TECHNICAL SUPPORT DOCUMENT

PART A

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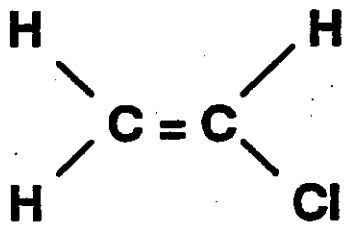

VINYL CHLORIDE

AS A TOXIC AIR CONTAMINANT

OCTOBER 1990

State of California

Air Resources Board

Stationary Source Division

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October 1990

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In preparing this report, the staff reviewed pertinent literature published through December 1989.

TECHNICAL SUPPORT DOCUMENT
PART A

REPORT TO THE AIR RESOURCES BOARD ON VINYL CHLORIDE

PUBLIC EXPOSURE TO, SOURCES, AND EMISSIONS OF VINYL CHLORIDE IN CALIFORNIA

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October 1990

PRELIMINARY EXPOSURE TO, AND SOURCES OF ATMOSPHERIC VINYL CHLORIDE IN CALIFORNIA

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I.

INTRODUCTION

Vinyl chloride (CH_2=CHCl) is a colorless, flammable gas at ambient temperature and pressure. The United States Environmental Protection Agency (EPA) lists vinyl chloride as a Group A carcinogen (Human Carcinogen) and the International Association for Research on Cancer (IARC) lists vinyl chloride as a Group 1 carcinogen (Agent of Human Carcinogenicity).

Part A of this report is an evaluation of vinyl chloride's uses, emission sources, ambient and indoor air concentrations, and population exposure in California. Also included are discussions of the physical properties and atmospheric persistence of vinyl chloride. California Health and Safety Code section 39655 (Assembly Bill 1807, 1983) states that substances listed by the EPA as hazardous air pollutants (section 112 of the Clean Air Act) shall be identified as toxic air contaminants (TACs) by the Air Resources Board (ARB). Therefore, because the EPA has listed vinyl chloride as a hazardous air pollutant, the ARB is directed by statute to identify vinyl chloride as a TAC.

The ARB is the state agency responsible for the identification of TACs in their non-pesticidal uses. The California Health and Safety Code section 39655 defines a TAC as "an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness or which may pose a present or potential hazard to human health". The findings of the Part A report are considered with the health effects findings (Part B report) of the Department of Health Services (DHS) to determine if a compound should be identified as a TAC by the ARB.

Several different limit of detection (LOD) values are mentioned in this report, including those used for: the establishment of the 1978 ambient air quality standard, the South Coast Air Quality Management District (SCAQMD) investigation of BKK and Operating Industries, Incorporated (OII) landfills, the Landfill Gas Testing Program, indoor air studies, and the ARB 20-station ambient toxic air contaminant monitoring network. These LODs indicate levels at or above which vinyl chloride concentrations are not only detectable, but are also quantifiable. In a number of the analyses listed, vinyl chloride was detected below the LODs. This report discusses the derivation of the LOD for each of the analyses.

In 1978, the ARB adopted an ambient air quality standard for vinyl chloride of 10 ppbv averaged over a 24-hour period in response to information associating the development of human cancer with exposure to vinyl chloride. The standard represents the limit of detection (LOD) for vinyl chloride in 1978 and is not currently recognized as health-protective. The standard specifies an analytical procedure with the same method of calculating the LOD as that used by the ARB's Monitoring and Laboratory Division (see Section A, Chapter III and Appendix VII). The identification of vinyl chloride as a toxic air contaminant would allow the implementation of health-protective control measures at concentrations below 10 ppbv.

Vinyl chloride is primarily used for the production of polyvinyl chloride (PVC). PVC is fabricated for use in several products of which many are used by the construction industry. Finished commercial PVC products are not expected to be significant sources of vinyl chloride due to current processing and shipping procedures. In California, the identified sources of vinyl chloride emissions are: landfills, publicly-owned treatment works (POTWs), and PVC production and fabrication facilities.

