and Fredrick 1941, as reported in USEPA 1980).

In chronic animals studies, rats fed 135 mg/kg trivalent antimony chloride for 10 days developed toxic symptoms including myocardial degeneration (Arzamastsev 1964, as reported in NAS 1980). Decreased hemoglobin and increased reticulocyte count were observed in guinea pigs treated for 10 days with 12 and 20 mg/kg trivalent antimony chloride (Arzamastsev 1964, as reported in NAS 1980). In a longer study, rats orally fed 200 mg antimony potassium tartrate died after 85 days (Flury 1927, as reported by NAS 1980). Decreased fertility was reported in rats exposed to 50 mg/kg metallic antimony by inhalation (Belyayeva 1967, as reported in NAS 1980). No evidence of carcinogenicity was observed in mice given 5 µg of antimony potassium tartrate per milliliter of drinking water throughout their lifetime (Kanisawa and Schroeder 1969, as reported in USEPA 1980).

The acute toxicity of antimony compounds on aquatic organisms is reflected by 96-hour LC50 values in cladoceran of 9 mg/liter for antimony potassium tartrate, and 21.9 mg/liter in the fathead minnow and 18.8 mg/liter in cladoceran for antimony trichloride. 96-Hour LC50 values for antimony trioxide are greater than 530 mg/liter in the bluegill and greater than 4.2 mg/liter in the mysid shrimp. For this same compound the 96-hour LC50 ranges from 6.1 to 8.3 mg/liter. Chronic toxicity was observed in cladoceran exposed to between 4.2-7.0 mg/liter antimony trichloride; and the fathead minnow exposed to greater than 7.5 µg/liter for antimony trioxide and between 1.1

and 2.3 mg/liter for antimony trichloride. Freshwater algae were affected by 0.61-0.63 mg/liter antimony trioxide while freshwater algae were not affected by levels of 4.2 mg/liter antimony trioxide (USEPA 1980).

EPA has established an ambient water quality criterion of 146 µg/liter for the protection of human health from the toxic properties of antimony ingested through water and contaminated aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for antimony.

Arsenic

The toxicity of arsenic varies according to the physical form and the oxidation state of the compound. In general, soluble trivalent arsenic compounds are more toxic than the pentavalent species, and inorganic arsenicals are also more toxic than organic arsenicals. Inorganic arsenate predominates in most waterways because of its stability and solubility.

Many epidemiological studies have linked the development of cancer to exposure to inorganic arsenic compounds. Evidence has come from the use of arsenicals as drugs, from geographical areas with high arsenic levels in the drinking water, and from occupational exposures of workers in mining operations, smelters, pesticide manufacture and vineyards. However, this evidence associating inorganic arsenic compounds with lung cancer in humans is still open to question. For skin cancer, however, a causal relationship between incidence and high-level exposures to inorganic arsenic compounds has been reported (Tseng et al. 1968, as reported in NAS 1977).

Symptoms of acute arsenic poisoning by ingestion include abdominal pain and vomiting, while acute poisoning by inhalation produces giddiness, headache, extreme general weakness, and later nausea, vomiting, colic, diarrhea, and pains in the limbs (Browning 1961, as reported in NAS 1977).

Although no animal experiments have demonstrated carcinogenicity of arsenic, several have shown that sodium arsenate and arsenite induce developmental malformations in a variety of test animals including chick embryos, hamsters, rats, and mice (USEPA 1980).

Arsenic has been shown to be toxic to both vertebrate and invertebrate freshwater aquatic organisms and saltwater aquatic organisms. Cladocerans have been reported to be more sensitive than fish, although certain invertebrates such as stoneflies may be more tolerant than fish. Static 96-hour LC50 values range from 0.812 mg/liter sodium arsenite for the scud to 22 mg/liter for the stonefish. However, less sensitive static 96-hour LC50 values of 26 mg/liter and 41.76 mg/liter sodium arsenite, respectively, were reported for goldfish and bluegill. One hundred percent of three different strains of freshwater algae were killed in 2 weeks in 2.32 mg/liter sodium arsenite. Arsenic has been shown to bioconcentrate in both fresh and saltwater organisms (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to arsenic through the ingestion of contaminated water and contaminated aquatic organisms. Since the zero level may not be attainable at the present time, a level of 2.2 ng/liter, corresponding to an estimated lifetime incremental increase of cancer risk of 10-6, was recommended.

EPA has not yet established an aquatic life water quality criterion for arsenic.

Asbestos

Asbestos is a term applied to numerous fibrous mineral silicates composed of silicon, oxygen, hydrogen, and metal cations. The two major groups of asbestos are serpentine (chrysotile) and amphibole. Numerous epidemiological studies have shown that long-term exposure to asbestos dust can lead to asbestosis and an increased risk of cancer.

Asbestosis is a chronic, progressive pneumoconiosis, characterized by fibrosis of the lung parenchyma. Other symptoms of the disease include cough, rales, finger clubbing, restrictive pulmonary dysfunction, and weight loss. In some cases the disease can lead to death. X-rays typically reveal small, irregular opacities in the lungs, often accompanied by pleural fibrosis, thickening, or calcification.

An association between inhalation of asbestos and an increased risk of cancer has been clearly established in epidemiologic studies. In a study of the mortality experience of 17,800 asbestos insulation workers from 1967 to 1976 by Selikoff et al. (1979, as reported in USEPA 1980), significantly increased incidences of lung tumors, mesotheliomas, cancer of the gastrointestinal tract, larynx, pharyxn, and buccal cavity, and renal tumors were reported.

Several studies of cancer incidence among factory workers employed in the manufacture of asbestos products have been reported (USEPA 1980). Investigators have shown a significant excess in total mortality in exposed workers, with important contributions from asbestosis, cancer of the lung, bronchus, and trachea, and neoplasms of the digestive organs and peritoneum (including peritoneal mesothelioma). Mesothelioma is a form of cancer that is very rare among individuals not exposed to asbestos. Increased risks of lung cancer and mesothelioma have also been reported among individuals exposed only indirectly to asbestos, including shipyard workers and groups living or working in an area of asbestos mining (USEPA 1980).

Several studies have considered the relationship between ingestion of asbestos in drinking water and gastrointestinal cancer. The human data is, however, inconclusive.

The carcinogenicity of asbestos has also been demonstrated in animals. Gross et al. (1967, as reported in USEPA 1980) reported 19 adenocarcinomas, 4 squamous cell carcinomas, and one mesothelioma among 72 rats exposed by inhalation to 86 mg/cu m chrysotile for 16 months or longer. No malignant

tumors were found in 39 controls. Rats exposed to various forms of asbestos at concentrations ranging from 10 to 15 mg/cu m for periods ranging from 1 day to 24 months showed an increased incidence of adenocarcinomas, squamous-cell carcinomas, and mesotheliomas. No tumors appeared prior to 300 days from first exposure, and none of these tumors appeared in control animals (Wagner et al. 1974, as reported in USEPA 1980).

Mesotheliomas have been produced in rats by intrapleural injection of 10 mg of asbestos (Reeves et al. 1971, as reported in USEPA 1980) and by intraperitoneal injection (Maltoni and Annoscia 1974, as reported in USEPA 1980). The carcinogenicity of asbestos administered by ingestion has not been demonstrated.

No data were available on the potential toxicity of asbestiform material to freshwater or saltwater organisms.

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to asbestos through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, a level of 30,000 fibers/liter, corresponding to a lifetime cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for asbestos.

Beryllium

Beryllium is relatively nontoxic to humans when ingested in food and water because absorption from the digestive tract is slight (NAS 1977). However, beryllium and several beryllium compounds can cause acute and chronic effects in humans. The most common effects of industrial exposure to beryllium are skin lesions: dermatitis, ulceration, and granulomas. Dermatitis has been regarded as a sensitizing reaction (Doull et al. 1980). Acute effects on the respiratory system -- known as acute berylliosis -- may occur following inhalation of beryllium at levels of 30 mg/cu m for the high-fired oxide, 1-3 mg/cu m for the low-fired oxide, and 0.1-0.5 mg/cu m for beryllium sulfate; effects are usually reversible after weeks or months (Reeves et al. 1979, as reported in IARC 1980). The acute response is characterized by nasopharyngistis, tracheobronchitis, and fulminating pneumonia (Doull et al. 1980).

Repeated inhalation exposure can lead to chronic berylliosis, with latent periods of 10 to 20 years from the first exposure commonly observed. Chronic berylliosis is characterized by dyspnea, chronic cough, weight loss, weakness, fatigue, and chest pain. Systemic impairments include granulomatous inflammation of the lungs; involvement of the striated muscle, liver, spleen, kidneys, and heart; disturbances in nitrogen and calcium metabolism; and immunological sensitization (Doull et al. 1980). Human case reports and epidemiologic studies provide suggestive but inconclusive evidence that beryllium is carcinogenic in humans. A positive correlation between beryllium concentrations in drinking water and cancer deaths in 15 regions of the

country was reported (Berg and Burbank 1972, as reported in USEPA 1980). The results of three epidemiological studies suggest that beryllium exposure increased the risk of cancer mortality; however, confirmatory studies are needed to evaluate the importance of other risk factors in beryllium-associated lung cancer cases (USEPA 1980).

In animal studies, beryllium has been shown to be acutely toxic to rats by intravenous injection at 0.44 to 0.51 mg Be/kg (as soluble beryllium salts), by the oral route at 9.7 mg/kg, and by inhalation at 42-194 µg/cu m (USEPA 1980). Chronic beryllium disease can be produced in experimental animals by inhalation of low concentrations of soluble beryllium salts. Rats exposed for up to 6 months to 35 µg/cu m of beryllium sulfate aerosol developed typical chronic pneumonitis along with granulomatous lesions and some neoplasms (Schepers et al. 1957, as reported in USEPA 1980). Exposure of monkeys to 35 µg/cu m of beryllium sulfate or intratracheal instillation of a 5 percent suspension of beryllium oxide resulted in chronic pneumonitis in all animals (Vorwald et al. 1966, as reported in USEPA 1980). Macrocytic anemia and osteosclerotic changes have also been reported in animals following chronic exposure to beryllium (USEPA 1980). Lung cancer and bone cancer (osteosarcoma) are the two types of malignancies commonly induced in experimental animals by exposure to beryllium compounds. In an inhalation study in which rats were exposed to beryllium sulfate at 2.8, 21, and 42 µg Be/cu m for 7 hours/day, 5 days/week, for a period of 18 months, pulmonary cancers were found in almost all animals at the two highest doses and in 62 percent at the low dose (Vorwald et al. 1966, as reported in USEPA 1980). Groups of rats were administered beryllium oxide calcined at temperatures of 500-1600oC by intratracheal instillation at a dose of 25 mg/kg; pulmonary cancers were reported in 25 to 100% of the animals (Spencer et al. 1968, as reported in USEPA 1980). Osteosarcomas have been produced in rabbits by intravenous injections or injections into the bone by numerous investigators. In one study, osteosarcomas were produced in 89% and 100% of the rabbits injected with beryllium oxide into the femur at doses of 220-400 and 420-600 mg, respectively, twice weekly for 1-43 weeks (Yamaguchi and Katsura 1963, as reported in USEPA 1980).

Acute beryllium aquatic toxicity data are available for the fathead minnow, guppy, and bluegill at levels of hardness ranging from about 20 to 400 mg calcium carbonate/liter. For these species, acute toxic effects were reported at concentrations ranging from 130 µg/liter to 3,200 µg/liter (USEPA 1980). A 48-hour EC50 for Daphnia magna of 2,500 µg/liter was reported. In a chronic life-cycle study with Daphnia magna, toxic effects were observed at 5.3 µg/liter (USEPA 1980). Toxic effects of beryllium on saltwater organisms have not been reported. In one study of freshwater plants, the growth of a green alga was inhibited at a beryllium concentration of 100,000 µg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to beryllium through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at present, a level of 3.7 ng/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has not yet established an aquatic life water quality criterion for beryllium.

Cadmium

Human toxicity resulting from exposure to cadmium is well-documented, the major effects being respiratory and renal toxicity. Numerous cases of acute cadmium poisoning in humans have been reported. Acute lethal doses by inhalation vary considerably depending on chemical form, particle size, and duration of exposure; the product of concentration x time representing a lethal dose of cadmium fumes has been estimated to be 2,600 mg/cu m. min, i.e., 2,600 mg/cu m for one minute, 26 mg/cu m for 100 minutes, and so on (Doull et al. 1980). The minimal toxic dose for an 8-hour inhalation exposure is estimated to be 1 to 3 mg/cu m (CEC 1978, as reported in Doull et al. 1980). Acute toxic effects of cadmium inhalation include irritation of the upper respiratory tract, chest pains, nausea and diarrhea, dizziness, and death usually due to massive pulmonary edema (Doull et al. 1980). Acute lethal cadmium doses by oral exposure in humans are estimated to range from 350 to 8,900 mg; major toxic effects of cadmium ingestion include nausea, vomiting, salivation, diarrhea, and abdominal cramps. Death due to shock, dehydration or delayed systemic effects (notably renal and cardiopulmonary failure) may occur (Doull et al. 1980).

The principal target organs following chronic exposure to cadmium are the lungs and kidney. Chronic inhalation of cadmium fumes and dust can lead to an emphysema-like condition with loss of ventilatory capacity, increased residual lung volume, and shortness of breath (Lauwerys et al. 1974, as reported in Doull et al. 1980). The kidney appears to be the most cadmium-sensitive organ, primarily because of its prediliction for accumulation of cadmium. Renal toxicity of cadmium was first reported in a study of industrial exposure to cadmium oxide dust in an alkaline storage battery factory (Friberg 1948, as reported in Doull et al. 1980). Workers exhibited consistent proteinuria and a reduced ability to concentrate urine. Glycosuria, hypercalciuria, aminoaciduria, and increased uric acid excretion have also been reported in workers (Kazantzis et al. 1963, as reported in Doull et al. 1980). Based on limited renal biopsies of affected and nonaffected workers, Friberg et al. (1974, as reported in Doull et al. 1980) have proposed a threshold concentration for renal toxicity of 200 µg Cd/g kidney cortex. An incident of chronic cadmium poisoning resulting from dietary intake was reported in Japan in the 1940's. The disease was named Itai-itai (or ouch-ouch). People consuming cadmium-contaminated rice developed osteomalacia with attendant spontaneous multiple bone fractures as well as porteinuria and glycosuria (Doull et al. 1980).

Several epidemiologic studies provide suggestive evidence that cadmium increases the risk of prostatic cancer in men. Lemen et al. (1976, as reported in Clayton and Clayton 1981) found an excess of cancers of the prostate among cadmium smelter workers. Five of eight deaths among a group of 74 alkaline cadmium battery workers who had been exposed to CdO dust for ten or more years were due to cancer, including three carcinomas of the prostate (Potts 1965, as reported in Clayton and Clayton 1981). In a survey of 248 workers exposed to CdO for one or more years, four deaths from prostatic

cancer, significantly more than expected, were observed (Kipling and Waterhouse 1967, as reported in Clayton and Clayton 1981). The evidence for carcinogenicity of cadmium in humans is not conclusive, however, because of the small study populations and confounding exposures to other elements which are known to be human carcinogens (USEPA 1980).

The toxic effects of cadmium observed in humans have been reproduced in experimental animals. The acute oral LD50 varies from approximately 100 mg/kg for soluble salts of cadmium to several thousand mg/kg for metallic cadmium powder or the insoluble selenide and sulfide. Rats exposed to a cadmium aerosol for 15 days developed lung inflammation followed by emphysema and fibrosis (Snider et al. 1973, as reported in Doull et al. 1980). Itai-itai disease has also been reproduced experimentally in rats given an excess of cadmium in a calcium deficient diet (Itokawa et al. 1974, as reported in Doull et al. 1980). In newborn animals, cadmium has been shown to cause cerebral and cerebellar damage. Cadmium also is toxic to the testes of rats and mice, and causes hyperglycemia and glucose intolerance in animals (Doull et al. 1980).

Animal studies have demonstrated that the injection of cadmium metals or salts causes sarcomas at the site of injection and testicular tumors (Leydig or interstitial cell tumors). Interstitial cell tumors and subcutaneous sarcomas were reported in rats following a single subcutaneous injection of 0.03 mmol cadmium chloride (Gunn 1963, as reported in Clayton and Clayton 1981). Cadmium metal, CdO, CdS, and cadmium sulfate have also elicited injection-site sarcomas (Clayton and Clayton 1981 and USEPA 1980). Several long-term feeding and inhalation studies have been carried out with cadmium compounds; the induction of tumors by these routes of exposure has not been observed (USEPA 1980).

Predicting the impact of cadmium on aquatic organisms is complicated by the variety of forms in which cadmium may be present, the differences in toxicity and availability of the various forms, hardness of the water, pH, temperature, and presence of other metal ions (USEPA 1980). The results of acute toxicity tests on cadmium with 29 freshwater fish and invertibrate species range from 1 to 73,500 µg/liter (as Cd); both EC50 and LD50 values are included in this range. Chronic toxicity was observed in Daphnia magna at concentrations ranging from 0.15 to 0.44 µg/liter, and in 12 freshwater fish species at concentrations ranging from 1.7 to 50 µg/liter (USEPA 1980). In a 42-day study of cadmium toxicity to the bay scallop, exposure to 60 and 120 µg/liter reduced growth by 42 and 69 percent, respectively (Pesch and Stewart 1980, as reported in USEPA 1980). A 48-day exposure of copepods to cadmium inhibited reproduction at concentrations greater than 44 µg/liter (D'Agostino and Finney 1974, as reported in USEPA 1980). Acute toxicity values for 5 species of saltwater fish ranged from 577 µg/liter for larval Atlantic silversides to 114,000 µg/liter for juvenile mummichog. Acute toxicity values for 26 species of saltwater invertibrates ranged from 15.5 µg/liter for the mysid shrimp to 46,600 µg/liter for the fiddler crab. In two life-cycle studies of mysid shrimp, toxic effects were observed at concentrations of 5.5 and 8.0 µg/liter (USEPA 1980). Growth reduction was the major toxic effect observed in several species of freshwater plants at concentrations ranging from 2 to 7,400 µg/liter (USEPA 1980). In studies of two species of saltwater phytoplankton, EC50 values of 160 and 175 µg/liter, based on growth inhibition, were reported (USEPA 1980).

EPA has established an ambient water quality criterion of 10 µg/liter for the protection of human health from the toxic properties of cadmium through ingestion of contaminated water and contaminated aquatic organisms.

EPA has established an aquatic life water quality criterion for freshwater species of 0.012 µg/liter (24-hour average), with a maximum not to be exceeded of 1.5 µg/liter. For saltwater species, the 24-hour average criterion is 4.5 µg/liter, with a maximum not to be exceeded of 59 µg/liter.

Chromium

The toxicity of chromium varies according to its valence state. The hexavalent and trivalent moieties are the biologically significant forms. Hexavalent chromium has long been recognized as a toxic substance, while trivalent chromium is considered to be relatively innocuous (NAS 1977).

Chromium has been shown to cause a variety of toxic effects. Certain chromium (VI) compounds are carcinogenic in humans (IARC 1980, USEPA 1980, NAS 1974, as cited in USEPA 1980). In occupational exposures, dermatitis, irritation of mucous membranes, injury to nasal tissue, changes in pulmonary dynamics, lung cancer, and renal and hepatic toxicity have been observed (NAS 1974, Borett et al. 1977, Mancuso 1951, Bloomfield and Blum 1928, USPHS 1953, and IARC 1980, as reported in USEPA 1980).

In animals, acute toxicity has been observed only when high doses were administered. For example, rats tolerated hexavalent chromium in drinking water at 25 ppm for 1 year, and dogs showed no effect from chromium as potassium chromate at 0.45-11.2 ppm over a 4-year period (NAS 1974, as reported in NAS 1977). Chromates, however, have been shown to be mutagenic in a wide variety of test systems (USEPA 1980). Chromium compounds also caused terata in hamsters (IARC 1980). With regard to carcinogenicity, intraosseous, intramuscular, subcutaneous, intrapleural, and intraperitoneal injections of chromium compounds produced tumors at the site of administration in rabbits, mice, and rats (NAS 1977). Intramuscular administration of lead chromate in rats produced renal carcinomas (IARC 1980).

Acute toxicity data for hexavalent chromium are available for 13 freshwater animal species; 96-hour LC50 values range from 67 µg/liter for a scud to 59,900 µg/liter for a midge. In saltwater species 96-hour LC50 values of hexavalent chromate range from 2,000 µg/liter for polychaete annelids and mysid shrimp to 105,000 µg/liter for the mud snail. Soft water 96-hour LC50 values for hexavalent chromium range from 17.6 mg/liter for fathead minnows to 118 mg/liter for bluegill; hard water 96-hour LC50 values for hexavalent chromium range from 27.3 mg/liter for fathead minnows to 133 mg/liter for bluegill. 96-Hour LC50 trivalent chromium values (chromium potassium sulfate) range from 3.33 mg/liter for guppies to 7.46 mg/liter for bluegill in soft water. The LC50 for fathead minnows exposed to potassium chromate in soft water was reported to be 45.6 mg/liter. Hexavalent chromium was also found to significantly reduce the growth and survival of chinook salmon at concentrations of 0.2 mg/liter. Chronic toxicity of hexavalent chromium was observed in the polychaete worm at concentrations ranging from 17

to 38 µg/liter; the mysid shrimp at concentrations ranging from 88 to 198 µg/liter; the rainbow and brook trout at concentrations ranging from 200 to 350 µg/liter; and the fathead minnow at concentrations ranging from 1,000 to 3,950 µg/liter. Algae and the giant kelp were affected at concentrations ranging from 1,000 to 5,000 µg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 50 µg/liter for the protection of human health from the toxic properties of chromium (VI) through ingestion of water and contaminated aquatic organisms. A human health criterion of 170 mg/liter was established for chromium III.

EPA has established an aquatic life water quality criterion for freshwater species of 0.29 µg/liter (24-hour average) for chromium VI, and a criterion of 2200 µg/liter (maximum level not to be exceeded) for chromium III. The aquatic life water quality criterion for saltwater species is 18 µg/liter (24-hour average) for chromium VI.

Copper

Copper produces a metallic taste in the mouth, nausea, vomiting, epigastric pain, diarrhea, and depending on the severity, jaundice, hemolysis, hemoglobinuria, hematuria, and oliguria. In severe cases, hepatic necrosis, gastrointestinal bleeding, anuria, hypotension, tachycardia, convulsions, and coma can occur (USEPA 1980). The toxic intake of inorganic copper for an adult male was reported to be greater than 15 mg per dose (Burch et al. 1975, as reported in USEPA 1980). Chronic oral exposure to copper has resulted in behavioral changes, diarrhea, and progressive marasmus in an infant (Salmon and Wright 1971, as reported in USEPA 1980).

Chronic toxicity in animals varies considerably in different species. Sheep are highly susceptible while rats are resistant to the effects of copper (USEPA 1980). Copper poisoning has been reported in swine at levels of 250 µg/g in the diet (Suttle and Mills 1966, as reported in USEPA 1980). Hepatic hemosiderosis developed in swine and rats fed copper acetate in a chronic oral feeding study (Mallory and Parker 1931, as reported in USEPA 1980). No information is available on the teratogenicity or mutagenicity of copper, although one report suggested that copper may increase the mutagenic activity of other compounds. The carcinogenic potential of copper has not been established (USEPA 1980).

Acute toxicity testing on copper has been conducted with 45 freshwater species and chronic tests with 15 species. Acute toxicity levels range from 0.0072 mg/liter for cladocerna in soft water to 10.2 mg/liter for the bluefish in hard water. Toxicity appears to decrease as the hardness of the water increases. Additional data for several species indicate that toxicity also decreases with increasing alkalinity and total organic carbon. Among the more sensitive species are daphnids, scuds, midges, and snails, which form the major food webs for both warm and cold water fish (USEPA 1980).

The acute toxicity of copper to saltwater animals ranges from 17 µg/liter for a Calonoid copepod to 600 µg/liter for the shore crab. A

chronic lifecycle test on mysid shrimp showed adverse effects at 77 µg/liter. Saltwater algae were adversely affected in concentrations ranging from 5 to 100 µg/liter. Oysters have been reported to bioaccumulate copper up to 28,200 times the ambient concentration. In long-term exposures, the bay scallop was killed at 0.005 mg/liter (USEPA 1980).

Chronic toxicity values for 15 species ranged from a low of 3.9 µg/liter for brook trout to 6.4 µg/liter for the northern pike. The two most sensitive species, bluntnose minnow and G. pseudolimnia, are both important food organisms. Copper toxicity has been evaluated on a wide range of plant species, with results similar to those for animals. Bioaccummulation does not appear to occur often in the edible portion of freshwater aquatic species (USEPA 1980).

EPA has not yet established ambient water quality criteria for the protection of human health from the toxic effects of copper because of the lack of sufficient information. However, using organoleptic data for controlling undesirable taste and odor of ambient water, the estimated level is 1 mg/liter.

The aquatic life water quality criterion for freshwater species is 5.6 µg/liter (24-hour average), with a level not to be exceeded of 12 µg/liter. For saltwater species, the 24-hour average criterion is 4 µg/liter; the maximum level not to be exceed is 23 µg/liter.

Cyanide

Hydrogen cyanide and its alkali metal salts are extremely toxic to humans and other mammals (USEPA 1980). By ingestion, the mean lethal dose of these substances is estimated to range from 50 to 200 mg for humans with death occurring generally within 1 hour (Gosselin et al. 1976, as reported in USEPA 1980). Inhalation of hydrogen cyanide gas at 0.1-0.3 mg/liter has caused death in 10-60 minutes in humans (Prentiss 1937 and Fassett 1963 as reported in USEPA 1980). The acute effects of cyanide poisoning mostly result from inhibition of cytochrome C oxidase, resulting in a blockage of oxidative metabolism and phosphorylation (Gosselin et al. 1976, as reported in USEPA 1980). The organ systems most profoundly affected are the heart and brain because of their high dependence on oxidative metabolism. Cyanide poisoning can also cause increased blood pressure (Heymans and Neil 1958, as reported in USEPA 1980). Exposure of humans to small amounts of cyanide compounds over long periods of time is reported to cause loss of appetite, headache, weakness nausea, dizziness, and symptoms of toxicity in the upper respiratory tract and eyes (Sax 1975).

Despite the high acute toxicity of cyanide, chronic exposure to sublethal doses does not appear to have serious adverse effects (USEPA 1980). Animal studies with dogs administered 0.5-2 mg/kg sodium cyanide once or twice each day for 15 months showed no evidence of pathophysiological changes in organ function or permanent alteration in intermediary metabolism (Hertting et al. 1960, as reported in USEPA 1980). In other studies rats were fed a potassium cyanide mixture equivalent to the LD50 for 25 days and dogs were fed 150 ppm sodium cyanide for 30 days without observing significant adverse effects

(Hayes 1967 and American Cyanamid 1959, as reported in USEPA 1980). No information is available on the mutagenicity and carcinogenicity of cyanides (USEPA 1980). However, thiocyanate, the major metabolic product of cyanide, has produced, in vivo, developmental abnormalities in the chick and ascidian embryo. (Nowinski and Pandra 1946 and Ortolani 1969, as reported in USEPA 1980)

The acute toxicity of cyanide to aquatic organisms has been demonstrated in over 35 species of fresh and saltwater animals. The 96-hour LC50 values range from 30 µg/liter for the saltwater copopod to 2326 µg/liter for the freshwater isopod. Most values, however, cluster between 50 and 200 µg/liter. Teratogenic effects have been observed in the Atlantic salmon, and reproductive disturbances have occurred in the bluegill, fathead minnow, brook trout, and rainbow trout at concentrations ranging from 5.4 to 62 µg/liter (USEPA 1980).

Chronic toxicity has been observed in the brook trout, fathead minnow, the bluegill, the isopod, and the scud in concentrations ranging from 8 to 34 µg/liter, depending on the species. Reduced swimming capacity was observed in the brook trout at 10 µg/liter, in rainbow trout at 20 µg/liter, and in the Cichlasoma binaculatum at 40 µg/liter. Aquatic plants are much more resistant to the effects of cyanide; effects are not observed until concentrations reach 3,000 µg/liter (USEPA 1980).

EPA has established an ambient water quality criterion of 200 µg/liter for the protection of human health from the toxic properties of cyanide through ingestion of water and contaminated aquatic organisms.

EPA has established an aquatic life water quality criterion for freshwater species of 3.5 µg/liter (24-hour average), with a maximum not to be exceeded of 52 µg/liter. EPA has not yet set a criterion for saltwater species.

Lead

Acute inorganic lead intoxication is rare (Casarett and Doull 1975). The most serious effects of chronic exposure in humans are seen in the hematopoietic system (decreased heme synthesis), nervous system (encephalopathy), and renal system (NAS 1977 and USEPA 1980). Blood lead concentrations of 25-30 µg/day in children and women and 35-40 µg/day in men have been associated with statistically significant increases in red-cell protoporphyrin (Zielhuis 1975, as reported in USEPA 1980). The noeffect concentration of lead in the blood on the developing human nervous system has been estimated at 55-60 µg/day whole blood (NAS 1977). Blood-lead concentrations in excess of about 50-60 µg/day have been associated with spermatoxic effects in men (Lancranjan et al. 1975, as reported in USEPA 1980). Lead has not been shown to be carcinogenic in humans, although one researcher has questioned the statistical methodology used in the epidemiological studies (Kanj et al. 1980, as reported in USEPA 1980).

Certain lead compounds have been found to produce tumors in some species of experimental animals. For example, in a 2-year feeding study on rats, lead acetate at concentrations of 1,000 ppm or more was found to induce renal

tumors; furthermore, the number of tumor bearing animals was dose dependent (Azar et al. 1973, as reported in USEPA 1980). Lead has been associated with teratogenic effects in chick embryos and rodents (USEPA 1980). Teratogenic effects were seen in the offspring of rats that had received a single intraperitoneal dose of 25-70 mg/kg on day 9 of gestation. Administration later in pregnancy induced fetal resorption without teratogenic effects (McClain and Beeker 1975, as reported in USEPA 1980). Chronic administration of lead in the drinking water of pregnant rats at concentrations up to 250 mg/liter was found to delay fetal development and increase fetal resorption; no teratogenic effects were seen (Kimmel et al. 1976, as reported in USEPA 1980).

Lead has been shown to be acutely toxic to freshwater animals over a range of concentrations from 124 to 542,000 µg/liter, depending on the species tested and the hardness of the water. The acute toxicity values for saltwater invertebrates ranged from 668 µg/liter to 27,000 µg/liter. Chronic tests have been conducted with two invertibrate species and six fish species with the chronic values ranging from 12 µg/liter for Daphnia magna to 174 µg/liter for the white sucker (USEPA 1980). Concentrations as low as 500 µg/liter were found to inhibit the growth of freshwater algae (USEPA 1980).

EPA has established an ambient water criterion of 50 µg/liter for the protection of human health from the toxic properties of lead through ingestion of water and contaminated aquatic organisms.

EPA has established an aquatic life water quality criterion for freshwater species of 0.75 µg/liter (24-hour average), with a maximum level not to be exceeded of 74 µg/liter.

Mercury

Elemental mercury is extremely toxic but is very poorly absorbed from the gastrointestinal tract. The oral toxicity of inorganic mercury salts depends on their solubility. Elemental mercury is transformed biochemically in bottom sediments to methyl mercury or other organic mercurial compounds (USEPA 1976). The organic form readily enters the food chain with concentration factors as great as 3,000 in fish (Hannerz 1968, as reported in USEPA 1976).

Acute poisoning in man has resulted from exposure to mercury vapor in concentrations ranging from 1.2 to 8.5 mg Hg/cu m with symptoms related to pulmonary effects (Casarett and Doull 1975). Chronic exposure to mercury vapor results in central nervous system effects including psychic and emotional disturbances, increased irritability, combativeness, defective patterns, ocular disturbances, and tremors. Kidney and gastrointestinal disturbances are often associated with chronic mercury exposure (AIHA 1966, as reported by Casarett and Doull 1975).

Symptoms of acute inorganic mercury poisoning include pharyngitis, gastroenteritis, vomiting followed by ulcerative hemorrhagic colitis, nephritis, hepatitis, and circulatory collapse (USEPA 1976). Renal toxicity occurs with chronic exposure to inorganic mercury (Casarett and Doull 1975).

The toxicity of alkyl mercury compounds, particularly methyl mercury, differs significantly from other organic mercurials. In addition to the environmental transformation of other forms of mercury to methyl mercury, this compound is readily absorbed through the lungs, and skin and gastrointestinal absorption under most circumstances is nearly complete (Casarett and Doull 1975). Methyl mercury will also readily cross the placental barrier causing fetal toxicity. In humans methyl mercury poisoning has produced fatalities, birth defects, and severe central nervous system effects (NAS 1977). The ingestion of fish containing mercury in Minamata, Japan, and the ingestion of wheat seed treated with phenyl mercuric acetate resulted in similar toxic effects including mental disturbance, ataxia, speech disturbances, hearing impairment, constriction of visual fields, increased tendon reflex, and involuntary movement. Hypoplasia and atrophy of the brain tissue have also been reported (Casarett and Doull 1975).

The toxicity of inorganic mercury to freshwater aquatic organisms was demonstrated in nine taxonomic orders from rotifers to fish. Acute toxicity values ranged from 0.02 to 2,000 µg/liter. The acute toxicity of mercuric chloride was reported for 26 species of saltwater animals including annelids, molluscs, crustaceans, echinoderms, and fishes. Species mean acute values range from 3.5 to 1,680 µg/liter. Fish are more resistant while molluscs and crustaceans are more sensitive. The acute toxicity of methyl mercury and other mercury compounds is available only for fish, limiting an estimate of the range of species sensitivity to the compound. Methyl mercury is the most toxic of the mercury compounds with chronic values for cladoceran and brook trout being 1.0 and 0.52 µg/liter, respectively. For inorganic mercury the chronic value for cladoceran is reported to be 1.6 µg/liter. Concentrations that affected growth and photosynthetic activity of one saltwater diatom and six species of brown algae range from 10 to 160 µg/liter. Adverse effects on reproduction of the mysid shrimp occurred at a concentration of 1.6 µg/liter. A bioconcentration factor of 40,000 was reported for methyl mercuric chloride in the oyster. In freshwater organisms, a bioconcentration factor of 23,000 was reported for inorganic mercury (USEPA 1980).

EPA has established a water quality criterion of 144 ng/liter for the protection of human health from the toxic effects of mercury ingested through water and contaminated aquatic organisms.

EPA has established an aquatic life water quality criterion for freshwater species of 0.20 µg/liter (24-hour average), with a maximum level not to be exceeded of 4.1 µg/liter. For saltwater species, the 24-hour average criterion is 0.10 µg/liter, with a maximum not be exceeded of 3.7 µg/liter.

Nickel

A significantly increased incidence of cancers of the lungs and nasal cavities has been found in epidemiologic studies of workmen in nickel smelters and refineries (NAS 1977). The implicated compounds are nickel subsulfide, nickel oxides, nickel carbonyl vapor, and soluble aerosols of nickel sulfate, nitrate, or chloride (NAS 1980). Toxic effects depend on the nickel compound to which a subject is exposed. Acute nickel carbonyl poisoning results in

immediate symptoms of headache, nausea, and insomnia, followed by constrictive chest pains, hyperpnea, cyanosis, and severe weakness (Sunderman 1970, Vuopala 1979, as reported in USEPA 1980). Nickel carbonyl exposures have also been associated with nephrotoxicity (Blandes 1934, Carmichael 1953, as reported in USEPA 1980). Exposure to occupational sources of nickel and exposure to such nickel-containing items as jewelry and tools have been associated with a characteristic nickel dermatitis (USEPA 1980).

Animal studies have shown that the oral toxicity of nickel and nickel salts is relatively low, but that parenteral injections of nickel salts are much more toxic (NAS 1977). The major symptoms of acute nickel toxicity are hyperglycemia and gastrointestinal and central nervous system effects (NAS 1977). One researcher found malformations in hamster embryos when the mother was exposed to unidentified nickel compounds administered parenterally at dosages ranging from 0.7 to 10.0 mg/kg (Ferm 1972, as reported in USEPA 1980). No teratogenic effects were seen when either nickel chloride (16 mg/kg) or nickel subsulfide (80 mg/kg) was administered to rats (Sunderman et al. 1978, as reported in USEPA 1980). Multigenerational reproductive studies have linked nickel to decreases in litter size, increased numbers of runts, and increased neonatal mortality (USEPA 1980). Male rats given daily oral doses of nickel sulfate at 25 mg/kg were completely sterile after 120 days (Watschewa et al. 1972, as reported in USEPA 1980). Several nickel-containing substances including nickel dust, nickel subsulfide, nickel oxide, nickel carbonyl, and nickel bicyclopentadiene have been found to be carcinogenic in animals upon inhalation or parenteral administration (NAS 1977).

The toxicity of nickel to freshwater animals decreases with increasing hardness of the water. For example, the LC50 for Daphnia magna was 510 µg/liter at a water hardness of 45 mg of calcium carbonate/liter. By comparison, the LC50 for the fathead minnow was 5.21 mg/liter at 45 mg of calcium carbonate/liter. In several life cycle or early life stage studies in freshwater fish, the fathead minnow, chronic toxicity was observed at concentrations ranging from 109 to 527 µg/liter (USEPA 1980). Nickel has also been shown to reduce the growth of several freshwater algae species at concentration ranging from 100 to 700 µg/liter. The LC50 values for saltwater animal species ranged from 152 µg/liter for mysid shrimp to 350,000 µg/liter for the mummichog fish. Growth reductions have been reported for a species of saltwater algae (USEPA 1980).

EPA has established an ambient water criterion of 13.4 µg/liter for the protection of human health from the toxic properties of nickel through ingestion of contaminated water and contaminated aquatic organisms.

EPA has established an aquatic life water quality criterion for nickel. For freshwater species, the 24-hour average criterion is 0.20 µg/liter, with a maximum not to be exceeded of 4.1 µg/liter. The 24-hour average criterion for saltwater species is 0.10 µg/liter, with a maximum not to be exceeded of 3.7 µg/liter.

Selenium

Although elemental selenium is relatively nontoxic, the soluble salts of selenium dioxide, selenium trioxide, some halogen compounds, and especially hydrogen selenite are toxic in humans (NAS 1977). Acute human exposure generally produces irritation to the eyes, skin, and mucous membranes and nausea, headaches, and a variety of respiratory disorders (NAS 1977). Chronic human exposures have produced such symptoms as depression, nervousness, occasional dermatitis, and gastrointestinal disturbance (NAS 1977). In addition, epidemiologic studies of children indicated a relationship between increased incidence of dental caries and consumption of small amounts of selenium while the teeth were developing; similar results have been seen in experimental studies of rats (NAS 1977).

Acute and chronic selenium toxicity have been observed experimentally in laboratory animals and also in domestic animals consuming plants with a high selenium content. Selenium disease in domestic animals, in its most serious form, first manifests itself by impaired vision, then paralysis, and ultimately death by respiratory failure (USEPA 1980). The concentrations of selenium in the diet which will produce chronic toxic effects depend on the chemical form of the selenium and other dietary components. (Fishbein 1977, as reported in USEPA 1980). In a chronic feeding study, young rats treated with sodium selenite showed growth depression when the diet contained 6.4 ppm selenium or more. Animals receiving concentration of 8 ppm or more died after the fourth week and showed enlargement of the pancreas, reduction of hemoglobin content, and increased serum bilirubin (Halverson et al. 1966, as reported in NAS 1977). Selenium has not been positively established as a carcinogen. Although some studies have reported increased tumor incidences in animals fed selenium, these results have not been sufficiently documented and are not in accord with other studies (NAS 1977). No reports of mutagenicity by selenium compounds are available. Selenium has been shown to be teratogenic in chick embryo tests, even when exposure is at low concentrations (NAS 1977). Malformations have also been seen in domestic mammals, but they were not reproduced in the only available study on laboratory animals in which hamsters received a near lethal intravenous dose of 2 mg/kg sodium selenite (Holmberg and Ferm 1969, as reported in USEPA 1980 and NAS 1977). In reproduction tests on rats, selenium has been associated with decreases in fertility and pup survival (NAS 1977).

Selenium is acutely toxic to aquatic invertebrates and to fish. Acute toxicity data for inorganic selenite is available for 13 species of freshwater animals and ranges from 340 µg/liter for the scud to 42,000 µg/liter for the midge. For selenate, acutely toxic concentrations range from 760 µg/liter for the scud to 12,500 µg/liter for the fathead minnow. Chronic toxicity for selenite and selenate compounds in freshwater organisms range from 88 µg/liter for rainbow trout to 690 µg/liter for *Daphnia magna* (USEPA 1980). Selenium compounds have also been shown to be toxic to aquatic and terrestial plants (USEPA 1980).

EPA has recommended an ambient water quality criterion of 10 ug/liter for the protection of human health from the toxic properties of selenium through ingestion of contaminated water and contaminated aquatic organisms (USEPA 1980).

EPA has established an aquatic life water quality criterion for freshwater species of 35 µg/liter (24-hour average), with a maximum not to be exceeded of 260 µg/liter. For saltwater species, the 24-hour average criterion is 54 µg/liter, with a maximum not to be exceeded of 410 µg/liter.

Silver

Silver exhibits moderate toxicity in humans. Acute oral doses of silver nitrate can cause abdominal pain and rigidity, vomiting, and convulsions. Exposure information for the case reports of acute poisonings is generally scanty. However, ingestion of 10 g is usually fatal (USEPA 1980).

The most common effect of chronic human exposure to silver is argyria (either generalized or localized) resulting from medical or occupational exposure. Generalized argyria is characterized by slate gray pigmentation of the skin, hair, conjunctiva of the eye, and internal organs resulting from deposition of silver in tissue. In severe cases of argyria, the respiratory tract may be affected. In localized argyria, only limited areas are pigmented, and in a condition called argyrosis, the tissues of the eye are pigmented (Clayton and Clayton 1981, Doull et al. 1980, and USEPA 1980). With improved work conditions, no cases of argyria from industrial exposures have been reported since the 1930's (Clayton and Clayton 1981).

Acute toxicity in experimental animals is associated predominantly with intravenous administration. Dogs injected with approximately 32 mg Ag/kg (as silver nitrate) in the pulmonary system developed edema, myocardial ischemia and lesions, and hypertension. When inorganic silver compounds were injected into animals intravenously, effects were primarily on the central nervous system (Hills and Pillsbury 1939, as reported in USEPA 1980). Large doses of colloidal silver administered intravenously have produced death due to pulmonary edema and congestion (USEPA 1980).

In subchronic and chronic animal studies, the predominant effects of silver administration have been on conditioned reflex activity and on the kidney. Klein et al. (1978, as reported in USEPA 1980) reported hemorrhages in the kidneys of rats given silver at 0.4 mg/liter drinking water for 100 days. At 1 mg/liter, changes in both the kidney and liver were observed (form of the metal unspecified). Several investigators have reported effects on conditioned reflex activity in rats given silver in drinking water (form of the metal unspecified) at concentrations between 0.5 and 20 mg/liter for periods of 1 to 11 months (Barkov and El'piner 1968, Kharchenko and Stepanenko 1972, and Zapadnyuk et al. 1973, as reported in USEPA 1980). Brain nucleic acid content in rats was also reduced at 0.5 mg/liter drinking water (form of the metal unspecified) (Kharchenko et al. 1973, as reported in USEPA 1980). In several studies, implanted foils and disks and injected colloidal suspensions of metallic silver have been found to produce tumors or hyperplasia. These effects are considered to be due to the particular form of the metal or to its being an exogenous irritant. Thus, silver is not considered to be carcinogenic (USEPA 1980).

In natural waters, silver exists primarily in the 0 and +1 oxidation states; the monovalent species is the form of greatest environmental concern. Silver is one of the most toxic metals to freshwater aquatic life. For invertibrate species, acute toxicity values range from 0.25 µg/liter for Daphnia magna to 4,500 µg/liter for the scud Gammarus pseudolimnaeus. Acute toxicity values for fish range from 3.9 µg/liter for the fathead minnow in soft water to 280 µg/liter for rainbow trout in hard water (USEPA 1980). In an 18-month study conducted by Davies et al. (1978, as reported in USEPA 1980) with freshwater trout, the rate of growth was decreased in fish exposed for two months at a concentration of 0.17 µg/liter, and mortality was 17 percent greater than the control in this dose group at the study end.

Acute toxicity values for saltwater organisms ranged from 4.7 µg/liter for the summer flounder to 1,400 µg/liter for the sheepshead minnow (USEPA 1980). In a life-cycle toxicity study with mysid shrimp, brood size was smaller than the control at a concentration of 33 µg/liter of silver (Lussier and Gentile 1980, as reported in USEPA 1980).

In various strains of freshwater algae, growth inhibition has been reported at silver concentrations ranging from 30 to 200 µg/liter (USEPA 1980). Phytotoxicity was reported in duckweed at a silver concentration of 270 µg/liter, and in waterweed at a concentration of 7,500 µg/liter (Brown and Rattigan 1979, as reported in USEPA 1980). In saltwater algae, reduced cell numbers were reported at 130 µg/liter (USEPA 1978, as reported in USEPA 1980).

EPA has established an ambient water quality criterion of 50 µg/liter for the protection of human health from the toxic properties of silver through ingestion of contaminated water and aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for silver. However, EPA has set maximum limits not to be exceeded of 1.2 µg/liter for freshwater organisms and 2.3 µg/liter for saltwater organisms.

Thallium

Numerous cases of thallium poisonings have been recorded, largely as a result of thallium's medicinal or rodenticidal uses. Minimum toxic doses in humans of between 3 and 15 mg Tl/kg have been reported (Clayton and Clayton 1981). Acute poisoning is characterized by gastrointestinal irritation, acute ascending paralysis, psychic disturbances, alopecia, and abnormalities of cardiac function (Clayton and Clayton 1981, Doull et al. 1980, and USEPA 1980). Autopsies in fatal cases have revealed damage to the gastric and intestinal mucosa, fatty infiltration of the liver and kidneys, and damage to the adrenal glands and central nervous system (Clayton and Clayton 1981). In the few reported cases of subchronic and chronic poisoning in humans, symptoms are similar to those for acute poisoning (USEPA 1980).