Available information shows that landfills are a potential major identified source-category of vinyl chloride emissions in California. Vinyl chloride has been detected in the ambient air near landfills as well as in the internal gas of landfills. Additional studies have shown that vinyl chloride can be formed in the many landfills where chlorinated organic compounds were disposed as well as landfills where vinyl chloride and halogenated industrial waste were disposed.

In this report, ambient monitoring data and meteorological data are used with an atmospheric dispersion model to estimate population exposure to vinyl chloride near two California landfills. The modeling estimates show that people living near these landfills may have been exposed to elevated levels of vinyl chloride. Also, preliminary data from the Landfill Gas Testing Program required by section 41805.5 of the 1986 California Health and Safety Code are presented (see Appendix VI for a table of the Landfill Gas Testing Program data). The results indicate there is a potential for elevated ambient vinyl chloride exposure for people residing near other landfills.

In addition to estimating ambient air exposure, this report also evaluates indoor air exposure to vinyl chloride. Based on limited monitoring data, indoor air exposure to vinyl chloride is probably not significant for the majority of the population. However, for people residing near some landfills, inhalation of indoor air may represent the most significant source of vinyl chloride exposure. This is because vinyl chloride can migrate underground from landfills and accumulate in nearby structures.

II.

PRODUCTION, USES AND EMISSIONS

Although vinyl chloride is not produced in California, several thousand tons are used each year in the state for the production of polyvinyl chloride (PVC). The PVC which is produced is primarily used by fabricators for the production of materials used by the construction, packaging, electrical, and transportation industries.

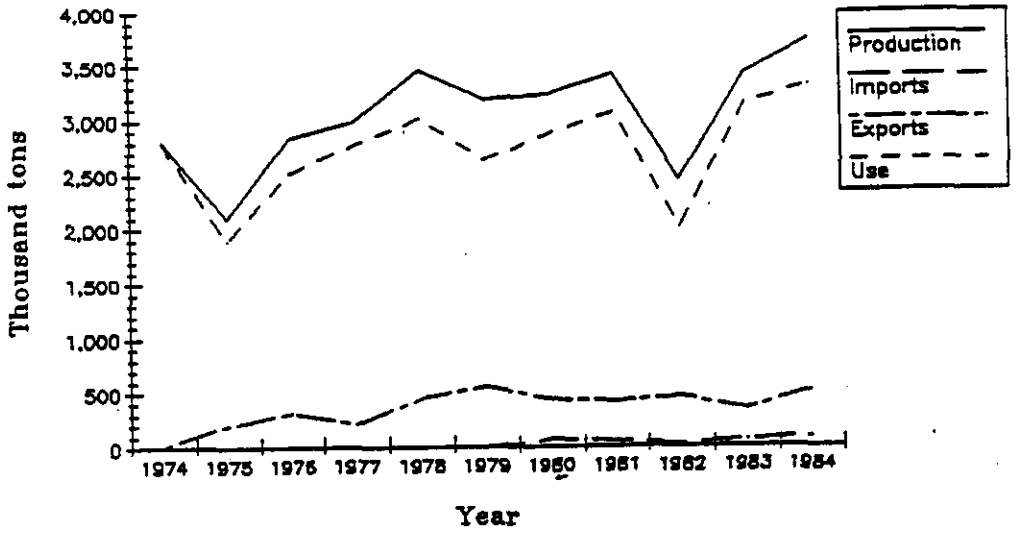
Based on available data, landfills are a potential major identified source-category of vinyl chloride emissions in California. Other known emission sources of vinyl chloride in the state include PVC production and fabrication facilities, and sewage treatment plants.