In animal studies, the acute toxicity of thallium compounds exhibits a particularly narrow range. Of 14 inorganic thallium compounds administered by various routes to five animal species, the lowest lethal doses or LD50 values

ranged from about 15 to 50 mg Tl/kg (Clayton and Clayton 1981). In a 90-day feeding study in rats, doses as low as 30 to 35 ppm in the diet produced marked growth depression after 30 days. The major histological change at these dietary levels was atrophy of the hair follicles and sebaceous glands of the skin. At 15 ppm in the diet, the major effect was alopecia (Downs et al. 1960, as reported in Clayton and Clayton 1981). Evidence for the teratogenicity of thallium is inconclusive. Thallium was administered to pregnant rats on gestation days 8-10 at 2.5 mg/kg/day or on days 12-14 at 2.5 and 10 mg/kg/day; both dose levels caused maternal toxicity. In the offspring, reduced fetal weight and increased incidences of hydronephrosis and missing or non-ossified vertibrae were reported (Gibson and Becker (1970, as reported in USEPA 1980). Dwarfism (achondroplasia) in rats has also been described as a teratogenic response to thallium salts (Nogami and Terashima 1973, as reported in Doull et al. 1980). Information is not available on the carcinogenicity of thallium; however, thallium has shown some mild anti-carcinogenic effects in experimental animals (USEPA 1980).

Acute toxicity of thallium to freshwater organisms is reflected in mean LC50 values for Daphnia magna of 1,400 µg/liter, for the fathead minnow of 1,800 µg/liter, and for the bluegill of 126,000 µg/liter (Dawson et al. 1977, Kimball manuscript, and USEPA 1978, as reported in USEPA 1980). Chronic effects have been observed in Daphnia magna at 100-181 µg/liter and in the fathead minnow at 40-81 µg/liter (Kimball manuscript, as reported in USEPA 1980).

In saltwater species, an LC50 value of 2,130 µg/liter has been reported for the mysid shrimp. The sheepshead minnow and tidewater silverside were less sensitive to thallium with 96-hour LC50 values of 20,900 and 24,000 µg/liter, respectively (USEPA 1978 and Dawson et al. 1977, as reported in USEPA 1980). Chronic effects were observed in the sheepshead minnow at concentrations between 4,300 and 8,400 µg/liter (USEPA 1978, as reported in USEPA 1980).

In freshwater algae, a 50% reduction in chlorophyll a and cell numbers was observed at 100-110 µg/liter (USEPA 1978, as reported in USEPA 1980). A 50% inhibition of photosynthesis at 4,080 µg/liter was reported for saltwater algae (Overnell 1975, as reported in USEPA 1980).

EPA has established an ambient water quality criterion of 13 µg/liter for protection of human health from the toxic properties of thallium through ingestion of contaminated water and aquatic organisms.

EPA has not yet established an aquatic life water quality criterion for thallium.

Zinc

Zinc salts are astringent, corrosive to the skin, and irritating to the gastrointestinal tract. Accidental oral poisonings in humans have produced such symptoms as fever, vomiting, stomach cramps, and diarrhea (Patty 1963). Occupational exposure to zinc oxide may result in a characteristic short-term syndrome. It generally occurs after a lapse of exposure, for example on

Monday mornings or after a holiday. The symptoms include chills and fever followed by remission after 24-48 hours, despite continued exposure (Casarett and Doull 1975). Zinc chloride fumes may give rise to a grey cyanosis, dermatosis, and ulceration of the nasal passages and in severe cases may result in acute pulmonary damage or death (Casarett and Doull 1975).

In feeding tests on rats, no adverse effects were observed at dosages up to 5,000 mg/kg. At 10,000 mg/kg, however, a cessation of growth and some deaths were seen (Rothstein 1953, as reported in CSWRCB 1963). No studies are available showing zinc to be teratogenic, mutagenic, or carcinogenic.

Zinc has been shown to be acutely toxic to freshwater animals over a range of concentrations from 0.090 to 58.1 mg/liter, depending on the species tested and the hardness and temperature of the water. In saltwater animals, the range was from 0.166 mg/ liter to 83 mg/liter. Zinc concentration from 0.030 to 21.65 mg/liter have been shown to reduce the growth of various freshwater plant species. A range of 0.050 to 25.5 mg/liter was found to inhibit growth in several saltwater plant species (USEPA 1980).

EPA has not yet established ambient water quality criteria for the protection of human health from the toxic properties of zinc because of the lack of sufficient data. However, using available organoleptic data for controlling undesirable taste and odor of ambient water, the estimated level is 5 mg/liter.

EPA has established an aquatic life water quality criterion for zinc. For freshwater species, the 24-hour average criterion is 47 µg/liter, with a maximum not to be exceeded of 180 µg/liter. The 24-hour average criterion for saltwater species is 58 µg/liter, with a maximum not to be exceeded of 170 µg/liter.

5. Polychlorinated Biphenyls

The toxic effects of polychlorinated biphenyls (PCBs) in humans are well-established. Most human exposures have occurred as a result of the episode in Japan in 1968 resulting from ingestion of rice oil contaminated with Kanechlor 400 or from industrial exposure to PCBs (USEPA 1980). In the Japanese poisoning incident, initial symptoms included eye discharge, acneform eruptions, pigmentation of the skin, dermatologic problems, swelling, jaundice, numbness of limbs, spasms, hearing and vision problems, and gastrointestinal disturbances. Blood changes were noted and liver biopsies revealed histopathological changes. It has been estimated that the average amount of PCB ingested by those affected was 2 g (Kuratsune et al. 1972, as reported in USEPA 1980). During 1968, ten live and two stillborn infants were delivered to parents poisoned with PCBs. Nine of the ten had abnormally pigmented skin. Birth weight and growth of the children was significantly lower than Japanese national standards. In four babies, gingival hyperplasia, tooth eruption at birth, bone abnormalities, facial edema, and exophthalmic eyes were also observed (Yamashita 1977, as reported in USEPA 1980). Many symptoms, reported in affected individuals four years after the poisoning episode, were highly persistent.

In an occupational setting in which PCB air levels were reported to be 5.2 to 6.8 mg/cu m, three cases of severe chloracne have been reported (Puccinelli 1954, as reported in USEPA 1980). Laboratory workers exposed to breathing zone concentrations of 0.014 to 0.073 mg/cu m complained of dry sore throat, skin rash, gastrointestinal disturbances, eye irritation, and headache (Levy et al. 1977, as reported in USEPA 1980). Changes in a liver function test (increased antipyrene clearance) was observed in workers occupationally exposed to PCBs for at least four years (Alvares et al. 1977, as reported in USEPA 1980).

In acute animal studies, PCBs are only slightly toxic. Oral LD50 values for the rat range from 0.79 to 3.17 g/kg (USEPA 1980). Toxic effects of acute doses of Aroclor 1242 include diarrhea, chromoacryorrhea, weight loss, unusual stance and gait, CNS deterioration, and histopathologic changes of the liver and kidney (USEPA 1980). The more significant toxic effects of PCBs are observed as a result of repeated exposures and are similar to those observed in humans. Adult Rhesus monkeys are particularly sensitive to PCBs. Aroclor 1248 at 100 or 300 ppm in the diet for 2 to 3 months (total intakes of 0.8-1.0 g and 3.6-5.4 g, respectively) caused high morbidity within one month and almost 100 percent mortality within three months (Allen 1975, as reported in USEPA 1980). Pathological changes of the liver are the most consistent changes occurring in mammals after exposure to PCBs. In one study by Kimbrough et al. (1972, as reported in USEPA 1980), rats fed Aroclor 1254 or 1260 at levels between 20 and 1,000 ppm for eight months showed histopathologic changes of the liver and porphyria. Adenofibrosis of the liver was observed at the higher doses. Liver pathology similar to that seen in rats has been reported in mice exposed to 1.5 mg PCB/day (Nishizumi 1970, as reported in USEPA 1980). PCB applied dermally to rabbits results in skin lesions and pathological changes in the liver and kidney (Vos and Beems 1971, as reported in USEPA 1980). Female Rhesus monkeys fed low levels of Aroclor 1248 (2.5 and 5 ppm) for 52 weeks developed periorbital edema, alopecia, erythema, and acneform lesions (Barsotti and Allen 1975, as reported in USEPA

1980); effects in males was less pronounced. Induction of liver microsomal enzymes has been demonstrated at dietary levels as low as 0.5 to 25 ppm. Other systemic effects in experimental animals include porphyria, increased thyroxin metabolism, ultrastructural changes in the thyroid, immunosuppression, and alterations in steroid metabolism (USEPA 1980). Administration of PCBs has resulted in adverse reproductive effects in rats, rabbits, mice, mink, and Rhesus monkeys. An increased estrus cycle and a decreased rate of implantation were observed in mice treated for ten weeks with 0.025 mg/day Clophen A60 (Orberg and Kihlstrom 1973, as reported in USEPA 1980). In a study of female Rhesus monkeys fed 2.5 or 5.0 ppm Aroclor 1248 in the diet for six months before mating, Barsotti and Allen (1975, as reported in USEPA 1980) observed a decreased rate of conception, live births, and neonatal body weights and an increase in neonatal deaths. The carcinogenicity of PCBs has been demonstrated in mice and rats. Liver tumors were reported in mice given PCB in the diet at levels of 500 ppm for 224 days (Ito et al. 1973, as reported in USEPA 1980) and 300 ppm for 330 days (Kimbrough and Linder 1974, as reported in USEPA 1980). Kimbrough et al. (1975, as reported in USEPA 1980) fed Aroclor 1260 to rats at levels of 100 ppm for 21 months and found hepatocellular carcinomas in 26/184 experimental animals but only one out of 173 controls. A 1978 National Cancer Institute bioassay (as reported in USEPA 1980) concluded that Aroclor 1254 was not carcinogenic in Fischer 344 rats, although a high frequency of hepatocellular proliferative lesions and an increase in carcinomas of the gastrointestinal tract (not statistically significant) were considered possibly associated with treatment.

The acute toxicity of PCBs to freshwater organisms has been measured in three invertibrate species with acute values ranging between 10 and 2,400 µg/liter, and in four fish species with acute values ranging between 2 and 300 µg/liter (USEPA 1980). In eleven life-cycle or partial life-cycle tests with three vertibrate and two fish species, chronic effects were reported from 0.2 to 15 µg/liter (USEPA 1980). In saltwater species, the mean acute values for the eastern oyster, brown shrimp, and grass shrimp were 20, 10.5 and 12.5 µg/liter, respectively (USEPA 1980). Two chronic studies have been performed on the sheepshead minnow; chronic effects were observed at 0.098 to 7.14 µg/liter (USEPA 1980). Available data for saltwater plants indicate that unicellular plants are affected by concentrations similar to concentrations that are chronically toxic to animals, while freshwater algae are somewhat less sensitive to PCBs (USEPA 1980).

EPA has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects due to exposure to PCBs through ingestion of contaminated water and aquatic organisms. Since the zero level may not be attainable at the present time, a criterion of 0.079 ng/liter, corresponding to a lifetime incremental cancer risk of 0.000001, was recommended.

EPA has established an aquatic life water quality criterion for freshwater organisms of 0.014 µg/liter as a 24-hour average and for saltwater organisms of 0.030 µg/liter as a 24-hour average.

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APPENDIX I

FATE OF PRIORITY POLLUTANTS IN PUBLICLY OWNED TREATMENT WORKS

Executive Summary

United States
Environmental Protection
Agency

Effluent Guidelines Division
WH-552
Washington DC 20460

EPA 440/1-82/303
September 1982

Water and Waste Management

EPA

Fate of Priority Pollutants in Publicly Owned Treatment Works

Final Report

Volume I

FATE OF PRIORITY TOXIC POLLUTANTS

IN

PUBLICLY OWNED TREATMENT WORKS

FINAL REPORT

VOLUME I

PREPARED FOR:

EFFLUENT GUIDELINES DIVISION

OFFICE OF WATER REGULATIONS AND STANDARDS

U.S. ENVIRONMENTAL PROTECTION AGENCY

WASHINGTON, D.C. 20460

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PREFACE

This document is being issued as the Final Report on a project initiated by the United States Environmental Protection Agency in 1978 to study the occurrence and fate of the 129 priority toxic pollutants in 40 Publicly Owned Treatment Works (POTW) and a supplemental study conducted at 10 additional POTW. This report consists of two volumes. Volume I contains the background and purpose of study, the POTW selection criteria, the sampling program details, the overall POTW data, evaluation of analytical results, and the preliminary conclusions of the study. Volume II contains the Daily Analytical Results which embody the basic data generated during the course of this study and which are the source for all other data compilations and analyses.

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I.

SUMMARY, RESULTS AND CONCLUSIONS

SUMMARY

In 1978, the United States Environmental Protection Agency (EPA) initiated a program to study the occurrence and fate of the 129 priority pollutants in 40 Publicly Owned Treatment Works (POTWs). The first phase of this work was a two-plant pilot study designed to set operating parameters for the remainder of the 40 POTW study. In October 1979, EPA's Effluent Guidelines Division (EGD) published a report summarizing the findings of the pilot study work, "Fate of Priority Pollutants in Publicly Owned Treatment Works-Pilot Study," EPA 440/1-79-300. Upon completion of half of the POTW project EGD published a report summarizing the findings for the first 20 POTWs, "Fate of Priority Pollutants in Publicly Owned Treatment Works-Interim Report," EPA 440/1-80-301.

In this final report, data from all 40 POTWs plus 10 supplemental POTWs sampled under a parallel project are presented. At most of these plants, a minimum of 6 days of 24-hour sampling of influent, effluent and sludge streams was completed. Each sample was analyzed for conventional, selected non-conventional, and priority pollutants.

Beyond presenting the occurrence and concentration of priority pollutants in the 40 POTWs (and 10 supplemental POTWs) other specific phenomena and relationships are evaluated in this report. These items include:

- Impact of industrial contribution on influent quality
- Treatment or removal of priority pollutants in POTWs
- Reduction of priority pollutants by individual POTW treatment processes
- POTW priority pollutant mass balances
- Daily variation of influent pollutant concentrations
- Effect of rainfall on priority pollutant levels in POTW influents
- Formation of chlorinated hydrocarbons through chlorine disinfection
- Quantification of pollutants found in sludges but not detected in POTW influents
- Correlation of influent and effluent priority pollutant levels.

RESULTS AND CONCLUSIONS

1. A total of 102 priority pollutants were detected, at least once, in POTW influents.

2. In general, the higher the industrial contribution to a POTW, the higher the concentration of priority pollutants in the POTW influents.

3. Based on the 40 POTW data base, 50 percent of secondary treatment plants achieved a minimum of 70 percent reduction of total priority pollutant metals, 82 percent reduction of total volatile priority pollutants, and 65 percent reduction of the total base neutral priority pollutants.

4. Tertiary treatment processes reduced priority pollutants slightly better than secondary processes. Primary treatment was less effective than either secondary or tertiary processes. Activated sludge, trickling filter, rotating biological contactor and pure oxygen activated sludge processes were approximately equally effective in reducing priority pollutant concentrations.

5. At plants where the metal priority pollutant mass balance was good, some organic priority pollutants in the influent were not always accounted for in the effluent or sludges. This indicates that, in general, a portion of the organic priority pollutants are biodegraded or, in the case of volatiles, stripped out of the wastewater.

6. The mass loading of priority pollutants in POTW influents was higher on weekdays than on weekends. This was true for the metals, volatiles and base neutral priority pollutants.

7. Heavy rainfall increased metallic priority pollutant mass loading at POTWs.

8. Certain priority pollutant chlorinated hydrocarbons increased slightly in concentration during chlorine disinfection.

9. Some pollutants not measured in POTW influents were regularly measured at high levels in the corresponding sludge streams.

10. For many conventional and priority pollutants, as influent concentrations increased effluent concentrations also increased. This implies that the removal rates for the priority pollutants are relatively constant and a fixed percentage of incremental loadings of these pollutants will be removed by secondary treatment.

APPENDIX J

TREATMENT CATALOG FOR THE CATALYTIC COMPUTER MODEL

TREATMENT CATALOG
FOR THE CATALYTIC COMPUTER MODEL

Catalytic, Inc.
Philadelphia, Pennsylvania

September 1980

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TREATMENT CATALOG
FOR THE CATALYTIC COMPUTER MODEL

The Treatment Catalog defines the basis for applying the unit processes that are to be considered for treatment and disposal of wastewaters and their residues. The unit processes now in the catalog are not to be construed as an all-inclusive list of commercially available wastewater treatment processes; however, they are sufficiently comprehensive to provide a "typical" treatment method for any of the pollutants expected to be encountered.

GENERAL DESCRIPTION

Each unit process is represented by a separate entry in the Catalog. The first part is a general description of the process, with a discussion of the design basis and applicable assumptions for simplification of the design procedure. The second part is a design data sheet (or sheets) in which the performance characteristics, design criteria, and key design features are specified under the following headings:

PROCESS
FUNCTION
PARAMETERS AFFECTED
EFFECTIVENESS
APPLICATION LIMITS
DESIGN
TREATABILITY FACTOR
COST PARAMETER
COST CURVE SCALE FACTOR
RESIDUES

MAJOR EQUIPMENT

The following paragraphs indicate the type of information presented under each of these headings and provide guidelines for your review and comment.

Process

The name of the process is shown at the top of the page. The unit processes are arranged in the catalog in three groups: wastewater treatment processes, sludge treatment processes; and special systems.

Function

The purpose of the unit process is stated in broad terms, for example, "Removal of dissolved organics" (Activated Sludge), and "Removal of suspended solids" (Filtration, Dual-Media). Different processes could have the same general function, as in the cases of Activated Sludge and Activated Carbon Adsorption.

Parameters Affected

Pollutants which are altered by the process are listed. The list is not necessarily all-inclusive, but includes at least the following:

1. Parameters for which effluent limitations were previously promulgated by the U.S. Environmental Protection Agency for the organic chemicals industry for BPT.
2. Parameters which may affect the applicability, effectiveness, and/or cost of other (downstream) unit processes.
3. General classes of pollutants (e.g., dissolved organics) are specified when a listing would be too lengthy. These classes include priority pollutants.

Pollutant characteristics which do not fall into any of the foregoing categories are generally omitted (e.g., color), even though they may be altered by the process. In some instances, a parameter may be included because of prevailing state regulations.

Effectiveness

The effectiveness of the process in reducing the affected parameters is stated. In some cases, this statement may consist simply of a percentage removal or some other reliably achievable result (e.g., effluent concentration). In other cases, the statement is more complex because the effectiveness of the process is sensitive to various design parameters and to the relative treatability of the waste stream. In those cases, it is necessary to relate the effectiveness of the process both to the waste to which it is applied and to the chosen design criteria. These relationships are expressed or implied in the effectiveness statement and are further defined under other headings in the catalog (e.g., Design Basis).

For most unit processes there are practical limitations on effectiveness. A "Limit of Effectiveness" statement is sometimes needed to stipulate that there is a limit on percentage removal achievable or that the effluent concentration will not be below a particular achievable level.

For those unit processes involving consideration of relative treatability of the particular wastewater, a "reference case" has been included in the effectiveness statement. This specifies the effectiveness achieved for a given wastewater with a defined treatability and specified design basis. The design of this unit process for other wastes with different treatability is relatable to the reference case. (See Treatability Factor and Cost Curve Scale Factor).

Application Limits

Waste characteristics that must be controlled within certain ranges in order for the process to function properly are described. For example, the pH of wastewater generally must be controlled within a range of 6.0-8.5 in order that a biological proc such as activated sludge, can be applied. (These are exceptions, however. For examp if the wastewater exists as a buffered solution in an aeration basin, then excursions of pH in the influent stream can be tolerated. These exceptions are not definable with the data available for this study.) If the waste characteristics are not within the prescribed range, then a preceding unit process (e.g.,

neutralization) is applied so as to bring the characteristic within the required range.

Design Basis

The process design basis is specified. In some cases, this consists of specifying a design loading such as an overflow rate or hydraulic residence time. In others, particularly when relative treatability has a major impact upon cost, a functional relationship between design ("size") and performance is indicated.

Treatability Factor

For each unit process, this heading will include absolute and relative treatability information for each pollutant affected by that unit process. This information is discussed systematically in the TREATABILITY Section of this document.

Cost Parameter

A cost curve has been developed for each unit process, relating capital cost to a basic parameter representative of the size (and, therefore, the cost). In some cases, a second cost parameter is required for adequate description of the cost of the system in terms of its size. The cost curve will then be a family of curves on the same graph.

Cost Curve Scale Factor

For those unit processes involving a treatability factor, the cost estimates (and cost curves) are based upon a reference case. The design of the process and its cost were determined for a given value of the treatability factor. The cost parameter, in those cases, does not fully define the system size; it is necessary to refer to the reference case to do so. For example, the cost parameter for activated sludge is flow rate. However, the cost of an aeration basin is determined by the volume of the basin, which is a function of both the flow rate and the detention time. The detention time is directly related to the treatability of the waste. In order to disassociate the system cost curve from the treatability

of the particular waste encountered in the reference case, flow rate was chosen as the cost parameter. Although the cost curve is based upon the reference case (at different flow rates), the size of a basin (and therefore the cost) can be determined for any application once the detention time is specified. This is achieved by comparing the detention time with that of the reference case and scaling the flow rate up or down as indicated. The detention time in this case (activated sludge), is termed the Cost Curve Scale Factor.

Another use of the Cost Curve Scale Factor is as a multiplier of a unit cost. This is used, for instance, in aeration, where the cost factor is individual-aerator horsepower and the scale factor is the number of aerators. The cost estimate for one aerator (including associated instrumentation, electrical connections, structural supports, etc.) appears in the cost curves and then is multiplied by the number of aerators (of the same horsepower) required.

Residues

Any residues (solid, gaseous, or liquid) generated by the process are identified as to type and quantity. Either additional unit processes are provided for their treatment and disposal, or processes already included in the treatment scheme for some other purpose are designed to handle them.

Major Equipment

The major equipment and facilities that must be installed for this unit process are identified. The key features that affect cost (such as materials of construction, operating mode, and process variants) are also indicated.

TREATABILITY

The treatability of various pollutants and product/process waste streams had to be assessed and quantified to enable the model to predict contaminant removals in the various unit processes. The problem of predic-

tion is different for conventional parameters (e.g., BOD, TSS) than for specific pollutants. These differences in assessment of treatability are highlighted in the following discussion of the requirements for modeling each treatment unit processes.

Equalization

No treatability factors are involved in this unit process. The function of equalization is to lessen the variability of the raw waste load. The average values of parameters are unaffected. The only exception is wastewater temperature, which will move toward the ambient air temperature during the 1-day holding time in the equalization basin.

Neutralization

Again, there are no treatability factors involved. The size of the basin is determined by the wastewater flow, while the chemical requirements are determined by acidity/alkalinity values (if available) or by pH values if acidity/alkalinity are not reported. When no data (acidity/alkalinity or pH) are reported, a neutral pH is assumed.

Oil Separation and Dissolved Air Flotation

There are no treatability factors for oil and suspended solids removal. Basin size is determined by wastewater flow rate. Oil and TSS removal are predicted on the basis of operating experience. Organic pollutants are assumed to be removed down to their solubility in water. (These solubilities are taken from chemical handbooks.)

Coagulation and Flocculation

The treatability factors required for this unit process are the chemical coagulant used, the ratio of chemical to pollutant, and the remaining solubility of the precipitate formed for ion being removed. These factors are obtained from operating experience and chemical handbooks, supplemented as necessary by laboratory tests on metal-ion compounds for which the literature provided inadequate solubility data.

Clarification

Specific treatability factors are required. Different overflow rates, effluent TSS concentrations, and underflow TSS concentrations for various types of suspended solids are given in the design data sheet for the clarification unit process.

Activated Sludge

The treatability factors required for this unit process are the biological reacti rate coefficient (k factor) and the maximum attainable percent removal. The k factor has been estimated by grouping chemicals according to relative biological degradability; k rates for five classes, ranging from extremely biodegradable to bio-static or toxic, have been assigned. The biological treatability of different che has been related to chemical structure and functional group. The k factors for produc BOD's have been estimated by taking the average of the k's for the raw materials, intermediates, and products that make up a particular product process. Other sources of biological kinetics data and maximum percent removals are Screening/Verification data and responses to the 308 questionnaire.

Aeration, Biological Processes

There are no treatability factors involved. The power required depends on the oxygen transfer rate in the wastewater, oxygen solubility, and oxygen utilization rate, which in turn depends on the amount of BOD to be removed and the amount of MLVSS under aeration.

Nutrients, Biological Processes

There are no treatability factors involved. The amounts of nitrogen and phosphor to be added are the amounts necessary to maintain a BOD:N:P ratio of 100:5:1, based on using anhydrous ammonia and 75 percent phosphoric acid.

Nitrification

The treatability factor is a temperature correction, which affects the design nitrification rate. The design data sheet includes a plot

of temperature vs. percent of design nitrification rate.

Denitrification

As with nitrification, the treatability is a temperature correction. The design data sheet includes a plot of temperature vs. percent of design denitrification rate.

Ozonation

Three treatability parameters are involved: 1) the ozone/pollutant ratio required for treatment; 2) the normal lower level of effluent treatment achieved; and 3) the upper level of effluent treatment normally expected. These values have been obtained for a few pollutant parameters from surveys of the technical literature.

Chemical Oxidation

The treatability factors are similar to those for ozonation, but the oxidation chemical must be specified in addition to the chemical/pollutant ratio and the lower and upper expected effluent values.

Activated Carbon Adsorption

There are four treatability factors needed, and they are:

1. Isotherm constant - Langmuir constant related to the slope of the isotherm plot.
2. Isotherm constant - Langmuir constant related to the intercept and slope of the isotherm plot.
3. Final value - lowest attainable effluent value mg/l.
4. Peclet number.

Activated Carbon Regeneration

There is no treatability factor involved. The function of the regeneration furnace is to remove the adsorbed organics from the spent activated carbon, so that the carbon can be reused in the adsorption unit.

Regeneration loadings have all been given the same value: 40 $lb/hr/ft^2$. This rate may vary for different chemicals adsorbed onto the carbon, as

indicated by activated-carbon and incineration equipment manufacturers. The value selected for the model is probably on the low side of the actual average.

Ion Exchange

The treatability factors needed for this unit process are resin type (cationic or anionic), resin exchange capacity, effluent concentration attainable, regeneration chemical to be used, and regeneration chemical dosage. These have been obtained from the literature and from vendor data.

Gravity Thickening

There are no treatability factors. The function of this unit process is to increase the solids concentration, and thus facilitate the operation of the subsequent sludge-handling processes.

Aerobic Digestion

The treatability factor in this unit process is a temperature correction, which has a significant effect on the rate of reduction of the volatile suspended solids in waste activated sludge from biological treatment. The equation for this effect is included in the design data sheet for Aerobic Digestion.

Vacuum Filtration/Pressure Filtration

There are no treatability factors. The function of either of these unit processes is further removal of water from the sludge to prepare the sludge for landfilling or incineration. Pressure filtration is the preferred pretreatment for incineration because it yields a drier sludge.

Landfill

There are no treatability factors. This is a method of ultimate disposal for ash residues and dewatered sludges.

Incineration

Sludge moisture content is the treatability factor in this unit process.

The moisture content determines whether the incineration of the sludge is self-sustaining, or whether (and how much) auxiliary fuel is required.

Ammonia Stripping

There are no treatability factors. The function of this unit process is to remove ammonia from wastewater by direct injection of steam, and thus meeet the limitation on ammonia concentration in the effluent.

Steam Stripping

The factors required for steam stripping are pollutant latent heat, azeotropic composition, molecular weight, achievable effluent concentration, activity coefficient, K-value (function of the activity coefficient and the partial pressure), stripping-steam requirements, and tray efficiency. This information is available from chemical manufacturers and handbooks. Steam stripping tray effieciencies at low effluent concentration must be verified by laboratory experiment, to confirm or modify the calculated values currently being used.

Solvent Extraction

Treatability factors needed for this unit process are: the solubility, latent heat, and specific heat of the pollutant; identification of the solvent; the solvent density; and solvent-pollutant distribution coefficient. Pollutant properties are obtained from manufacturers and chemical handbooks.

Two solvents have been chosen for solvent extraction: tricresyl phosphate and a mixture of C_{10}-C_{12} paraffins (mostly straight-chain). Solvent density can be obtained from manufacturers or from the literature. The distribution coefficient for tricresyl phosphate was estimated from the literature. Its affinity for phenol is estimated to be eight times that of benzene for phenol; since the distribution coefficient for a benzene-phenol system is about 2.5, the distribution coefficient for tricresyl phosphate has been assumed to be 20. The distribution coefficient for the C_{10}-C_{12} paraffin has been assumed to be 30, based on textbook discussions and examples of solvent extraction where it was used to remove chlorinated

hydrocarbons. These assumptions need to be confirmed by laboratory experiment.

Cooling Tower/Heat Exchanger/Steam Injector

There are no treatability factors. A cooling tower or heat exchanger is introduced into the unit process trail to reduce the wastewater temperature to meet the operating requirement of a biological treatment system, or to conform to the temperature limitation on discharge of treated wastewater to a receiving stream or other body of surface water. A steam injector may be required in winter to heat a waste stream for biological treatment.

Deep-Well Disposal

There are no treatability factors. The function of the deep well is ultimate disposal of liquid wastes.

Lime Handling

There are no treatability factors. The function of this unit process is to provide lime (or caustic) for neutralization and other unit processes.

TREATMENT CATALOG AND MODEL SEQUENCING RULES

This treatment catalog and the computer model derived from it do not include all the possible treatment unit process alternatives. The inclusion or exclusion of specific unit-process types or configurations does not imply that the process types included in this treatment catalog are the only ones applicable to the treatment of the subject waste streams. The processes that have been included were chosen because they are widely used and provide representative treatment costs.

Chemical plants should have flow and/or contaminant equalization someplace in the treatment system, and possibly also in connection with storage or monitoring of the treated effluent. The cost of this unit can be effectively represented by the rules stated herein.

Many plants will have to transfer the waste streams by means of a lift station someplace in the treatment system. If they do not so require, sewer complexities or other waste-handling problems will add some increment

of cost, and that increment is assumed to be equivalent in cost impact to a lift (pumping) station.

Almost every plant requires some form of neutralization, even if only on an intermittent basis, and some type of facilities for this purpose will be provided.

Liners for large earthen basins will be of the synthetic-membrane type, rather than clay. Landfill liners will also be of the synthetic-membrane type.

UNIT PROCESS COST DEVELOPMENT

The unit process costs were developed by preparing detailed Flow Sheets, sizing the equipment, obtaining vendor quotations for major items, and then using standard estimating procedures to determine installed costs. The basic design parameters appear in the Treatment Catalog. Flow Sheets were constructed to establish the kinds of equipment required for a unit process. Equipment was sized according to specifications in the Treatment Catalog where specified, and by standard engineering calculations for equipment items or processes that were not so specified. After equipment was sized, an equipment list was prepared for estimating purposes.

This equipment list includes the identification and size of each significant item of hardware for each size range of each treatment unit process. It includes all mechanical equipment, basins, structures, vessels, and piping, but does not cover labor costs or instrumentation. The Catalytic Estimating Department estimators used this list as the basis for determination of the installed cost of a unit process (based on costs in the St. Louis, Mo. area as of June 1977). These installed costs included taxes, insurance, legal fees, contingencies, and overhead. The flow diagram for each unit process shows all instrumentation and thus serves as a basis for estimating the instrumentation costs.

Obviously, it is not practical to prepare cost estimates for all possible sizes of a unit process. On the other hand, it is not good practice

to use one cost estimate (base case) as the basis for all sizes, because the cost of a treatment facility is not necessarily directly proportional to its size. Consequently, a compromise approach was taken, involving four separate cost estimates generally covering a unit process size range of three orders of magnitude. For all unit processes designed to treat the wastewater forward flow to a treatment plant, flow rates of 0.2, 1, 5, and 20 MGD were used as the basis for the cost estimates.

The four ensuing cost estimates were used to establish the cost curves, which indicate the installed cost of each unit process as a function of the "cost parameter" involved. This cost parameter, for example, may be a flow rate, basin volume, or surface area. Although there can be only one "cost parameter" for a given unit process, there are other variables that may affect the cost curve. Thus, the "cost parameter" may be multiplied by a "scale factor" to provide the necessary adjustment. These scale factors are indicated on the design data sheet for each unit process.

In addition to the costs of the individual unit processes, there are many miscellaneous costs that must be considered such as: home office engineering, site development work, utility and general piping, electrical requirements, a control building, and a sanitary sewage pumping station. These costs are introduced into the model as functions of the total capital cost of the unit processes, the total operating horsepower, and the number of unit processes. The overall miscellaneous cost is then allocated back to the individual unit processes. This allocation of the miscellaneous costs to each unit process reflects the ratio of the individual unit process cost to the total cost of all the unit processes, as defined by the cost curves.

EQUALIZATION AND SURGE STORAGE

Start with an equalization basin with capacity equivalent to the 24-hour filling volume based on average total wastewater forward flow, and with a separate surge basin with a capacity equivalent to a similar 12-hour filling volume. Provide a pumpi station, equipped with two pumps each with a capacity equal to 120 percent of the daily average total wastewater forward flow.

If the ratio of the maximum daily flow to the average daily flow (or the ratio of the average to the minimum) exceeds 2.0, increase the size of the basins in accorda with the following formula:

New Size = Base Size x (Larger Flow Ratio - 2.0) + 1.0

If the ratio of the maximum calendar-month flow to the average daily flow (or the ratio of the average to the minimum) exceeds 1.5, increase the size of the basins in accordance with the following formula:

New Size = Base Size x 1.5 (Larger Flow Ratio - 1.5) + 1.0

If both ratios are in excess of the designated limits, calculate both increases, but use only the larger of the two.

All equalization basins are equipped with mixers. The design power demand on the mixers is 0.01 HP per 1,000 gallons of wastewater volume, and costs are based on floating mechanical mixers.*

In addition to reducing the flow variability and pollutant concentrations, equali will cause the wastewater temperature to approach ambient temperature. This change is accounted for with a temperature balance model that includes heat gains from influent wastewater, mechanical action, and solar radiation, and heat losses from effluent flow, evaporation, and surface and sidewall convection/conduction.

See the attached design data sheet for additional details.

* This power requirement is based on providing adequate mixing, and is not intended to prevent all influent suspended solids from settling.

EQUALIZATION/SURGE STORAGE

FUNCTION:	Equalization of flow and concentration. Capture of concentrated spills to process sewer.
PARAMETERS AFFECTED:	Hourly fluctuations of flow and pollutant concentration. Wastewater temperature
EFFECTIVENESS:	Essentially complete homogeneity of average daily flow, excluding major spills. Spills diverted to separate surge basin if detected in time. Reduces excessive temperature.
APPLICATION LIMITS:	None
DESIGN BASIS:	Equalization Basin Capacity: 24-hour detention time for average daily flow.* Mixing Energy: 0.01 HP/1,000 gallons. Surge Basin Capacity: 12-hour detention time for average daily flow.* Equalization will always be provided.
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow rate
COST CURVE SCALE FACTOR:	Based on flow variability
RESIDUES:	Solids accumulation dredged every 5 years.
MAJOR EQUIPMENT:	Equalization Basin and Surge Basin Construction. 550,000 gal: earthen basin with membrane liner (concrete abrasion pads under mixers) 200,000-550,000 gal: earthen basin with concrete-lined sides ** 20,000-200,000 gal: lined carbon steel tank ** 20,000 gal: stainless steel tank Mixers: Floating mechanical mixers Lift Station for each basin

* Capacity will be greater if the maximum:average or average:minimum flow ratios exceed the limits specified in the general discussion.

** No surge basin for these sizes.

NEUTRALIZATION

The basic component of the neutralization unit is a 2-chamber tank with design retention times of 5 minutes for the first chamber and 20 minutes for the second chamber. Normally, the value of 120 percent of the average wastewater flow is used in conjunction with these residence times to calculate the size of the chambers. However, if neutralization is to precede equalization for any reason (e.g., to avoid the use of expensive corrosion-resistant materials in an oil separator, which must precede equalization), the flow value used in this calculation is 200 percent of the average wastewater flow.

Both chambers are equipped with mixers and with pH controllers for acid and base addition. Facility design and capital costs for acid addition are based on sulfuric acid addition for all ranges. The base-addition facilities (and the related costs) are different for systems of different sizes. Caustic is specified for small systems (less than 500 lb/day), hydrated lime for systems requiring 500-800 lb/day, and quick lime for those requiring more than 8,000 lb/day. Lime storage silos and slakers, and caustic make-up facilities are not part of this unit process; they are included under "LIME HANDLING".

Chemical dosages are calculated whenever the acidity or alkalinity of the raw wastewater is known. For cases where this information is not available, a continuous demand of 200 mg/l of sulfuric acid is assumed. This value is considered valid for these cases where no acidity or alkalinity information is available because it is highly likely that alkalinity or pH data would be available if higher alkalinity were present. For systems

whose data indicate an essentially neutral condition, a continuous demand of 50 mg/l is used, to accomodate the occasional pH swings that can be expected in normal operation.

NEUTRALIZATION

FUNCTION:	pH Adjustment
PARAMETERS AFFECTED:	pH Acidity Alkalinity
EFFECTIVENESS:	Will achieve control within a pH unit range of -0.5 to +1.0 of target.
APPLICATION LIMITS:	None
DESIGN BASIS:	Dual (acid/base) titration with lime and sulfuric acid. Dosages: 50 mg/l of lime or H_2SO_4 for neutral wastes 200 mg/l of lime or H_2SO_4 for undetermined wastes Actual alkalinity or acidity where data are available Two-stage reaction chamber, having five-(5) and twenty-(20) minute detention times, based on 120 percent of average flow.
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow Rate
COST CURVE SCALE FACTOR:	2.0, when neutralization precedes equalization.
RESIDUES:	To be determined on case-by-case basis. Depending upon waste characteristics, gases may evolve (e.g., H_2S), or inert solids may be generated.
MAJOR EQUIPMENT:	Reaction chambers with mixers: 0.2 MGD - Concrete, Acid-Brick-Lined 0.2 MGD - Fiberglass Tanks Sulfuric Acid Storage Tank, carbon steel Sulfuric Acid Feed Pumps (centrifugal type) with a closed-loop recycle. Dual pH Control Units (one for coarse controls, one for fine control) with panel. Caustic or Lime Storage and Feed Equipment, carbon steel

OIL SEPARATION AND DISSOLVED-AIR FLOTATION

Since these two unit process are so frequently and so closely related, they are discussed together. However, each process is available to the model separately.

Before selecting the unit process to be applied, determine the characteristics of the oil, grease, and other floating and floatable materials present in the subject product/process, to determine the applicability (effectiveness) of each of these two treatment process. This is the basis for selection of either process or of both proce: in series. In all cases, the design flow rate is 120 percent of the average wastewate: flow, and a minimum of two (2) units, each at 50 percent of design capacity, will be provided.

A chemical (coagulant) mix tank and a feed system are listed for Dissolved Air Flotation (DAF). This equipment is to be used only with DAF and not with gravity oil separation.

When no information is available, the following rules apply:

For oil and grease concentrations:	
Greater than 150 mg/l	Oil Separation followed by Dissolved-Air Flotation for all cases.
Between 35 and 150 mg/l	
Feed to subsequent activated sludge or chemical coagulation steps.	Oil Separation with effluent at 35 mg/l or 50 percent removal, whichever is lower.
All others, including direct discharge.	Oil Separation followed by Dissolved-Air Flotation with effluent at 10 mg/l.
Below 35 mg/l	Oil Separation with effluent at 10 mg/l.
For floatable solids:	Dissolved-Air Flotation in all cases.

See the attached design data sheets for more details.

Disposal of separated solids and oils is described at the beginning of the Sludge-Handling section of this treatment catalog, and the available Sludge-treatment trains are listed in the accompanying table there.

OIL SEPARATION

FUNCTION:	Removal of floating oil and solids
PARAMETERS AFFECTED:	Floating oil Floatable or Floating Solids
EFFECTIVENESS:	Can achieve effluent concentration of 35 mg/l floating oil, or 10 mg/l floating oil for low-influent concentrations.
APPLICATION LIMITS:	None
DESIGN BASIS:	Overflow Rate = 1,000 gpd/ft^2 unless specific data indicate otherwise. Maximum horizontal velocity = 3 ft/min.
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow rate
SCALE FACTOR:	None
RESIDUES:	Oil and sludge Quantity to be determined upon application
MAJOR EQUIPMENT:	Splitter box, concrete, with acid-proof lining *Oil Separation Unit, concrete with acid-proof coating *Skimming Mechanism *Bottom Flight Scrapers Sludge Removal Pumps (positive-displacement type) Oil Sump, concrete, with acid-proof coating Oil Pumps Slop-Oil Tank, FRP

* Minimum of two (2) units

DISSOLVED-AIR FLOTATION

FUNCTION:	Removal of suspended and colloidal materials.
PARAMETERS AFFECTED:	TSS Free oil
EFFECTIVENESS:	80 percent removal efficiency Limits of effectiveness: TSS will not be reduced below 30 mg/l Free oil will not be reduced below 10 mg/l
APPLICATION:	Flow, TSS, temperature and pH must not be highly fluctuating.
DESIGN BASIS:	50 percent recycle Pressurized recycle aeration for 2 minutes ?@ 50 psig Overflow rate 2 gpm/ft^2 Preceded by flocculation
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow rate
RESIDUES:	Float: Characteristics to be defined upon application
SCALE FACTOR:	None
MAJOR EQUIPMENT:	Rectangular flotation clarifier with skimmer and bottom sludge removal; pressure tank; controls (carbon steel or concrete) Centrifugal compressor, carbon steel Flocculation chamber, carbon steel or concrete Polymer storage and feed system (housed in steel-sided building), fiber-reinforced plastic Chemical mix tank, concrete Chemical mix tank agitator, carbon steel Sludge pump, carbon steel Float sump, concrete or carbon steel Float pump, carbon steel

CHEMICAL PRECIPITATION, COAGULATION, AND FLOCCULATION

This unit process is used for removal of heavy metals and solids specifically noted as requiring coagulation, and for removal of specific dissolved materials such as sulfates or fluorides. Proper design requires a file of solubility data for each potential parameter. (Dissolved-air flotation has its own chemical feed system, and does not utilize this unit process).

Coagulation/flocculation is followed by clarification or filtration, depending upon the quantity and nature of the solids produced. The occurrence of a large amount of good floc favors the use of clarification; conversely, small amounts and relatively poor floc favor the use of filtration.

Separate mixing and flocculation chambers are used, with a mix time of two (2) minutes and a floc time of twenty (20) minutes, based on 120 percent of the average flow. Since it is seldom possible in practice to achieve theoretical levels, dissolved materials will be removed to a level 1.5 times the solubility of the resultant precipitated material in water. Lime and polyelectrolyte will be used as typical chemicals for costing purposes. A polyelectrolyte dosage of 1 mg/l is used in all cases.

See the attached design data sheet for more details.

COAGULATION AND FLOCCULATION

FUNCTION:	Conversion of dissolved, colloidal, and certain suspended solids to settleable suspended solids.
PARAMETERS AFFECTED:	Dissolved solids (TDS) Heavy metals
EFFECTIVENESS:	Dissolved ion removal to 1.5 times the solubility of the precipitated material in water.
APPLICATION LIMITS:	pH, depending upon ions to be removed.
DESIGN BASIS:	Mix time: two (2) minutes for coagulation. Floc time: twenty (20) minutes. Chemical dosage: 1.5 x stoichiometric requirement for precipitation. Polyelectrolyte dosage: 1 ppm.
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow
SCALE FACTOR:	Square root of the number of treatment chemicals used
RESIDUES:	Precipitate is removed by CLARIFICATION or DUAL-MEDIA FILTRATION, depending upon concentration and floc characteristics.
MAJOR EQUIPMENT:	Influent splitter box (concrete) *Reactor Chambers (concrete), with agitators *Flocculation chambers (concrete), with flocculators Polymer storage and feed systems

* Minimum of two (2) units

CLARIFICATION

The clarification process is used for removal of primary suspended solids, chemically-produced suspended solids from coagulation and flocculation, and biological suspended solids from biological unit processes. Pollution parameters affected are total suspended solids (TSS), plus those specific parameters made insoluble through chemical precipitation.

All clarification is categorized as one of three types: primary clarification without chemical treatment, primary clarification with chemical treatment, and secondary clarification following biological treatment. Special applications, such as lime settling prior to ammonia stripping, may be associated with any of the three basic forms.

The design basis and effectiveness for the clarification process are based on separate overflow rates for primary sludges, activated sludge (based on the Food/Microorganism (F/M) ratio), other biological sludges (nitrification/denitrification), alum sludges, iron sludges, and lime sludges. In the case of activated sludge, correction factors for effluent quality are included for influent MLSS concentrations and dissolved solids. These correction factors are included in the design data sheet which follows.

In all cases, two clarifiers (in parallel) are included, to insure continuous operation. Each clarifier has a capacity of 50 percent of the total design flow.

The effluent suspended solids (TSS) levels indicated on the specification sheet for activated sludge - 30 mg/l for F/M range of 0.2-0.6 and 40 mg/l for F/M range of 0.05-0.19 - seem well supported by the analysis of the November 1977 308 data. (The data involved in this analysis represent long-term averages of all the plants for which data were available.)

These data, however, do <u>not</u> support any consistent relationship between influent BOD to the activated sludge system and the effluent TSS from the clarifier. Nevertheless, there is a sound basis for a relationship

between the solids loading in the clarifier and the effluent TSS. As the activated sludge mixed liquor suspended solids (MLSS) increases for a given size of clarifier, there should also be an increase in the suspended solids concentration in the clarifier overflow. Consequently, an increase in effluent TSS from this relationship would take into account the higher MLSS required when the influent BOD is higher and/or where the biological reaction is lower because of a lower temperature.

The proposed TSS correction in the Treatment Catalog for a change in MLSS is:

$$\text{Addition to base-level effluent TSS} = \frac{\text{MLSS}-1000}{50}$$

This correction, plus a TSS correction for change in Total Dissolved Solids $\frac{(\text{TDS}-4000)}{100}$ would provide for a maximum clarifier effluent TSS, as shown in the following sample calculation for a system with an F/M ratio of 0.2-0.6, an MLSS concentration of 4000 mg/l, and an influent TDS concentration of of 10,000 mg/l:

$$\begin{aligned} \text{TSS} &= 30 + \frac{4000-1000}{50} + \frac{10{,}000-4000}{100} \\ &= 30 + 60 + 60 \\ &= 150 \text{ mg/l} \end{aligned}$$

This correction procedure is in agreement with industry's comments on the Treatment Catalog on a previous organic chemicals study for the National Commission on Water Quality (NCWQ).

CLARIFICATION

FUNCTION:	Removal of suspended solids
PARAMETERS AFFECTED:	Suspended solids (TSS)
APPLICATION LIMITS:	None

EFFECTIVENESS/ DESIGN BASIS:

Type of Solids	Overflow Rate (gpd/ft^2)	Effluent TSS (mg/l)	Underflow Concentration (%)
Primary Chemical	800	50	3
Alum	500	20	1.5
Iron	700	20	3
Lime	800	20	10
Sulfide	500	50	2
Activated Sludge*			
F/M: 0.2-0.6	500	30	1
F/M: 0.05-0.19	500	40*	1
Other Biological**	400	30	1

*Influent MLSS correction = $\frac{MLSS-1000}{50}$

Influent TDS correction = $\frac{TDS-4000}{100}$

Addition to effluent TSS as described in the preceeding discussion

**For biological nitrification and denitrification systems, the lowest overflow rate will be used. Effluent and TSS and underflow concentrations will be weighted averages.

TREATABILITY FACTOR: As per design basis

COST PARAMETER: Surface area

COST CURVE SCALE FACTOR: None

RESIDUES: Depending upon application (source):

Clarifier bottom sludge of the quantity and characteristics defined above.

CLARIFICATION (Continued)

MAJOR EQUIPMENT:

Clarifiers:

- Two provided, each with capacity for 50 percent of the waste water flow*
- Concrete bottom and side walls
- Clarifier mechanism with gear drive and motor
- Peripheral weir and baffle
- Surface skimmer and scum pit
- Scum pump

Sludge pumps (applies for primary or other chemical sludges only; for recycle pumps for activated sludge process, see ACTIVATED SLUDGE).

Influent splitter box (concrete)

* Could be more than two clarifiers. These would be equal in size, and together would have capacity for 100% of the wastewater flow.

FILTRATION, DUAL-MEDIA

The filtration process is used for the removal of suspended solids, such as residu biological solids in settled effluents from secondary treatment and residual chemical solids after alum, iron, or lime precipitation and settling. In these applications, filtration may serve either as a necessary preliminary step to further treatment (such as carbon adsorption or ion exchange), or as a final polishing step following other processes. In cases where chemical treatment is used and only a small quantity of floc is generated, filtration rather than clarification is used to remove the solids from the wastewater.

Oil, at low influent levels, can also be removed by this unit process.

The design for filtration is based on a hydraulic loading that varies with influer TSS concentration from 2.5 gpm/ft^2 to 7.9 gpm/ft^2, with a backwash rate of 20 gpm/ft^2 for 15 minutes per cycle. In conjunction with the water backwash, air scouring is provided at a rate of 5 SCFM/ft^2. An agitated holding tank, to receive backwash, is sized to hold 125 percent of the anticipated backwash from one filter. Even for low flows, a minimum of two operating filters and one spare will be specified. For higher flows (those requiring three or more operating filters) there will be no designa spare, but calculation of the required surface area will be based on 120 percent of the average wastewater flow, to facilitate switching from the operating mode to the backwash or standby mode without sacrifice of performance.

The cost parameters are based on pressurized downflow dual-media filters.

Backwash handling could vary for different applications of dual-media filtration. The backwash water may be recycled to the head end of the treatment plant, sent to the sludge-treatment facilities, or handled in a separate disposal system. This Treatment Catalog, however, specifies a separate backwash holding tank and then incorporates the solids handling into the sludge-treatment train.

FILTRATION, DUAL-MEDIA

FUNCTION:	Removal of suspended solids
PARAMETERS AFFECTED:	TSS
EFFECTIVENESS:	Limits of effectiveness: 90 percent TSS removal 75 percent oil removal Effluent TSS not less than 5 mg/l
APPLICATION LIMITS:	Free oil: 35 mg/l Influent TSS: 200 mg/l
DESIGN BASIS:	Hydraulic loading = $8.1 \times 10^{(-0.00255)}$ TSS Backwash water @ 20 gpm/ft^2 for 15 minutes per cycle Backwash air @ 5 SCFM/ft^2 for 3 minutes
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow rate
COST CURVE SCALE FACTOR:	None

RESIDUES:	Filter backwash waste
MAJOR EQUIPMENT:	Filters Pressurized downflow dual-media filters with 5-ft total bed depth, carbon steel Influent collection sump, concrete Feed pumps (centrifugal type) Filter effluent holding tank, carbon steel (if required)* Backwash pumps (if required)* Air compressors for backwash air

* Large units can utilize filtered forward flow from adjacent filter compartments for backwash.

ACTIVATED SLUDGE

The activated sludge process is used to remove dissolved and colloidal biodegradable organic material. Pollution parameters affected are BOD, COD, TOC, TOD, and specific soluble organic materials proven to be degradable (e.g., phenol). Other parameters, such as ammonia and suspended solids, are affected by the installation of this process, but are not the reason for its use.

Activated sludge is available to the sensitivity model in only two forms: conventional activated sludge; and extended aeration. Since chemical industry wastes tend to require long detention times, there are two elements in the breakpoint between the two forms for the model: 12-24 hours detention time; and 90 percent removal. The logic here is that if treatment (at whatever degree of removal) is accomplished in 12 hours or less, then conventional activated sludge is adequately descriptive. On the other hand, if 90 percent removal has <u>not</u> been achieved in 24 hours, then extended aeration is indeed the process in operation. Between 12 and 24 hours, conventional activated sludge will be used with a sliding scale on operational limits, as listed in the design data sheet.

Today, there are many forms of biological treatment, and each has its own particular features or advantages. In general, the alternatives to activated sludge are used either to lower the cost from that of activated sludge for the same or better performance, or to take advantage of a particular feature of the alternative system in order to bring the effluent of a problem waste to the quality usually expected from activated sludge. As an example of the second reason, some wastes will produce a biological floc that will not settle well, thus the making effluent suspended solids concentration high. Certain variations of the activated sludge process enhance settleability and can be the reason for varying the process.

These variations are available to new sources, but generally are not readily available to a plant that had installed a treatment plant before such variations were invented or sufficiently tested. Therefore, both forms of this process are available to the model in two modes. The first is the new-source mode, which involves the assumption that proper design of pretreatment and proper selection of the specific form of the biological treatment process can produce an effluent with "normal activate sludge process characteristics" at equivalent activated sludge cost (plus the cost of appropriate pretreatment). The second mode is the existing-source mode, which includes specific allowances for those factors that have an impact on effluent quality Typical examples are the effects on effluent suspended solids from influent BOD concentration and from dissolved solids in the wastewater.

Biological treatability reaction rate coefficients are determined as accurately as possible from actual treatment plant data in the industry. Where such data are unavailable, pilot or laboratory-scale data are used. The values chosen represent the treatment mode most widely used for the wastes in question. If pilot or laborator data are used, K rates are chosen from systems with aeration-basin detention times as close as possible to 24 hours. Only single-stage systems are used.

Application limits, as shown in the following design data pages, trigger the need for pretreatment if exceeded. (The various unit processes used for pretreatment and the manner of their application are detailed in other sections of this catalog). The suspended solids limitation is intended to protect the activated sludge biomass from becoming too heavily loaded with non-volatile solids. The ammonia limitation is to prevent ammonia toxicity. The decision as to whether ammonia must be removed (because of its status as a pollutant) is made elsewhere in the treatment catalog.

ACTIVATED SLUDGE

FUNCTION:	Removal of dissolved organics
PARAMETERS AFFECTED:	BOD COD (TOC, TOD) Phenol Other specific organic materials
EFFECTIVENESS:	Soluble BOD_5: removal up to 99 percent for easily degradable materials and 97 percent for those difficult to degrade. Conventional activated sludge (CS) will be used up to 24 hours detention or 90 percent soluble BOD removal, whichever is greater. Beyond that level, extended aeration (EA) will be used. Effluent BOD_5: Soluble fraction not less than 10 mg/l; suspended solids fraction = 0.3 lb of BOD_5 per lb of solids leaving final clarification. Phenol Removal: Although cases will vary, pure phenol removal will be high (99% or greater) in steady-load, acclimated systems, with removal dropping off as the phenol" analysis reflects substituted phenols and other phenolic compounds. Regardless of removal rates, bottom level limits will reflect the influent concentration and expected variability. Oil Removal: 50 percent COD, TOC and TOD removal: Although cases will vary, in general the removal of these parameters will reflect the biodegradable fraction of each parameter.

APPLICATION LIMITS:	pH		6.0 - 9.0
	Temperature		50 - 100°F
	Oil		35 mg/l
	TDS		10,000 mg/l
	TSS		(25 mg/l + .05 MLVSS)
	Heavy Metals:	Pb	1.5 mg/l
		Zn	1.5 mg/l
		Cr	1.5 mg/l
		Cu	0.5 mg/l
		Ni	0.5 mg/l
		CN	3.0 mg/l
	Phenol:		300 mg/l (steady load)
			100 mg/l (fluctuating load)
	NH_3:		(500 + 0.05 BOD_5)

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DESIGN BASIS:

$S_o (S_o - S_e) = K_T X S_e t$

where S_e = effluent BOD concentration (dissolved), mg/l
S_o = influent BOD concentration (dissolved), mg/l
K_T = reaction rate coefficient at operating temp. (^oC), day -1
X = mixed liquor volatile suspended solids, mg/l
t = aeration time, day

$$F/M = \frac{S_o}{Xt}$$

0.05 to 0.6 day^{-1}	overall range
0.20 to 0.6 day^{-1}	range for a conventional system
0.05 to 0.2 day^{-1}	range for extended aeration

X = 4,000 mg/l

TREATABILITY FACTOR:

$K_T = K_{20^oC} (1.06)^{T-20}$

where T = temperature (^oC)

TEMPERATURE LOSS RATE:

A model that includes heat gains from the influent wastewater flow, mechanical action, and from biological and chemical reactions, plus solar radiation and losses including evaporation from the liquid surface and the sidewalls.

COST PARAMETERS:

Flow rate

COST CURVE SCALE FACTOR:

Aeration time

RESIDUES:

Excess activated sludge
lb dry solids produced (net)/lb BOD removed =
up to 24 hours detention: 0.3 lb/lb
24 hours detention: 0.3-(t-24)(0.003) lb/lb
solids 80 percent volatile

MAJOR EQUIPMENT:

Aeration basins
Two (2) basins, each with capacity for 50 percent of

design wastewater flow; common influent splitter box.

Construction:

550,000 gallons - all concrete

550,000 gallons - earthen basin with membrane liner; concrete abrasion pads under aerators (see AERATION)

Aerators - See AERATION

Clarifiers - See CLARIFICATION

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ACTIVATED SLUDGE (Continued)

Sludge recycle pumps: Three centrifugal pumps (including spare), each with capacity to pump 33.3 percent of the design wastewater flow at 1 percent solids concentration.

Monitoring and control devices
- Sludge-wasting control
- Sludge-recycle control
- DO monitor, temperature monitor
- pH monitor and control system

Nutrient storage and feed - See NUTRIENTS

Defoamer storage and feed

AERATION, BIOLOGICAL PROCESSES

Aeration is required in a biological unit process to supply the dissolved oxygen needed for sustaining biological growth reactions, and to provide mixing to keep the bio-mass suspended in the system. The biological processes requiring aeration are activated sludge, nitrification, and aerobic digestion. Aeration requirements and the equipment for activated sludge and nitrification are covered in this section, while the aeration facilities for aerobic digesters are covered under AEROBIC DIGESTION.

The design basis is the power required either to supply oxygen or to accomplish mixing, whichever is greater. The oxygen requirement for activated sludge is based on the lb O_2/lb BOD_5 removed. For nitrification, a requirement for nitrogen oxygen demand is added to the oxygen required for BOD removal. The mixing requirement is determined on the basis of horsepower per 1000 gallons of wastewater under aeration.

The cost parameters for these aeration systems are based on platform-mounted surface turbine aerators. See the attached design data sheet for a more detailed description of the design basis.

AERATION, BIOLOGICAL PROCESSES

FUNCTION: Supply dissolved oxygen and mixing for activated sludge and nitrification processes.

PARAMETERS AFFECTED: Dissolved oxygen (DO)

EFFECTIVENESS: Will maintain at least 2.0 mg/l DO in aeration basin at 30°C (summer conditions).

APPLICATION LIMITS: 100mg O_2/l per hour

DESIGN BASIS: Power Supplied = Power Required plus one extra aerator horsepower equivalent.

Activated Sludge

Power Required (at 85% motor and gear efficiency)

$$P_r = \frac{N_T}{3.0 \text{ lb } O_2/\text{hr/horsepower}}$$

subject to a minimum of 0.1 HP/1,000 gallons (under aeration).

Required Oxygen Transfer (lb/hr) at Standard Conditions

$$N_T = \frac{N_a}{\frac{C_{ss}P - C_L}{C_s} \quad (1.025)^{T-20}} = \frac{N_a}{0.4756}$$

$= 0.7$

$= 0.9$

C_{ss} = 7.63 mg/l @ 30°C

C_s = 9.17 mg/l @ 20°C

C_L = 2.0 mg/l

$P = \frac{\text{Barometric pressure at plant site}}{\text{Barometric pressure at sea level}} = 1$

T = 30°C

Oxygen Utilization Rate (lb/hr)

N_a = a' (lb BOD removed/hr) + b'
(lb MLVSS under aeration)
a' = 0.7 lb O_2/lb BOD removed
b' = see graph (on following page)

Nitrification

Oxygen Utilization Rate (lb/hr)

N_a=a'(lb BOD removed/hr) + b'(lb MLVSS under aeration) + 4.6 (lb NH_3 -N + Organic-N applied/hr)

Power Required is then computed as for activated sludge.

TREATABILITY FACTOR:	None
COST PARAMETER:	Installed horsepower per aerator
COST CURVE SCALE FACTOR:	Number of aerators
RESIDUES:	None
MAJOR EQUIPMENT:	Surface turbine-type aerators, mounting platforms, walkways, and concrete abrasion pads.

NUTRIENTS, BIOLOGICAL PROCESSES

A nutrient addition system is required for a biological unit process so that sufficient nitrogen and phosphorus will be present in the wastewater to insure that neither nutrient becomes the limiting factor in the biological growth reactions. In some cases, a sufficient amount of either one or both nutrients may already be present in the wastewater, which reduces or eliminates the need for a nutrient supply system. This is determined on a case-by-case basis. Capital costs are based on stora and feeding facilities for phosphoric acid and/or anhydrous ammonia.

NUTRIENTS, BIOLOGICAL PROCESSES

FUNCTION:	Supply nutrients to biological processes, such as activated sludge, if not already present in wastewater in required amounts.
PARAMETERS AFFECTED:	None
EFFECTIVENESS:	Supply enough nitrogen (N) and phosphorus (P) to maintain biological unit processes.
APPLICATION LIMITS:	Only enough nitrogen and phosphorus added to maintain a small residual of nitrogen and phosphorus in effluent from biological unit process.
DESIGN RATES:	BOD:N:P ratio = 100:5:1 using 75 percent phosphoric acid (H_3PO_4) and anhydrous ammonia (NH_3)
TREATABILITY FACTOR:	None
COST PARAMETER:	Nitrogen deficiency Phosphate deficiency
COST CURVE SCALE FACTOR:	None
RESIDUES:	None
MAJOR EQUIPMENT:	Ammonia storage tank (carbon steel), with ammonia feed system, including water bath, immersion heater, evaporator, and ejector - if usage is over 2 tons/week. Ammonia cylinders and ejector - if usage is equal to or less than 2 tons/week. Phosphoric acid storage tank, - fiber-reinforced plastic, (if usage is over 200 lbs/day) Phosphoric acid, feed pumps (metering type).

NITRIFICATION, BIOLOGICAL

The biological nitrification unit process is used for ammonia removal, when required, following an activated sludge system with a detention time of less than 24 hours. Because activated sludge systems with detention times greater than 24 hours (extended aeration) operate at an F:M of 0.2 or lower, it is assumed that nitrif- takes place concurrently with carbonaceous BOD removal, and does not always require an additional step. In almost all cases, the nitrification unit process follows an act sludge process. However, if the discharge from some other type of treatment unit process has an easily biodegradable waste with a BOD_5 of less than 125 mg/l and a BOD_5/TKN ratio of less than 3.0, then the nitrification unit process can be considere for direct use. The pollution parameters affected by this unit process are ammonia nitrogen (NH_3-N), organic nitrogen (TKN), BOD, COD, and TOC. Ammonia compounds are converted biologically, first to nitrites (NO_2) and then to nitrates (NO_3). Nitrog in these forms can be converted to nitrogen gas (N_2) by anaerobic denitrification (See DENITRIFICATION, BIOLOGICAL).

The design parameters are based on lb NH_3-N and lb TKN removed per lb MLVSS per day, with a correction factor for temperature. The cost parameter is based on flow, with a cost-curve scale factor for aeration time.

Application limits, as shown in the specification pages, will either trigger the need for pretreatment or rule out this unit process for ammonia removal.

NITRIFICATION, BIOLOGICAL

FUNCTION:	Conversion of NH_3-N and Organic-N to NO_2-N and NO_3-N
PARAMETERS AFFECTED:	NH_3-N Organic-N (TKN) BOD_5 COD TOC
EFFECTIVENESS:	NH_3-N + Organic-N removal to 2 mg/l Soluble BOD_5 removal to 5 mg/l
APPLICATION LIMITS:	pH 7.5-9.0 Temperature 50-100°F BOD_5 125 mg/l BOD_5/TKN 3.0 TDS 10,000 mg/l Total Nitrogen 2,000 mg/l
DESIGN BASIS:	Basin configuration = complete mix Nitrification rate = 0.3 lb NH_3-N + TKN removed/lb MLVSS/day @ 30°C and pH 8.5 MLVSS = 2000 mg/l

$$HT = \frac{N_o - N_1}{q_N \times X_1}$$

where:

HT = hydraulic detention time (days)
N_o = influent NH_3-N + Organic-N (mg/l)
N_1 = effluent NH_3-N + Organic-N (mg/l)
q_N = nitrification rate (day^{-1})
X_1 = MLVSS (mg/l)

Aeration Tank D.O. 2.0 mg/l

TREATABILITY FACTOR:	Temperature correction

COST PARAMETERS: Basin volume

COST CURVE SCALE FACTOR: Aeration time

RESIDUES: Excess sludge:
lb dry solids produced/lb NH_3-N + Organic-N removed = 0.5
lb dry solids produced/lb BOD_5 removed = 0.3

MAJOR EQUIPMENT: Aeration basin:
Two provided, each @ 50 percent capacity, with one 10'-deep influent splitter box
Construction:
550,000 gal. - all concrete
550,000 gal. - earthen basin with membrane liner

(The number of baffles is estimated on the basis of complete mixing.)
Aerators: See AERATION, BIOLOGICAL PROCESSES
Clarifiers: See CLARIFICATION
Sludge recycle pumps:
Three centrifugal pumps (including spare) each @ 33.3 percent of plant design flow
Monitoring and control devices
Sludge wasting control
Sludge recycle control
DO monitor
pH monitor and control system
Defoamer storage and feed
Temperature monitor

DENITRIFICATION, BIOLOGICAL

The anaerobic denitrification unit process is used for the conversion of nitrates and nitrites to free nitrogen, following a nitrification unit process or an extended-aeration unit process. The pollution parameters affected are nitrates (NO_3) and nitrites (NO_2).

The design parameters are based on lb NO_3-N + NO_2-N per lb MLVSS per day, with a correction factor for temperature. The cost parameters are based on flow, with cost-curve scale factors for reaction time.

Effluents from nitrifying units are exceptionally free of BOD. For this reason, denitrification is a very slow process unless a readily oxidizable source of carbonaceous material is added. For the purpose of design, methanol is used as the source of carbon because it is more completely oxidized and produces less sludge for disposal, and is more cost effective than other source of carbon. The effluent from the denitrification unit is flash-aerated prior to clarification, to oxidize the excess methanol and to strip entrapped nitrogen gas from the sludge in order to improve its settling characteristics.

See attached specification pages for a more detailed description of the design basis.

DENITRIFICATION, BIOLOGICAL

FUNCTION:	Conversion of NO_3-N and NO_2-N to free nitrogen
PARAMETERS AFFECTED:	NO_3-N NO_2-N
EFFECTIVENESS:	NO_3-N removal to 1.0 mg/l NO_2-N removal to 1.0 mg/l
APPLICATION LIMITS:	pH 6.0 - 8.0 Temperature 10 - 38°C (50 - 100°F) NO_3-N + NO_2-N 500 mg/l TDS 10,000 mg/l
DESIGN BASIS:	Basin configuration - complete mix Denitrification rate - 0.16 lb NO_3 + NO_2-N removed/lb MLVSS/day @ 30° and pH 7.0 Dissolved Oxygen 0.1 mg/l MLVSS = 2000 mg/l $HT = \frac{D_o - D_1}{g_D x X_1}$ HT = hydraulic detention time (days) D_o = influent NO_3 + NO_2-N (mg/l) D_1 = effluent NO_3 + NO_2-N (mg/l) g_D = denitrification rate (day^{-1}) X_1 = MLVSS (mg/l) Methanol requirements = 4 lbs methanol/lb NO_3-N + NO_2-N
TREATABILITY FACTOR:	Temperature correction
COST PARAMETER:	Flow rate
COST CURVE SCALE FACTOR:	Reaction time

RESIDUES:

Excess sludge
lbs dry solids produced/lb NO_3+NO_2-N removed = 0.7

MAJOR EQUIPMENT:

Reaction Basins (uncovered): Two provided, each @ 50 percent of capacity, with concrete influent splitter box.
Construction:
550,000 - all concrete
_550,000 - earthen basin with membrane liner; concrete mixer-abrasion pads
Mixers: 0.5 HP/1000 cu ft (0.067 HP/1000 gal)
Clarifiers: See CLARIFICATION
Sludge Recycle Pumps:
Three centrifugal pumps (including spare) each @ 33.3 percent of plant design flow.
Monitoring and Control Devices:
Sludge wasting control
Sludge recycle control
pH monitoring and control system
Temperature monitor
Methanol Feed System:
Methanol storage tank (if usage is over 350 lb per day), carbon steel surrounded by a concrete dike
Methanol feed pumps (metering type)
Aerated Stabilization Chamber, concrete:
Detention time (0.5 hr)
Aerators: (0.1 HP/1000 gallons)
Acid storage tank (carbon steel) and feed pumps

OZONATION

The ozonation treatment process is used for the removal of cyanide and/or phenol, but only on individual process-unit streams or in plants with low wastewater flow.

The two principal features of the design basis for the ozonation process are: 7 lb O_3/lb cyanide and/or phenol for the ozone-generating equipment; and 30 minutes retention time for the contactor chambers.

The concentration of ozone (O_3) is 1.0-2.0 percent (by weight), or about 20 grams/cubic meter. Efficiency of usage is assumed to be 70 percent.

Waste air/ozone from the contactors will be returned to the compressor section, with 33 percent discharged to the atmosphere as a purge stream.

Equipment considerations limit the capacity of the ozonation unit to 2.0 MGD. Plate-type ozonators are used.

OZONATION

FUNCTION:	Removal of cyanide and phenol
PARAMETERS AFFECTED:	Cyanide Phenol Other organics
EFFECTIVENESS:	Cyanide removal - to less than or equal to 1 mg/l Phenol removal - total
APPLICATION LIMITS:	None
DESIGN BASIS:	Ozone dosage = 7 lb_3O_3 per lb (CN + phenol), 20 GM O_3/meter^3 Contact time = 30 minutes
TREATABILITY FACTOR:	None
COST PARAMETER:	Ozone usage rate (lb/day) and Flow
COST CURVE SCALE FACTOR:	Concentration of pollutants
RESIDUES:	None
MAJOR EQUIPMENT:	Ozone generator (air-fed) with compressor, cooler, water knockout drum, and dryer. Closed contact chamber (2-Stage), with venting system

CHEMICAL OXIDATION

Chemical oxidation is used in removal of cyanide and other organic and inorganic materials. This unit process is needed when the pollutant involved is not amenable to other types of treatment, for either technological or economic reasons. Cyanide removal by alkaline chlorination is used on individual-process waste streams when the cyanide concentration is too high for effective removal in a biological treatment system. To minimize the risk of formation of chlorinated organics, chlorine is used only when no feasible alternative is available.

Other available chemical oxidants include permanganates and hydrogen peroxide, which can be used as required for removal of specific organic and inorganic pollutants. However, for the purposes of preparing the cost estimate for this unit process, it was assumed that chlorine would be used. Oxidation by ozone, which would be generated on-site, performs the same function as oxidation by purchased oxidants, but the costs generally are more capital-intensive. (Ozonation is included in this Treatment Catalog as a separate unit process).

The choice among the various types of oxidation systems is essentially an economics decision.

CHEMICAL OXIDATION

FUNCTION:	Removal of cyanide and/or other organic and inorganic materials.
PARAMETERS AFFECTED:	Specific organic or inorganic pollutants.
EFFECTIVENESS:	Specific for each oxidant/pollutant combination. Total cyanide destruction by chlorine.
APPLICATION LIMITS:	TSS 50 mg/l maximum
DESIGN BASIS:	Contact time First stage 10 minutes Second stage 30 minutes Chemical requirements (CN destruction) 15 parts chlorine per part CN^- 17 parts NaOH* per part CN^- pH 8-9.5
TREATABILITY FACTOR:	None
COST PARAMETER:	Basin volume
RESIDUES:	Oxidized impurities that form TSS
MAJOR EQUIPMENT:	Two-stage, concrete reaction vessel pH control system ORP control system Oxidation-chemical feed system (for chlorine: chlorine vaporizer, chlorinator, and circulation pumps).

* A chemically equivalent amount of lime can be substituted for the 17 parts NaOH.

ACTIVATED CARBON ADSORPTION

The activated carbon process is used to remove dissolved organic material. Pollution parameters affected are COD, TOC, TOD, and specific soluble organic material adsorbable by carbon. In most cases, activated carbon is used as an individual-stream pretreatment process; however, in other cases, activated carbon treatment is used as a final treatment process following biological treatment.

Activated carbon adsorption rate coefficients are determined as accurately as possible from actual treatment plant data in industry when available. Where such data are unavailable, pilot or laboratory-scale data are used. Values chosen represent the adsorption rate most typical of the wastes in question for a variety of types of carbon. No attempt is made to optimize a particular brand of carbon to the particular wastes in question.

The time required to reach the "breakthrough point" is one of the most important design factors in fixed-bed adsorption processes. The approach taken to model the activated carbon adsorption process was to apply a method for predicting breakthrough times to yield: size, performance, cost, and operation data for the required carbon system.

Non-equilibrium fixed-bed column dynamics is suggested as the most applicable adsorption method for controlling water pollutants. Non-equilibrium theories take into account the nature of driving forces which control the transport phenomena of solutes from solution. Equilibrium theories assume the resistance to mass transfer to be negligible.

Three models were presented which deal with the theoretical treatment of non-equilibrium column dynamics: 1) pore diffusion; 2) homogeneous solid diffusion; and 3) kinetic reaction (or bilinear adsorption). All three models are based on the fundamental concept of a material balance for the adsorbate in the liquid phase and on the solid adsorbent.[1]

The model selected for analysis was the lumped parameter model (also

called the Glueckauf Model). This model combines the diffusional resistances described by the pore diffusion model and homogeneous solid diffusion model into a single parameter.

The approach taken to model the activated carbon adsorption process was the application of a method for predicting the breakthrough time of a pollutant from the known breakthrough time of a similar pollutant.[1] A breakthrough curve for benzene was selected as the reference case from which all other breaktrough times were predicted for those pollutants treatable by carbon.

Various pretreatment unit processes, such as filtration or clarification for suspended solids removal, frequently are required prior to the use of activated carbon. These processes are not included here, but they are detailed in other sections of this catalogue.

On-site carbon regeneration is considered only when carbon usage exceeds 1,000 lb carbon/day (see ACTIVATED CARBON REGENERATION). When carbon usage is below 1,000 lb/day, disposal is by landfill or outside contract regeneration.

ACTIVATED CARBON ADSORPTION

FUNCTION:	Removal of dissolved organics.
PARAMETERS AFFECTED:	COD, TOC, specific organic materials.
EFFECTIVENESS:	Removal of pollutant is subject to the adsorption limits for the compounds present.
APPLICATION LIMITS:	Suspended solids: 25 mg/l Free oil: 10 mg/l
DESIGN BASIS:	Langmuir adsorption isotherm theory for multicomponent mixtures. Design is based on the breakthrough time of the critical pollutant. The reference case is the breaktrough curve for benzene. Backwash @ 20 gpm/ft^2 for 15 minutes/cycle. On-site carbon regeneration only for plant using more than 1,000 lb carbon/day. See ACTIVATED CARBON

REGENERATION.

TREATABILITY FACTOR:	Isotherm constants Q = Langmuir constant related to slope B = Langmuir constant related to intercept and slope Final attainable value
COST PARAMETER:	Total bed volume
COST CURVE SCALE FACTOR:	None
RESIDUES:	Spent carbon If 1,000 lb/day, see ACTIVATED CARBON REGENERATION. If 1,000 lb/day, disposal by landfill or contract hauler. Backwash effluent. Drainage and transport water.

MAJOR EQUIPMENT:

Moderate-Flow Unit (0.4 MGD)

Adsorbers
Fixed-bed, pressurized, downflow contactors; minimum of two in series, plus a spare (all lined carbon steel).
Minimum depth: Diameter ratio = 1:1

Regenerated-carbon storage tank, lined carbon steel
Spent-carbon holding tank, rubber-lined carbon steel
Effluent holding tank, carbon steel
Backwash pumps
Backwash storage tank, carbon steel, (with agitator)
Backwash return pumps
Spent-carbon slurry pumps
Surface-spray pumps

If on-site carbon regeneration is involved (1,000 lb/day carbon), see ACTIVATED CARBON REGENERATION.

High-Flow Unit (0.4 MGD)

Feed pumps
Adsorbers, lined carbon steel
Pulsed-bed, fluidized, upflow contactors; minimum of two contactors, plus a spare.
Minimum depth: Diameter ratio = 1:1.

Regenerated-carbon storage tank
Spent-carbon storage tank
Spent-carbon slurry pumps
Regenerated-carbon loading pumps

ACTIVATED CARBON REGENERATION

The activated carbon regeneration process is used only when the carbon usage exceeds 1,000 lb/day. The parameter affected is the adsorption capacity of the carbon and there is a 10 percent carbon loss per cycle during regeneration. The carbon is regenerated on a column-by-column basis when the effluent quality reaches the limiting effluent requirement.

The design parameters are based on a multiple-hearth furnace with a carbon loading of 40 lb/day/square foot, plus 20 percent for down time. The cost parameters are based on the effective hearth area required.

Activated carbon regeneration by multiple-hearth furnace is the only regeneration process used in the model. Other regeneration processes (such as solvent washing and acid or caustic washing) were investigated, but were considered less desirable. See the attached design data page for a more detailed description of the design basis.

ACTIVATED CARBON REGENERATION

FUNCTION:	Remove and thermally oxidize adsorbed organics from spent activated carbon, for reuse of the carbon.
PARAMETERS AFFECTED:	Restoration of carbon adsorption capacity
EFFECTIVENESS:	Complete combustion of off-gases
APPLICATION LIMITS:	None
DESIGN BASIS:	Multiple-hearth furnace with afterburner on top hearth Carbon loading: 40 lb/day per ft^2 of hearth surface area Temperature: 1700°F to 1800?°F Surface area required: design plus 20 percent for down time Regeneration fuel: 8,000 Btu/lb of carbon Carbon loss: 10 percent per cycle
TREATABILITY FACTOR:	None
COST PARAMETER:	Total hearth area
COST CURVE SCALE FACTOR:	$(\text{No. of Furnaces})^{0.8}$
RESIDUES:	Clean off-gas and ash, representing the carbon losses
MAJOR EQUIPMENT:	Regeneration furnace (multiple-hearth) w/stacks and afterburner Quench chamber Venturi scrubber Separator Venturi recirculation tank and pumps Caustic storage and feed system, carbon steel Combustion and shaft cooling air blowers Fuel-oil storage and feed system, carbon steel Carbon transfer pumps Feed slurry tank Dewatering screw conveyor Asphalt slab, surrounded by a concrete wall, for storage of a 14-day supply of carbon during furnace downtime.

ION EXCHANGE

The ion exchange process is used to remove dissolved contaminants that are not amenable to other forms of treatment. Pollution parameters affected are cyanide, ammonia, and specific soluble heavy metals. In almost every case, ion exchange is used as a pretreatment process on a single product/ process waste stream.

The design parameters are based on the ion exchange resin capacity (lb/cubic foot of resin) for cyanide, ammonia, and specific metals. The values chosen represent the resin capacity most typical of the wastes in question for the type of resin being used. No attempt is made to optimize a particular brand of resin to the particular waste in question; thus, values are chosen of a variety of types of resin. Other design parameters include hydraulic loading and regeneration rates. The cost parameters are based on the working-bed volume required to achieve the desired effluent quality.

Various pretreatment unit processes are required preliminaries to the use of ion exchange, e.g., filtration or clarification for suspended solids removal. These processes are not included here, but they are detailed in other sections of this catalogue.

Ion exchange resins are regenerated, by rinsing with clean water and concentrated brine solutions, after the bed capacity is exhausted. The type of treatment system to be used on the regeneration wastes depends on the particular contaminants removed by the ion exchange.

See the attached design data page for a more detailed description of the design basis for ion exchange.

ION EXCHANGE

FUNCTION:	Removal of dissolved contaminants
PARAMETERS AFFECTED:	Cyanide Ammonia Metals Total Dissolved Solids (TDS)
EFFECTIVENESS:	Removal subject to ion-exchange rates for compounds present
APPLICATION LIMITS:	Suspended solids - 25 mg/l Oil - 10 mg/l
DESIGN BASIS:	Hydraulic loading: 1.5 gpm/ft^3 10 gpm/ft^2 (12 bed volumes/hr) Regeneration rate: 6 bed volumes @ 0.5 gpm/ft^3 (4 bed volumes/hr) Ion exchange capacity: 2 lb cyanide/cubic ft of resin 1 lb ammonia/cubic ft of resin 4 lb Zn, Ni, Cu/cubic ft of resin 3 lb Cr/cubic ft of resin

TREATABILITY FACTOR:	None
COST PARAMETERS:	Working bed volume and flow
COST CURVE:	Number of beds
RESIDUES:	Spent regenerant Cyanide - 15% NaCl solution (precipitation with $FeCl_3$) Ammonia - 15% NaCl,$CaCl_2$, CaO mixed solution (see AMMONIA STRIPPING) Zn,Ni,Co - 5% H_2SO_4 solution (precipitation with lime) Cr - 10% NaOH solution (chromic acid recovery) Backwash effluent
MAJOR EQUIPMENT:	Exchangers, Fiber-Reinforced Plastic (FRP) Fixed-bed pressurized downflow contactors, minimum of two in series. Minimum depth/diameter ratio = 1:1 Regenerant make-up tank, FRP Spent regenerant collection and treatment tanks, FRP Ion-exchange resin Regeneration and sludge pumps Regenerant treatment and make-up tank agitators

SLUDGE/RESIDUE HANDLING AND DISPOSAL

GENERAL

Aerobic digestion, gravity thickening, vacuum filtration, pressure filtration, landfill, and incineration are the Treatment Catalog unit processes for handling and disposal of sludges and other residues generated by biological and physical-chemical unit processes. A specific sludge/residue may require only one of these specific unit processes, or it may require treatment by a sequence of several unit processes.

Aerobic digestion is used to stabilize waste activated sludges, gravity thickening to concentrate chemical and biological sludges, and vacuum or pressure filtration for the dewatering of chemical and biological sludges. Ultimate disposal of sludges is either by landfill alone or by incineration followed by landfill. Federal and state regulations, land availability, sludge type, and other factors affect the decision on whether to landfill directly or to incinerate a processed sludge. Therefore, the option to examine both landfill and incineration of a sludge/residue is available for most of the treatment trains that will be encountered.

For each particular sludge or sludge combination from a biological or physical-chemical unit process, there is an assigned treatment train(s) for processing that sludge/residue. The treatment trains to be used on all sludges/residues are summarized on the attached table, which presents the sludge/residue type on the left and the treatment train options on the right side for that particular sludge/residue. When the quantity of a sludge/residue is small, the treatment train concept is not used, for economic reasons; instead, a private contractor is utilized to handle the sludge/residue.

In this sludge-handling section of the Treatment Catalog, the unit pro-cesses used in the sludge/residue treatment trains are examined on an individual basis. For each of the six sludge unit processes mentioned,

there is a narrative and a design data sheet(s) detailing its applicability, operation, design basis, association with other sludge unit processes, and other information necessary to model the unit process under consideration accurately.

TABLE 1. SLUDGE TREATMENT SUMMARY

Sludge/Residue	Disposal Options*	Gravity Thickening	Aerobic Digestion (Bio-Sludge Only)	Vacuum/ Pressure Filtration	Incineration	Landfill
Process Primary Sludge (Primary Clarifier)	L	X		X		X
	I		Option Not Available			
Incinerator Scrubber Sludge	O.C		Option Available			
Oil/Oily Solids	L		Option Not Available			
	I				X	
	O.C		Option Available			
Floatable Chemical Solids	L			X		X
	I			X	X	
	O.C		Option Available			
Waste Activated Sludge (WAS)	L	X	X	X		X
	I	X		X	X	
	O.C		Option Available			
Chemical Sludges from Flocculation/Clarification and Neutralization	L	X		X		X
	I		Option Not Available			
	O.C		Option Available			
Chemical plus Biological Sludge (WAS)**	L	X	X	X		X
	I	X		X	X	X
	O.C		Option Available			
Process Primary Sludge plus (WAS)	L	X	X	X		X
	I	X		X	X	
	O.C		Option Available			
Extraction/Distillation Residues	L		Option Not Available			
	I				X	
	O.C		Option Available			
Incinerator Ash	L					X
	I		Option Not Available			
	O.C		Option Available			
Non-Organic Filter	L	X		X		X
	I		Option Not Available			
	O.C		Option Available			
Backwash from Tertiary Sand Filter	L	X	X	X		X
	I	X		X	X	
	O.C		Option Available			
Throw-away Activated Carbon	L					X
	I		Option Not Available			
	O.C		Option Available			

* L = Landfills; I = Incineration; O.C. = Outside Contractor.

** For the Incineration option, biological and chemical sludges are thickened separately; the chemical sludge is landfilled, and the bio-sludge is incinerated. For the Landfill option, the two types of sludge are thickened separately, the bio-sludge is digested, and then the sludges are combined in a conditioning unit before dewatering.

GRAVITY THICKENING

Gravity thickening is used to concentrate waste activated sludge, chemical sludges, primary sludges, and certain combinations of these types of sludge. Concentr of these sludge solids in a gravity thickener results in cost savings in subsequent sludge dewatering.

The gravity thickener used in the model is a mechanical type with a picket sludge-collection device used to promote thickening. Thickened sludge that collects on the bottom of the thickener is pumped to a dewatering device as required. For biological sludge thickeners, recycled final effluent water is available to suppress odors associated with septic conditions. The continuous supernatant flow from sludge thickening is pumped to the head end of the treatment plant.

Determination of the thickener surface area, which dictates associated costs, is based on solids surface loading in $lb/ft^2/day$. Typical solids loadings vary depend on the type of sludge being thickened, as indicated on the attached design data sheet. For sludge combinations, a weighted average approach is used to define the solids loading to the thickener. Underflow solids concentration at the thickener depends on the type of sludge being concentrated, as well as on the solids loading. For sludg combinations, a weighted average technique is used to determine thickener underflow concen-trations. For more details on the thickener, see the attached design data sheet.

GRAVITY THICKENING

FUNCTION: Increase the solids concentration

PARAMETER AFFECTED: Concentration of suspended solids

EFFECTIVENESS/ DESIGN BASIS: Solids, underflow concentration:

Type of Sludge	Solids Loading ($lb/ft^2/day$)	Underflow Conc. (%)
Primary	20	9
Waste Activated	5	2.5
Lime	40	15
Alum	24	3
Iron	15	6
Combined	Weighted Average	

Overflow quality: 500 ppm TSS

APPLICATION LIMITS: None

TREATABILITY FACTOR: None

COST PARAMETER: Surface area

COST CURVE SCALE FACTOR: None

RESIDUES:
Scum
Thickened sludge at various solids concentrations, depending on the type of sludge.
Supernatant liquor - 500 mg/l suspended solids

MAJOR EQUIPMENT:
Thickener tank, carbon steel: 15-ft liquid depth
Thickener mechanism with skimmer (center feed, peripheral overflow), carbon steel
Underflow sludge pumps (positive-displacement type)

AEROBIC DIGESTION

Aerobic digestion is used to reduce the volatile suspended solids (VSS) content of a waste activated sludge (WAS) if the sludge is to be dewatered and hauled directly to a landfill for final disposition. When the WAS is to be incinerated, aerobic digestion is not used, because a digested sludge has a lowered percentage of VSS, making it less efficient as an incinerator fuel. Aerobic digestion is preceded by gravity thickeners in all sludge-handling systems.

The aerobic digester utilized in the model has a basin similar to that of an activated sludge system. The digester basin is 12 feet deep with a three-foot freeboard. Basin size determines the materials of construction and also the number of aerators required for proper digestion of the activated sludge. Aerobic digester basins are not covered or heated.

Digested sludge from this unit process is pumped to a dewatering unit. The dewatering unit is not normally operated continuously (24 hr/day), but the feed rate to the digester is relatively constant. Therefore, the water level will vary somewhat, and fixed-mounted surface aerators cannot be used. Floating, low-speed, surface-turbine aerators are provided.

Waste activated sludge going to the digester has a solids concentration of 2.5 percent, of which 80 percent is volatile. In the digester, the volatile material in the sludge will be reduced by 4 percent per day at 20°C, to a maximum reduction of 70 percent. A temperature correction factor is included on the specification sheet for operating temperatures different from the normal of 20°C. For further information on the aerobic digester, refer to the attached design data sheet.

AEROBIC DIGESTION

FUNCTION:	Reduction of volatile suspended solids (VSS) in waste activated sludge (WAS). Stabilization of WAS.
PARAMETER AFFECTED:	VSS
EFFECTIVENESS:	VSS Reduction = 4 percent/day @ 20°C, to a maximum of 70 percent
APPLICATION LIMITS:	pH of basin: 6.0-9.0 Basin temperature: 55-100°F
DESIGN BASIS:	VSS/TSS = 0.8 Influent solids concentration = 2.5 percent Hydraulic retention time = 15 days under normal conditions. Can be varied for temperature and VSS reduction considerations. Aerators: 0.1 HP/1,000 gallons
TREATABILITY FACTOR:	Temperature correction: percent reduction @ T = (% Reduction @ 20°C) $(1.06)^{T-20}$, where T = calculation temperature (°C)
COST PARAMETER:	Sludge - flow rate (gpd)
COST CURVE SCALE FACTOR:	Hydraulic retention time
RESIDUES:	Digested biological solids
MAJOR EQUIPMENT:	Digester basin (12' SWD + 3' Freeboard) 300,000 gal: steel tank, above ground 300,000 gal: earthen basin, with plastic membrane liner Floating, low-speed mechanical aerators Sludge-transfer pumps (progressive-cavity, variable-speed)

VACUUM FILTRATION

Vacuum filtration, pressure filtration, and centrifugation all are accepted sludg dewatering techniques. The choice is usually based on technical effectiveness, and depends on the type of sludge to be dewatered. Vacuum filtration is discussed here; pressure filtration is covered in a separate section of this Catalog.

The rotary vacuum filter is used in the model to dewater chemical sludges, biological sludges, and their various combinations. The sludges are fed to the vacuum filter at various solids concentrations from the sludge thickener. Lime and ferric chloride are used to condition all biological sludges for dewatering; conditioning is also required for some chemical sludges. The quantities of conditioners required are defined as a certain weight percentage of the sludge being dewatered. (See the attached design data sheet for the lime and ferric chloride requirements for different sludges.) The resulting sludge cake from the vacuum filter is either sent to a contro landfill or incinerated. Filtrate from the vacuum filtration operation is pumped to the head end of the plant.

Filter surface area is the basis for determination of the associated costs of a vacuum filtration system. Factors which affect filter surface areas are filter yield and operation time. Filter yield, defined as pounds of dry sludge filtered per hour per square foot of surface area, varies depending on the type and quantity of sludge being filtered. Filter yields for both chemical and biological sludges are pre on the attached design data sheet. In the case of combined sludges, weighted averages determine filter yields. Performance of the designed vacuum filter is dependent on the type of sludge. Cake moistures for different sludges are also presented on the attached design data sheet. In combination sludge cases, weighted average techniq again are used to determine sludge cake moisture.

VACUUM FILTRATION

FUNCTION: Dewatering of sludge

PARAMETERS AFFECTED: Sludge Moisture Content

EFFECTIVENESS/ DESIGN BASIS: Chemical requirements, filter yields, expected cake solids:

Sludge Type	Lime %	$FeCl_3$ %	Filter Yield lb/hr/sq ft	Expected Cake Solids %
Biological	15	4	2	12
Primary	8	1.5	8	25
D.A.F. Chemical Float	8	1.5	8	25
Sulfide	20	-	1.2	20
Incinerator Scrubber Sludge	8	1.5	8	25
Iron	20	-	1.2	20
Alum	20	-	0.8	20
Lime	-	-	10	40
Combined		Weighted Average		

Filtrate Quality: 1,000 ppm TSS (maximum)

TREATABILITY FACTOR:	None
COST PARAMETER:	Filter media surface area
RESIDUES:	Filtrate (return to head end of plant) Filter cake - see effectiveness/design basis

MAJOR EQUIPMENT:	Vacuum filters (two)
	Sludge conditioning unit
	  Mix chamber with mixers, carbon steel
	  $FeCl_3$ storage and feed unit, rubber-lined carbon steel
	  Lime storage and feed system, carbon steel
	Filtrate return system (receivers and pumps), carbon steel
	Cake-conveying units, carbon steel
	Cake storage units, carbon steel
	  Hopper
	  Bin activator
	Vacuum pumps (two) including silencers
	Facilities for equipment shelter
	Building sump and pumps

PRESSURE FILTRATION

Pressure filtration (filter press) is available as an alternative to vacuum filtr It is applicable to all types of sludges and is used whenever the required solids conc for the dewatered sludge is higher than the maximum achievable by vacuum filtration.

The application of pressure filtration encountered most frequently is the dewater of waste activated (biological) sludge prior to incineration. Filter presses are used in this situation because they can produce a dewatered sludge with a solids concentrat of 35 percent or more, which is high enough for self-sustained combustion (no auxiliar fuel required). Filter presses can also be used to dewater chemical or primary sludge that are to be disposed of in a landfill; their advantage is a greater reduction in sl volume, which could be an important factor if the landfill is small relative to demand or if the haul distance to the landfill is significant.

The pressure filtration system is sized on the basis of 8 hours per day, 5 days per week operation. A minimum of two filters is provided, which permits the design sludge load to be processed by the remaining filter (or filters) over a longer period of time (e.g., 16 hours per day for a 2-filter system) when one filter is out of servi

PRESSURE FILTRATION

FUNCTION: Dewatering of sludge

PARAMETER AFFECTED: Sludge moisture content

EFFECTIVENESS/ DESIGN BASIS: Chemical requirements, filter yields, expected cake solids:

Sludge Type	Lime %	$FeCl_3$ %	Sludge Loading* lb/ft^2/cycle	Expected Cake Solids %
Biological	8	15	1.5	35
Process primary	5	10	2.2	45
D.A.F. Chemical float	5	10	2.2	45
Sulfide	30	-	2.2	40
Incinerator Scrubber Sludge	5	10	2.2	45
Iron	-	30	1.6	40
Alum	-	30	1.6	40
Lime	-	-	3.6	50

*Three cycles per 8-hour shift

Diatomaceous-earth Requirement: approx. 8 lb/100 sq ft of filter area

TREATABILITY FACTOR:	None
COST PARAMETER:	Filter plate area
RESIDUES:	Filtrate - to clarification Cake - to incineration or landfill
MAJOR EQUIPMENT:	Feed pumps Contact tank, carbon steel Surge tanks, carbon steel Ferric chloride tank, rubber-lined carbon steel Ferric chloride pumps Filters, carbon steel/cast iron Cake conveyors, carbon steel Filtrate tank, carbon steel Precoat blower Bag breaker Precoat storage bin, carbon steel Precoat feeder Precoat slurry tank Precoat pumps

LANDFILL

Landfill is the method of ultimate disposal for ash and dewatered sludge in the treatment model. Depending on existing legislation and the particulars of a given situation, an individual company may either landfill on unused land on plant property, purchase land on a suitable site away from plant property, or utilize a public or privately-owned landfill. For the purposes of the model, it is assumed that a company will construct and operate a controlled landfill on a suitable site on its own property, away from the production area. The location, design, construction, and operation of the controlled industrial landfill are such as to minimize degradatio of air and water quality in the immediate area of the landfill.

In the initial design of a controlled landfill, area requirements are of prime concern. The landfill is designed to handle all dewatered sludges/residues produced in a 20-year period; but operation is based on 2-year cells. Loading rates to the landfill vary depending on the type of sludge being landfilled, and on the dewatering method used (vacuum or pressure filtration). The attached design data sheets present all landfill loadings and other design parameters.

The area and loading requirements presented are based on a mixture of sludge and soil to a final solids concentration of 80 percent. The landfill area designated for each landfill cell is adequately diked and has easy access for earthmoving and dumping equipment; to account for this extra area requirement, the basic cell area requirement for sludge and soil is increased by 25 percent. While one cell is being utilized, the next 2-year cell is being constructed, thus providing flexibility in oper The landfill cell area is lined with a plastic membrane liner to contain leachate flow A 2-foot layer of sand drainage material is placed above this liner on the landfill bottom. Any leachate that develops percolates through the landfill sludge layer, hits the drainage material and then drains down the slightly-sloped landfill bottom toward a central leachate collection basin. Besides collecting leachate, this basin also collects rainwater. Central-collection-basin water is treated by a package physic chemical treatment system. Leachate monitoring wells are strategically placed around the landfill to determine whether there is any leachate contamination of groundwater. In addition, an underdrain system is provided to intercept any leachate that leaks through the liner and prevent it from reaching the groundwater.

LANDFILL

FUNCTION: Ultimate disposal of sludge

APPLICATION LIMITS: None

DESIGN BASIS: Loading rate based on adding sludge and soil to 80 percent solids:

Sludge	Sludge Solids Loading: Vacuum Filter	Sludge Solids Loading: Pressure Filter	(Dry tons/ Acre-ft) From Process
WAS	27	103	-
Primary	71	169	-
Lime	170	270	-
$FeCl_3$	49	124	-
Alum	49	122	-
Sulfide	49	122	-
Fly Ash	-	-	318

Operating time: 20 years with 2-year cells
Final area requirement: 1.25 x sludge area requirement
Landfill depth: 10 ft.

TREATABILITY FACTOR: None

COST PARAMETER: Land area requirements based on sludge application rate.

COST CURVE SCALE FACTOR: None

RESIDUES: Leachate
Runoff

MAJOR EQUIPMENT: Membrane-lined earthern landfill cells, 2-yr capacity each
Bulldozer
Leachate monitoring wells around landfill
Wide-tire dump truck(s)
Leachate collection basin, earthern basin, membrane-lined
Leachate collection sump (concrete), with transfer pump.
Package treatment plant feed pumps in concrete sump.
Package treatment plant
Leachate-monitoring underdrain system

INCINERATION

Incineration is available as a volume-reduction method for organic primary sludges and waste activated sludges, and as a final disposal method for oils and the liquid residues from extraction/distillation. Two types of incinerators (the multiple-hearth and vertical liquid waste type) are used in the model, depending on the particular type of waste to be incinerated. If biological sludge, oil, or liquid residue quantities are below a defined limit, the residue is handled by contractor disposal rather than by incineration.

The multiple-hearth incinerator is used to burn liquid residues from extraction/distillation and biological sludges with or without waste oils. Gases produced by residue combustion pass through a venturi scrubber for particulate removal. If removal of contaminants in a vapor phase is also required, the off gases will then be passed through a packed-tower alkaline scrubber. Hearth area requirements are based on a loading of 8 lb/hr/sq ft.

Depending on the calorific value of the wastes being burned in the incinerator, auxiliary fuels may be necessary to support combustion. The attached design data sheet presents typical fuel values of typical wastes and of No. 2 fuel oil, which is the auxiliary fuel to be used when necessary to assist combustion. Storage and feeding facilities for No. 2 fuel oil are provided even if the heat value of the wastes is high enough to sustain combustion, because auxiliary fuel is considered necessary for incinerator start-up.

When liquid wastes (oils, extraction/distillation residues) are burned without being mixed with biological sludges, a liquid waste incinerator is used. Waste gases are cleaned with a venturi scrubber, followed by a packed-tower alkaline scrubber, which is considered mandatory for liquid waste incineration. The combustion chamber volume is based on a heat release value Of 40,000 Btu/hr/cu ft.

No spare units are provided for incineration and scrubbing facilities. In lieu of duplicate units, a storage vessel that has the capacity to hold two weeks' feed (average rate) to the incinerator is provided.

Separate design data sheets are presented for Sludge Incineration (Liquid Optional) and for Liquid Incineration.

SLUDGE INCINERATION (with LIQUID OPTIONAL)

FUNCTION: Volume reduction

PARAMETERS AFFECTED: Sludge volume

EFFECTIVENESS:

Type of Waste	Ash Remaining After Combustion
Organic primary sludge	35%
Waste activated sludge	35%
Grease, scum, oily wastes	5%

APPLICATION LIMITS: Sludge must be dewatered before incinceration

DESIGN BASIS:

$$\text{Hearth area} = \frac{\text{Total lb of waste/hr}}{\text{8.0 lb/hr/sq ft}}$$

Total heat release = (lb waste/hr) x (avg Btu/lb)
+ (lb aux fuel/hr) x (Btu/lb)

Typical fuel values of wastes

Type of Waste	Fuel Value Btu/lb dry solids
Organic primary sludge	6,500
Grease, scum, oily wastes	16,000 (pure fraction only)
Waste Activated Sludge	8,000 (volatiles only)

Typical fuel value of No. 2 fuel oil = 18,000 Btu/lb
@ 7.25 lb/gal
Typical fuel requirements of water = 2,000 Btu/lb
Operating time: 24 hrs/day; 5 days/week

TREATABILITY FACTOR: Sludge moisture content

COST PARAMETER(S): Lb/hr of wet sludge

COST CURVE SCALE FACTOR: None

RESIDUES: Ash (see EFFECTIVENESS for percent)
(See LANDFILL)
Venturi scrubber slurry
Spent packed-tower scrubber liquor (if liquid wastes are present)

MAJOR EQUIPMENT:

Multiple-hearth incinerator, including cooling-air fan for center shaft and combustion-air blowers (supplied as vendor package).
Sludge handling system (storage vessel, bin vibrator, conveyors, controls), carbon steel.
Gas-scrubbing unit (Venturi type).
Exhaust blower
Separator, carbon steel.
Venturi recycle pumps.
Venturi recycle tank
Ash-handling system (conveyors, storage vessel, vibrator)
Packed tower (optional).
Caustic storage and feed system, carbon steel.
Liquid waste or auxiliary fuel system (storage vessel, pumps, controls).
Gas-quenching system, carbon steel.
Vent stack.
Afterburner.
Feed storage tank and bin vibrator, carbon steel.
Asphalt slab, surrounded by concrete wall, for storing a 14-day supply of sludge when the incinerator is not in operation.

LIQUID INCINERATION (OILS, EXTRACTION/DISTILLATION RESIDUE)

FUNCTION: Ultimate disposal

PARAMETERS AFFECTED: All organics

EFFECTIVENESS: Total combustion; negligible residue

APPLICATION LIMITS: NONE

DESIGN BASIS: Total heat release = (lb waste/hr) x (avg Btu/lb) + (lb aux. fuel/hr) x (Btu/lb)

$$\text{Minimum incinerator volume} = \frac{\text{Total heat released (Btu/hr)}}{\text{40,000 Btu/cu ft/hr}}$$

Typical fuel values of wastes:

Type of Waste	Fuel Value (Btu/lb)
Oils	16,000
Phenolics	14,000
Paraffins	19,000
Aromatics	17,500
Cyclics	18,700
Olefins	19,500

Operating time: 24 hr/day; 5 days/week

TREATABILITY FACTOR:	Moisture content of the waste
COST PARAMETER:	Required heat release
COST CURVE SCALE FACTOR:	None
RESIDUE:	Venturi ash scrubber slurry Spent packed-tower scrubber liquor.
MAJOR EQUIPMENT:	Incinerator Waste injector assembly Combustion air blower Liquid waste storage and feed system, stainless steel Fuel-oil storage & feed system, carbon steel Venturi scrubber unit Cyclone separator Venturi recycle tank, with agitator Venturi recycle pumps Packed tower Packed tower recirculation tank with agitator Gas quenching system Vent stack, carbon steel Gas-emission monitoring equipment

AMMONIA STRIPPING

Ammonia stripping removes ammonia from pre-limed wastewaters by using direct-contact steam as the heat input. Two stripping towers are used, each with an independent collection system. Each tower has the same wastewater flow rate, with 200 gallons per minute specified as the upper limit for flow to a column.

Pre-liming is necessary to facilitate the removal of ammonia, and a pH of 10.5 or higher is specified for the system. Ammonia removal is 99 percent of the influent rate, and the attainable effluent limit is set at 50 ppm of ammonia.

The ammonia is collected overhead and sent to a spary absorber, where it is reacted with dilute sulfuric acid to produce ammonium sulfate, which is recovered as a crystalline powder.

AMMONIA STRIPPING

FUNCTION:	Removal of ammonia from wastewater by direct injection of steam into a distillation column.
PARAMETERS AFFECTED:	Ammonia concentration
EFFECTIVENESS:	99 percent removal of ammonia, or 50 ppm of ammonia in the effluent.
APPLICATION LIMITS:	TSS: 50 mg/l pH: 10.5 NH_3-N: 500 mg/l
DESIGN BASIS:	Flow = average of the average and high values Stripping steam rate: 1.4 lb/gal of feed Bottoms temperature: 232°F 24 actual trays at 24" spacing 25 weight percent ammonia vapor leaving dephlegmator Sulfuric acid (10%) rate = two times the stoichiometric requirement 60-foot high column 400 gallon per minute maximum flow per column
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow per stripping column
COST CURVE SCALE FACTOR:	For two or more operating columns (plus a spare), multiply by: $(\text{number of columns}/2)^{0.8}$
RESIDUES:	Ammonium sulfate recovered as product
MAJOR EQUIPMENT:	Sieve-tray stripping columns, carbon steel Bottoms cooler

Bottoms flash tanks
Dephlegmators
Accumulators
Spray absorption tower
Crystallizer
Centrifuge
Rotary drum dryer
Slurry tanks
Sulfuric acid feed tank

STEAM STRIPPING

Steam stripping causes the separation of water-immiscible (or slightly water-miscible) materials from a wastewater stream by means of direct-contact steam as the heat input. For the purpose of this design calculation, the concentration of the organic pollutant in the influent wastewater stream is assumed to be at or below the solubility of the pollutant at ambient conditions.

The influent stream is preheated by the effluent stream in a counter-current heat exchanger. It enters the stripping column near the top, and cascades down over a number of trays. Steam, which is added at the bottom of the column, causes the organics to vaporize, and the water/organic azeotrope exits at the top. The vapors are condensed, and the water and organic phases separate in a decanter. The organic phase is either recovered or incinerated. The water phase, which is saturated with the organic contaminants, is recycled to the top of the column.

It is possible that the organics removed will be highly toxic. Therefore, the removal efficiency of this unit process must be constant and the operation must be uninterrupted. Accordingly, each steam stripper will be provided with a spare unit at 100 percent of design capacity. This should insure that maintenance problems will not adversely affect the degree of treatment provided.

STEAM STRIPPING

FUNCTION:	Separation of specific dissolved organics from wastewater
PARAMETERS AFFECTED:	Concentration of organics, temperature
EFFECTIVENESS:	Removal to achievable outlet concentration, usually 50 g/l
APPLICATION LIMITS:	TSS: 50 mg/l Oil: 100 mg/l
DESIGN BASIS:	Design flow = 120 percent of the average flow Maximum number of trays = 22 Maximum column diameter = 6 ft Tray spacing = 2.5 ft Organic concentration: No higher than its solubility at ambient conditions
TREATABILITY FACTOR:	Pollutant molecular weight Overall column efficiency Pollutant latent heat of vaporization Achievable effluent concentration (each pollutant) Steam requirement (each pollutant) Vapor-liquid equilibrium ratio. Activity coefficient (deviation from ideal-solution behavior)
COST PARAMETER:	Diameter of the column
COST CURVE SCALE FACTOR:	Number of columns For two or more operating columns (plus a spare), multiply by: $(\text{number of columns}/2)^{0.8}$ Number of trays

RESIDUES:	Distillate is decanted; water phase is returned to column; organic phase is recovered or incinerated.
MAJOR EQUIPMENT	Feed tank, carbon steel Distillation columns with sieve trays, carbon steel Feed preheater, carbon steel Condensers, carbon steel Accumulator/decanter, carbon steel Organic-phase pumps Water-phase recycle pumps Column feed pumps Bottoms pumps

SOLVENT EXTRACTION

This unit process is used for removal of dissolved phenolics or other organics from a contaminated stream in cases where distillation is inapplicable or too costly, such as with some azeotropic mixtures or with mixtures that have components whose boiling points are very close. Extraction is capable of 99.5 percent removal or reduction to an effluent concentration of 10 mg/l.

The optimum solvent for extraction is selected upon consideration of several properties, including; solubility, density (difference between solvent and water), interfacial tension, selectivity, distribution coefficient, chemical inertness, viscosity, flammability, pH, ease of solvent recovery, and cost.

SOLVENT EXTRACTION

FUNCTION:	Removal of dissolved phenolics or other organics into a water-immiscible solvent stream.
PARAMETERS AFFECTED:	Organic or phenolic concentration.
EFFECTIVENESS:	99.5 percent removal or 10 ppm of the pollutant and residual solvent in the effluent.
APPLICATION LIMITS:	Temperature: 50 to 150°F pH: 6.0 to 9.0 TSS: 25 mg/l Solute concentration must be less than the solubility of the solute in water. Boiling point of the organic must be less than 230°C. Removal to 99.5 percent or 10 ppm organic.
DESIGN BASIS:	120 percent of flow Contact time = 20 minutes
TREATABILITY FACTOR:	Distribution coefficient Solubility of the pollutant in water Latent heat of the pollutant

	Pollutant specific heat
COST PARAMETER:	Flow
COST CURVE SCALE FACTOR:	Percent removal efficiency
RESIDUES:	The phenolic or other organic compound is recovered from the solvent by distillation in a solvent-recovery system.
MAJOR EQUIPMENT:	Extraction column with extract reflux, carbon steel Recovered-solvent pump Solvent storage tank Solvent feed pump Pre-heater (solvent and/or water stream), stainless steel Spent solvent pump Spent solvent filter Solvent-recovery distillation column Reboiler Condenser Effluent transfer pump Accumulator Pump for removal of recovered pollutant

COOLING TOWER/HEAT EXCHANGER/STEAM INJECTOR

Cooling towers or heat exchangers may be required to lower the temperature of a wastewater stream for either or both of two reasons. First, if a biological syst is used, its temperature may not exceed 105°F; therefore, the influent stream must be cooled prior to treatment. Second, water quality criteria of the receiving stream may place a maximum temperature limitation on the plant's wastewater discharge; in this case, the treated effluent wastewater must be cooled.

Heat exchangers are used for small flows, and cooling towers for larger ones. Thus, cooling towers are specified whenever the required surface area of a heat exchanger would exceed 5,000 sq ft. Area requirements are based on shell-and-tube, countercurrent heat exchangers. Because the tubes can be individually valved off for maintenance purposes, no spare exchanger is provided. Cooling towers are the mechanical-draft type, which use fans to improve the rate of heat transfer. The water is cascaded down the tower through splash bars, causing continuous shearing that results in maximum contact with the rising air, whose upward flow is induced by a fan at the top of the tower. The tower generally lowers the water temperature to within 3 to 5°F of the wet-bulb temperature of the incoming air. Multiple towers (or cells) are provided whenever practicable.

In the winter, it may be necessary to heat a waste stream prior to biological treatment. This is done by direct injection of steam. An energy balance is used to determine the steam requirements. Because the cost of generating the steam is much greater than the cost of the equipment needed to add it, no capital cost curve (and no design data sheet) is included for this feature of the unit process; onl operating costs are calculated, based on pounds of steam per day.

COOLING TOWERS

FUNCTION:	To cool a wastewater stream to the operating temperature of a subsequent unit process, or to meet receiving stream temperature requirements
PARAMETERS AFFECTED:	Wastewater temperature
EFFECTIVENESS:	Approach to within 3-5°F of wet-bulb temperature of air
APPLICATION LIMIT:	Desired temperature cannot be lower than 5°F above the wet-bulb temperature of air
DESIGN BASIS:	Design flow = 110 percent of the average flow Wet-bulb temperature = 78°F
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow rate
COST CURVE SCALE FACTORS:	Change in wastewater temperature Difference between effluent wastewater temperature and the wet-bulb temperature of the air
MAJOR EQUIPMENT:	Cooling tower cells, wood Top-mounted fans, steel Hot and cold wells, concrete Vertical feed pumps, steel Chlorine storage and feed system, steel

HEAT EXCHANGER

FUNCTION:	Cooling of a wastewater stream in an exchanger, using cowater as the heat-transfer medium.
PARAMETERS AFFECTED:	Wastewater temperature
EFFECTIVENESS:	Cooling normally to within $5^{o}F$ of the inlet cooling water temperature
APPLICATION LIMITS:	Desired temperature cannot be lower than $5^{o}F$ above the inlet cooling water temperature. Maximum heat transfer Area = 5,000 sq ft
DESIGN BASIS:	Countercurrent flow Overall heat transfer coefficient U = 100 Change in cooling water temperature is half the change in the wastewater temperature
TREATABILITY FACTOR:	None
COST PARAMETER:	Heat transfer area
COST CURVE SCALE FACTOR:	None
MAJOR EQUIPMENT:	Concrete wet well with carbon steel feed pumps Shell-and-tube heat exchanger, carbon steel

DEEP-WELL DISPOSAL

Deep wells are used as a means of ultimate disposal of liquid wastes. This unit process, however, is not permitted in all areas of the country, and is used only when in compliance with state and local regulations.

All wastewater outside a pH range of 6.5 to 8.0 must be neutralized prior to injection, and the maximum allowable suspended solids concerntration is 10 mg/l. However, the neutralization and filtration facilities needed to meet these limits are not included as part of this unit process.

All estimates are based on a well depth of 3,500 feet. Flow rates vary from 0.02 to 1.5 MGD, and a constant discharge pressure of 500 psi is assumed.

DEEP-WELL DISPOSAL

FUNCTION:	Ultimate disposal of liquid waste
PARAMETERS AFFECTED:	None
EFFECTIVENESS:	Total disposal
APPLICATION LIMITS:	Where permitted by law Where subsurface geology is suitable Suspended solids 10 mg/l pH 6.5-8.0
TREATABILITY FACTOR:	None
COST PARAMETER:	Flow rate
SCALE FACTOR:	None
RESIDUES:	None
MAJOR EQUIPMENT:	Injection pumps, carbon steel Deep-well, 3,500 ft

LIME HANDLING

Lime (or caustic) addition is required in connection with several unit processes, including neutralization, coagulation, vacuum filtration, pressure filtration, and sludge incineration. If the lime requirement exceeds 8,000 lb/day of hydrated lime, quick lime is used instead. If less than 500 lb/day are needed, lime is replaced by caustic.

Both quick lime and hydrated lime are supplied by truck and unloaded by blowers to storage silos. Automatic feeding equipment is used to slurry the lime to a 10 perc concentration. Slakers are provided when quick lime is involved. Spares are providec for all feeding equipment such as pumps, volumetric feeders, and slakers. Only one lime storage silo (or one liquid caustic storage tank) is provided, since neither of these should have to be taken out of serivce.

The lime slurry is stored in agitated pits, from which it is pumped to the other unit processes. The slurry pipeline is operated as a loop, past all "user" unit proce and then back to the slurry storage pits. The slurry feed pumps run continuously, even when there is no demand for lime. This is necessary to provide a constant moveme in the line to prevent clogging. It also provides sufficient pressure in the line to satisfy the demand immediately.

If caustic is used, the system consists of a liquid caustic storage tank and feec pumps.

LIME HANDLING

FUNCTION:	Provide lime slurry (or caustic) to other unit processes.
PARAMETERS AFFECTED:	None
EFFECTIVENESS:	Not applicable
APPLICATION LIMITS:	500 lbs/day: Caustic 500-8,000 lbs/day: Hydrated Lime 8,000 lbs/day: Quick Lime
DESIGN BASIS:	Lime silo capacity sized for two weeks storage, or

for one week storage plus minimum bulk delivery load, whichever is larger.

TREATABILITY FACTOR:	None
COST PARAMETER:	Lbs/day of hydrated lime
SCALE FACTOR:	None
RESIDUES:	None
MAJOR EQUIPMENT:	Caustic: Caustic storage tank, carbon steel Caustic feed pumps, carbon steel Hydrated Lime: Storage silo, carbon steel Bag filter, carbon steel housing Bin vibrator, carbon steel Lime feeder (plus warehouse spare), carbon steel Slurry tanks with agitators, carbon steel Feed pumps, carbon steel Quick Lime: Storage silo, carbon steel Bag filter, carbon steel housing Bin vibrator, carbon steel Lime slakers with grit collectors, carbon steel Lime feeders, carbon steel Slurry tanks, carbon steel

APPENDIX K

DESCRIPTION OF MODEL COMPONENTS AND USE

APPENDIX K

DESCRIPTION OF MODEL COMPONENTS AND USE

This Appendix describes the computer model developed for EPA by Catalytic, Inc. that can evaluate the costs and performance of various wastewater and sludge treatment technology trains in treating specific wastestreams generated in the Organic Chemicals and Plastics/Synthetic Fibers Industry. The Model has three distinct components: (1) the permanent files which contain data on the design criteria, costs, and performance of the treatment trains, (2) the 28 treatment technology program modules which model the design, performance, and cost of each unit treatment process; and (3) the control programs that sequence the treatment units and estimate the overall costs. Details of each of these three major components are discussed in this section. The relationships between the files and programs are depicted in Figure K-1, and the design assumptions incorporated into each treatment technology module are stated in Appendix J, the Treatment Catalogue.

A. PERMANENT FILES

The Model programs draw data from eight separate permanent files in executing the design and cost estimating routines. These data files are separate from the treatment technology modules and control programs in order to facilitate not only updating the cost and treatment performance information but also overriding the default values built into the Model. During each Model run the user may override the default values with new values.

The eight data files, discussed in sequence below, are:

- Master Process File
- Parameter and Treatment Selection File
- Effluent Target File
- Unit Process Sequence Rules Files
- Plant Adder File
- Capital Costs File
- Operating Costs File
- Costs Allocation Rules File

1. Master Process File. It was not practical to evaluate regulatory options for each of the thousands of OCPSF production plants. Using pollutant loadings obtained during the Verification phase sampling program, responses to Section 308 questionnaires, and engineering judgment, a Master Process File was developed. For 176 major product/processes, this file estimates the flow and loadings for conventional, nonconventional and priority pollutants, as well as rate constants for BOD_5 and COD. The user may employ the Master Process File to simulate wastewaters from the Generalized Plant

Configurations (GPC's) or from any combination of the 176 product/processes. Alternatively, the user can specify other raw waste loads and flows.

2. Parameter and Treatment Selection File. The Parameter and Treatment Selection File contains pollutant-specific treatability information that is used to calculate the treatment performance of eight of the treatment unit processes for each pollutant considered treatable by that technology. The information tabulated for each of the various technologies is:

- Activated Carbon Adsorption -- control constant, two sets of the Langmuir adsorption constants Q and b (one for influent values below the control constant and another for influent values above the control constant), lowest effluent concentration assumed by the Model to be achievable, and Peclet number.

- Activated Sludge -- reaction rate constant and lowest modeled effluent concentration.

- Chemical Oxidation -- applicable oxidizing chemical, ratio of oxidizing chemical to pollutant, and lowest and highest predicted effluent concentrations assumed by the Model to be achievable.

- Chemical Precipitation -- water solubility of the pollutant, applicable coagulating chemical, and ratio of coagulating chemical to pollutant.

- Ion Exchange -- type of resin (e.g., cationic or anionic), resin exchange capacity, lowest effluent concentration assumed by the Model to be achievable, type of regeneration chemical, and dose of regeneration chemical.

- Ozonation -- ratio of ozone to pollutant, lowest and highest effluent concentrations assumed by the Model to be achievable.

- Solvent Extraction -- distribution coefficients for the solvents paraffin and tricresyl phosphate; water solubility, latent heat, and specific heat of the pollutant.

- Steam Stripping -- molecular weight (used in calculating the molar reflux ratio), column efficiency, latent heat of vaporization, lowest predicted effluent concentration, steam dosage, vapor/liquid equilibrium ratio, and activity coefficient.

The Parameter and Treatment Selection File also contains information on how each treatment unit process is typically used (e.g., in-process, pretreatment of comingled streams, or end-of-pipe treatment).

3. Effluent Target File. The Effluent Target File (ETF) contains concentration limitations equivalent to the concentrations presented in EPA's Multi-Media Environmental Goals for Environmental Assessment (IERL, Research Triangle Park, N.C. 1977). Override options allow the user to evaluate any desired set of target concentrations for conventional, nonconventional, and priority pollutants. This override capability facilitates analyzing the sensitivity of treatment cost to target effluent concentration.

4. Unit Process Sequence Rules File. The Unit Process Sequence Rules File contains rules for arranging the unit treatment processes into sequences consistent with OCPSF industry wastewater engineering practice. This file also contains the rules for inserting necessary ancillary unit processes into a treatment train (e.g., addition of nutrients where needed for activated sludge; filtration where necessary before activated carbon adsorption).

5. Plant Adder File. If commanded to do so by the user, this file adds wasteloads and flows from plant washdown, sanitary and utility waste disposal, spills, and other non-process sources of plant waste load. Only flow and conventional pollutants (e.g., suspended solids and BOD) data are contained in this file. To simulate these non-process flows and loadings, the file increases the total flow by 50 percent of the estimated product/process flow calculated by the Master Process File; the file increases the loadings of the individual conventional pollutants by factors ranging from five to fifteen percent.

   Following user option decisions, the Model's programs design and assess performance of various treatment systems using data from the five files discussed above. The data in the three remaining permanent files--the Capital Costs File, Operating Costs File, and Cost Allocation Rules File--are utilized solely for estimating treatment system costs.

6. Capital Costs File. The Capital Costs File contains the equations for capital cost curves for each unit process. Each cost curve was developed by estimating the cost of the entire unit treatment process, including required mechanical equipment, electrical equipment, tanks, piping, and system back-up equipment, for four different sizes of the treatment unit process. Curves relating cost to size were developed from these four estimates. For unit processes where equipment requirements for small systems were significantly different than for large systems, a second costing curve for small units was generated. For those unit process costs described by one curve, precision at the small-system end of the curve was increased by defining the curve in small segments. The Capital Costs File

reflects the CE Plant Cost Index from Chemical Engineering Magazine, July 1977, of 204.7 . The user can update costs by specifying a new Chemical Engineering Index. The July 1982 CE Plant Cost Index value was 314.2.

7. Operating Costs File. The Operating Costs File contains two types of costs or file elements. The first type is organized by unit process and includes the elements listed in TABLE K-1. For each technology, the Operating Cost File contains the wastewater-flow dependent values for shifts per day and service water flow that are necessary to calculate the cost for each element.

   The second set of elements in the Operating Costs File are those items consumed during operation of each treatment technology and include chemicals, electrical energy and other utilities such as fuel oil; the unit costs for these items are listed in TABLE K-2. For each run, each treatment technology program module calculates the quantity of each item consumed annually by operation of the treatment technology. The annual cost of each item consumed is the product of the quantity consumed and the unit cost of the item. The Model user may override any of these unit costs.

8. Cost Allocation Rules File. This file stores the information necessary to allocate capital and operating costs back to each product/process in proportion to that product/process's contribution to the overall loading of those pollutants which necessitate treatment.

In addition to the eight permanent files just discussed, during a run the Model stores relevant portions of the permanent files and any data calculated in temporary working files.

B. TREATMENT TECHNOLOGY PROGRAM MODULES

Design, performance, and cost information on the 31 specific technologies has been programmed into the Model. This technology information differs from the information in the Treatment Catalogue written by Catalytic in the mid-1970's in that this version includes the following: additional unit processes; improved specifications for each technology; allowable influent quality and pollutant removal rates that have been revised to reflect the new data obtained through recent treatability studies and the Section 308 questionnaires; more accurately defined capital and operating cost data and cost scale factors for each unit process; changes incorporating current industry design practices, observed performance, cost information and other comments from reviews by the Chemical Manufacturers Association. The individual treatment technology modules encoded into the computer Model are discussed in Appendix J, The Treatment Catalogue. The 31 technologies include: wastewater treatment technologies; processes, such as nutrient addition to activated sludge, which are ancillary to the wastewater treatment technologies; one wastewater disposal process (deep well injection); and sludge treatment and disposal technologies.

TABLE K-1

COST FACTOR FOR EACH ELEMENT IN OPERATING COST FILE

ELEMENT	COST FACTOR
Direct Labor	Shifts per Day
Supervision	Percent of Direct Labor
Overhead	Percent of Direct Labor
Laboratory Labor	Percent of Direct Labor
Maintenance	Percent of Capital Cost
Services	Percent of Capital Cost
Insurance and Taxes	Percent of Capital Cost
Service Water	Thousand Gallons per Day

TABLE K-2

OPERATING COST FILE
UNIT COSTS

UNIT	COST
Energy	$0.02/kw-hr
Fuel	0.46/gal
Steam	0.0045/lb
Lime	0.0149/lb
Acid	0.0215/lb
Ammonia	0.0789/lb
Phosphate	0.604/lb
Sodium Sulfide	0.1375/lb
$FeCl_3$	0.045/lb
Alum	0.0645/lb
Polymer	2.00/lb
Activated Carbon	0.52/lb
Methanol	0.0696/lb
Waste Hauling	0.0004/lb-mile
Residue Disposal	0.018/lb
Solvent-Undecane	0.0137/lb
Solvent-Tricresyl Phos	0.76/lb
Caustic	0.1575/lb
Chlorine	0.0713/lb
P. Permanganate	0.48/lb
H. Peroxide	0.386/lb
NaCl	0.0199/lb

NOTE: Costs are in July, 1977 dollars.

In addition to the design criteria, sludge generation quantities, and costing data that are generated by the treatment technology programs, limits on allowable influent concentrations and variability are defined within each treatment technology program. The variability (ratio of maximum to minimum concentration) for each pollutant is determined by the values in the Master Process File and is reduced appropriately when waste streams merge. As an example of checking the influent limits, if activated sludge treatment is selected, the influent is checked for temperature, pH, and concentrations of oil and grease, ammonia, TSS, TDS, formaldehyde, sulfide, phenol, and various metals. If the influent waste stream concentration of any of these pollutants violates the concentration limits or variability prescribed as acceptable to the selected treatment process, Model control programs insert one or more appropriate treatment technologies upstream as pretreatment. Only after all parameters satisfy the limits for the treatment technology originally chosen (activated sludge in this example) will the Model design that treatment technology unit. Appendix J discusses and lists the design assumptions and influent quality requirements for each treatment technology.

Using the design assumptions and, as needed, the pollutant-specific information in the Parameter and Treatment Selection File, these technology modules size the treatment facilities, calculate the quantities of sludge generated by the treatment process, and specify the cost and scale variables necessary for estimating the cost of treatment. Seven of the modules (e.g., the activated sludge temperature modification program) merely calculate numbers that are used by other modules and have no internal cost variables. TABLE K-3 lists the cost and scale factors of all the modules except those seven.

C. MODEL LOGIC CONTROL PROGRAMS

The overall model logic control programs were developed to allow maximum flexibility in manipulating the data files and the treatment technology program modules. The ten major control programs, their primary functions, their handling of the permanent files, and user (operator) options are diagrammed in FIGURE K-1 and are discussed next. The Model is not interactive; the user selects his override options, including raw wasteload specifications, before the run begins.

The control programs operate sequentially. The Model may be operated in either of two modes: (1) Model Selection Run Mode, where the Model selects the unit treatment processes, sequences and sizes a system that will treat the raw waste load to meet the target limits and estimates the costs of the system or (2) Specified Unit Process Train mode, where the user selects the major unit treatment processes, and the Model adds necessary ancillary processes, sequences and sizes them, and estimates the costs of the system. For both modes, PARAM, the first program in the control sequence, extracts data from the Master Process File and the Parameter and Treatment Selection File necessary to select the major and ancillary treatment units, as appropriate. The two modes and their routes are described in more detail next.

TABLE K-3

COST AND SCALE FACTORS FOR EACH UNIT PROCESS

UNIT PROCESS	COST FACTOR	SCALE FACTOR
Equalization	Flow Rate	Flow Variability
Neutralization	Flow Rate	Chemical Use Rate
Oil Separation	Flow Rate	--
Dissolved Air Flotation	Flow Rate	--
Coagulation/Flocculation	Flow Rate	--
Clarification	Surface Area	--
Dual Media Filtration	Flow Rate	--
Activated Sludge	Flow Rate	Aeration Time
Aeration	Installed Horsepower per Aerator	Number of Aerators
Nutrient Addition	Nitrogen/Phosphate Deficiency	--
Nitrification	Flow Rate	Aeration Time
Denitrification	Flow Rate	Reaction Time
Ozonation	Ozone Usage Rate and Flow Rate	--
Activated Carbon Adsorption	Working Bed Volume	Number of Beds
Activated Carbon Regeneration	Total Hearth Area	--
Gravity Thickening	Surface Area	--
Aerobic Digestion	Sludge Flow Rate	Hydraulic Retention Time
Vacuum Filtration	Filter Media Surface Area	--
Controlled Landfill	Land Area Requirements from Sludge Application Rate	--
Solid and Liquid Incineration	Sludge Moisture Content	Required Heat Release
Solvent Extraction	Flow	% Removal Efficiency
Ion Exchange	Working Bed Volume and Flow	Number of Beds

-- = No factor.

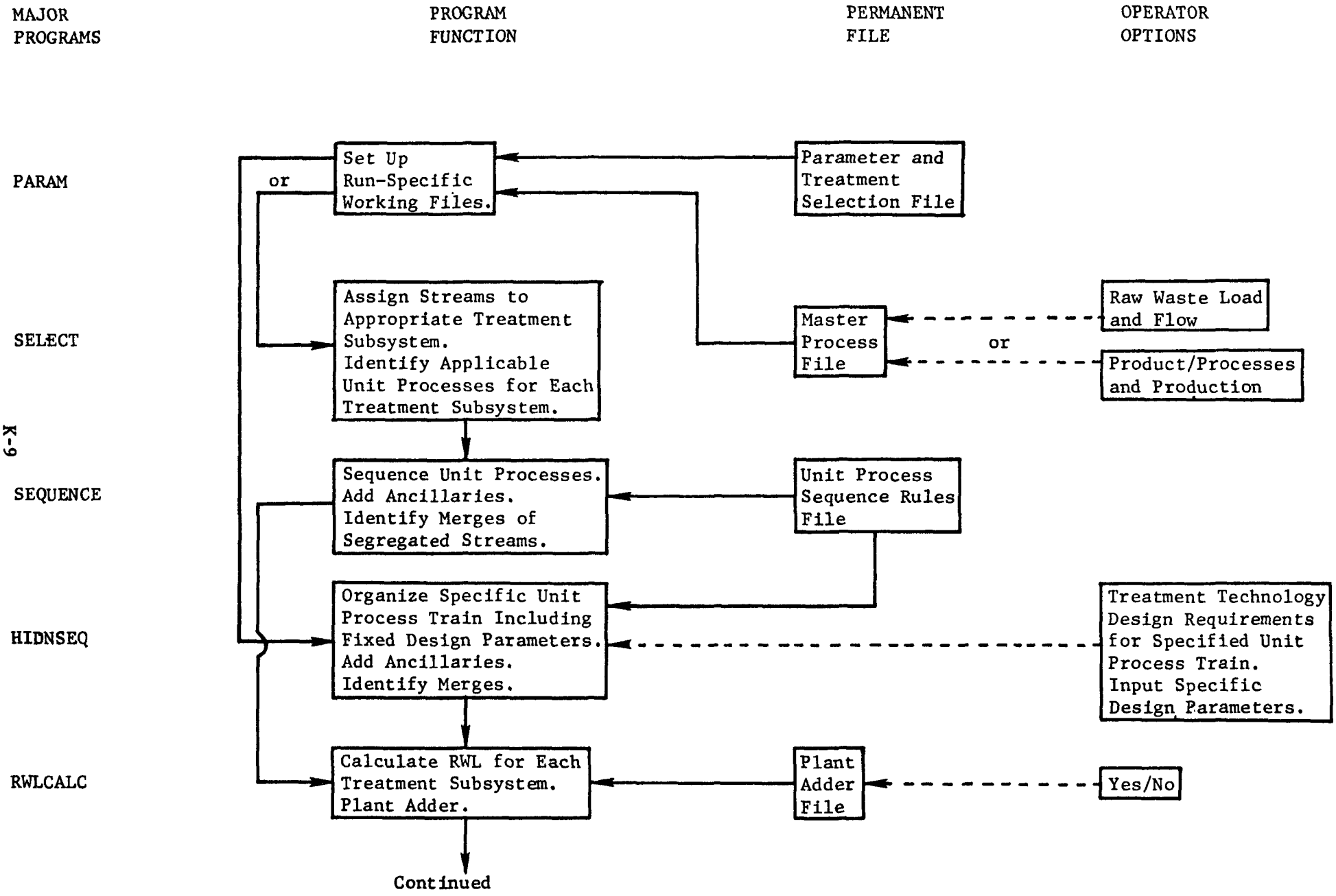

FIGURE K-1

SCHEMATIC OF MAJOR MODEL COMPONENTS

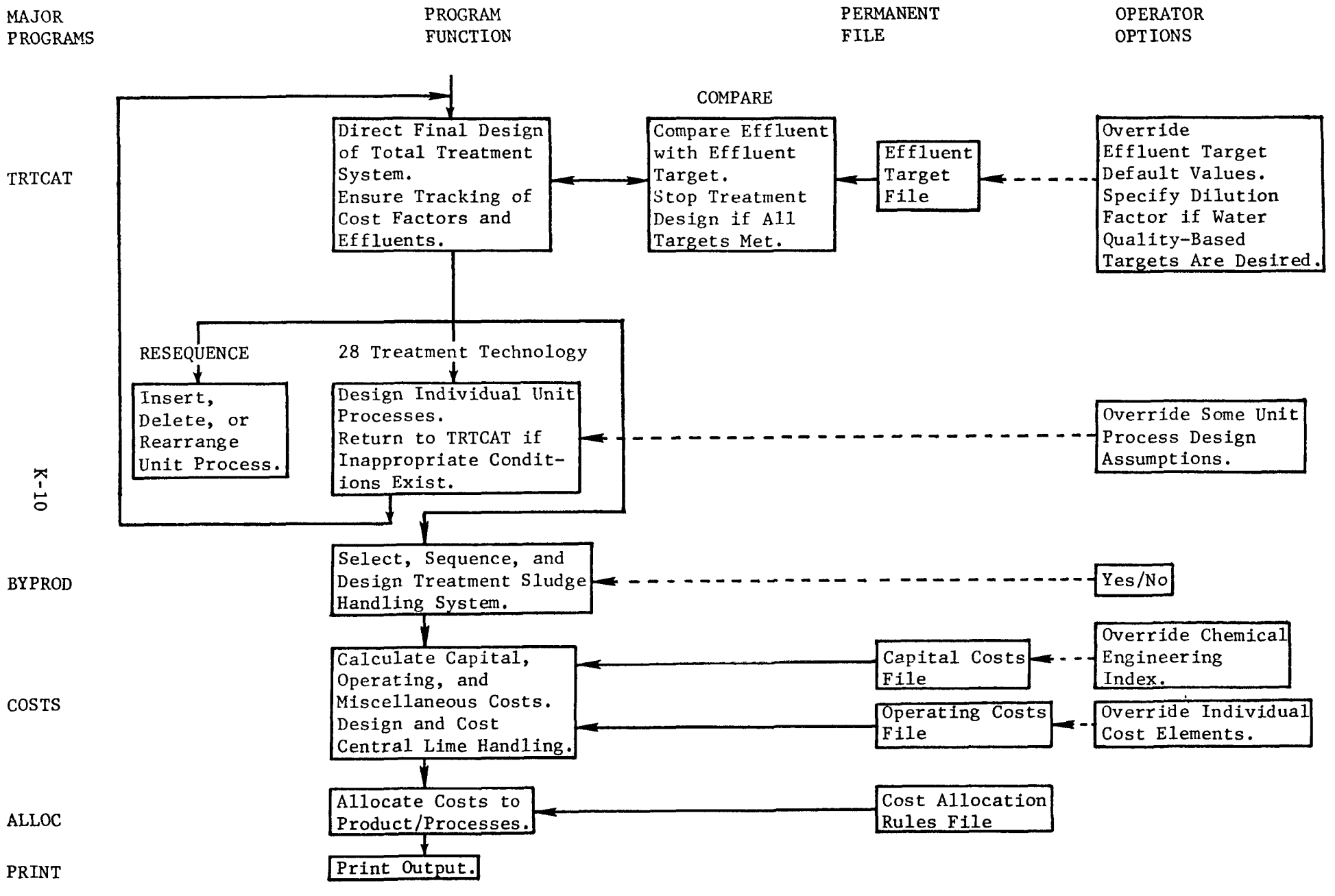

FIGURE K-1 (CONCLUDED)

SCHEMATIC OF MAJOR MODEL COMPONENTS

1. Model Run Modes

a. Model Selection Run Mode

If the Model Selection Run Mode has been input, SELECT develops a skeletal treatment train by first assessing how much of each pollutant in each product/process waste stream each in-process control technology can remove. If removal is sufficient (the precise control percentage varies somewhat depending on operator input), that technology will be considered by subsequent programs in the total treatment design. Selection of applicable end-of-pipe unit processes is then performed similarly.

After the potential treatment technologies for both segregated and combined waste streams have been identified, they are organized by SEQUENCE into the sequence which would typically be found in the industry. The Unit Process Sequence Rules File and information previously extracted from the Parameter and Treatment Selection File contain the information required for the sequencing of the technologies. All flow junctions of segregated streams are identified at this stage. Any redundant treatment unit processes are eliminated and appropriate ancillary processes (e.g., secondary clarification after activated sludge) are inserted.

b. Specified Unit Process Train (SUPT) Mode

In the SUPT mode, the user specifies the treatment train to be used, activating the program HIDNSEQ. Like the SEQUENCE program, HIDNSEQ inserts appropriate ancillary technologies and tracks merge points (flow junctions) as streams are comingled. In addition, HIDNSEQ allows the operator to specify key design parameters for any or all of the technologies included in the overall design.

2. Subsequent Steps for Both Modes

Once the skeletal treatment train has been developed in either the Model Selection or SUPT mode, the control program RWLCALC calculates raw waste loads for segregated and combined waste streams. Where streams are combined, RWLCALC calculates a dampened combined variability at the merge point from the variability data for the individual streams.

To this point in a run, the control programs have primarily combined file data with user input directives to generate a generalized treatment train that treats the specific pollutants in the waste streams. The next program, TRTCAT, coordinates the detailed design of the system through the use of the treatment technology program modules and two auxiliary programs, RESEQUENCE and COMPARE.

TRTCAT starts the actual design by calling the appropriate treatment technology module to design the most upstream unit process in the generalized treatment train. If the constituents in the stream(s) to be treated violate the acceptable influent quality specifications for that technology, RESEQUENCE inserts appropriate pretreatment. Once the pollutant concentration and variability meet the influent quality specifications for the unit process, the treatment technology program sizes the unit and develops cost and scale

factors. Additionally, the program calculates the pollutant reductions achieved by the unit process. Using the waste load data for the treated stream, TRTCAT then designs the next downstream unit process.

Iteration continues until all unit processes (and protective pretreatment, as needed) in the generalized treatment train have been designed. If, however, all effluent targets are met before all the unit processes specified have been designed, the COMPARE program stops adding further treatment, and TRTCAT transfers all design and cost data files to the COSTS program. If the user has chosen to design and estimate costs for appropriate treatment sludge handling processes, the BYPROD program will be executed before COSTS. BYPROD is analogous to a combination of the Parameter and Treatment Selection File and SELECT, SEQUENCE, and TRTCAT, except that it treats the wastewater treatment sludges rather than the wastewater. The BYPRODUCT-SUPT program operates on sludges and is analogous to the SUPT mode for wastewater treatment. Since BYPROD-SUPT was never used during the OCPSF study, it is not depicted in Figure VIII-1.

COSTS is the control program for estimating the treatment systems costs. It calculates individual unit process capital costs from their respective cost and scale factors combined with the Capital Costs File data. The method for calculating operating costs is contained in the Operating Costs File. Because entire lime requirements for a treatment system cannot be determined until the total system is designed, the COSTS program itself designs a central lime handling facility and calculates its costs. COSTS then calculates the miscellaneous costs, such as piping, and adds them into the overall capital cost of the facility.

One final control program is an available option. In OCPSF facilities, one product line may contribute disproportionately to treatment costs. The ALLOC program can allocate both effluents loadings and costs back to the responsible product/processes.

More complete documentation of this Model is in Appendix L of EPA's November 16, 1981, Contractors Engineering Report - Analysis of Organic Chemicals and Plastics/Synthetic Fibers Industries.

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