A. PRODUCTION

Commercial production of vinyl chloride in the United States began in 1936. During the first year of production, two thousand tons were produced (CEN, 1984). With a reported annual U.S. production of 3.8 million tons, vinyl chloride ranked 21st on a list of the most produced chemicals in the United States in 1984 (CEN, 1985a). Figure II-1 shows the production, imports, exports, and use of vinyl chloride from 1974 through 1984 (CEN, 1985a: US DOC, 1985a; and US DOC, 1985b]. During this 10-year period, vinyl chloride production increased at an average annual rate of 3% (CEN, 1985a). More recent estimates for U.S. vinyl chloride production are 4.7 million tons and 4.2 million tons for 1985 and 1986, respectively (CEN, 1987).

FIGURE II-1

NATIONAL VINYL CHLORIDE PRODUCTION, IMPORTS, EXPORTS, AND USE



Two facilities in California currently use vinyl chloride to produce PVC. Two other facilities in the state that were producing PVC ceased production, one in 1982 and the other in 1985 (Personal Communication, 1985a, 1985b, 1985c; and Zwiacher, W. et al., 1983).

B. CURRENT AND PROJECTED USES

About 96 Percent of the vinyl chloride produced in the U.S. is used to manufacture PVC. The remainder is either exported or used to manufacture 1,1,1-trichloroethane (methyl chloroform) (U.S, DH&HS, 1978; and McPherson, W., 1979). Sixty percent of the PVC is used for fabricating various plastic materials used by the construction industry. Specifically, PVC is used by the construction industry for pipe fittings, flooring, paneling, and roofing. PVC is also used by the packaging, electrical, furnishings, transportation, recreation, apparel, and medical industries.

The growth of the vinyl chloride industry is closely tied to PVC use. Historical data for California show the number of housing units in the construction industry increased from approximately 1.0 million units in 1981 to 1.8 million units in 1986 (U.S, DOC, 1987). If this growth in the construction industry continues, the PVC use by this industry is also expected to increase. Data are not available to forecast the use of PVC in other sectors. However, the total United States demand for PVC has been forecasted to increase by approximately 3 to 5 percent annually from 1985 to 1990 (CMR, 1985).

C. LANDFILLS: A POTENTIAL MAJOR EMISSION SOURCE

Landfills are estimated to be a potential major source-category of vinyl chloride emissions in California. However, because landfills vary in the amount and composition of wastes they accept as well as the waste disposal methods used, estimating total vinyl chloride emissions for the

state's hundreds of landfills is not possible. To better understand why all landfills are potential vinyl chloride emission sources, this section presents information on the types and number of landfills in California, the disposal methods employed, the causes of vinyl chloride emissions from landfills, vinyl chloride emission estimates for landfills, and some methods used to control landfill emissions.

1. Types of Landfills

There are three types of landfills in California: Class I sites (e.g., BKK, located in West Covina) which accept all types of wastes including hazardous materials; Class II sites [e.g., Operating Industries, Incorporated (OII), located in Monterey Park] which normally accept only "non-hazardous" wastes but can accept certain types of hazardous wastes (ARB, 1982b); and Class III (municipal or sanitary landfills) sites which can accept only household wastes. In California, there are twenty Class I sites (at present, only two of the 20 sites are accepting hazardous waste), approximately 200 Class II sites, and approximately 2000 unclassified and Class III sites (ARB, 1982b; WRQCB, 1990).

2. Land Disposal Methods

Landfarming, surface impoundments, and landcovering are often used as waste disposal methods in California. These disposal methods may be practiced by more than one type of landfill. For instance, any of the three types of landfills may employ landcovering as a disposal method. However, only Class I and II sites may contain surface impoundments. In addition, the same landfill may employ more than one disposal method. For landfarming, heavy oil sludge is spread several inches thick over the land. The sludge is then cultivated into the soil at frequent intervals. This cultivating process ensures a better aerobic decomposition of the wastes (Thibodeaux and Hwang, 1982). Surface impoundments, often called evaporation ponds or lagoons, are used to dispose of certain types of liquid wastes. As the name implies, surface impoundments allow the wastes to be evaporated into the atmosphere.

Landcovering is most often used at Class III sites or municipal landfills. In landcovering, wastes are spread over the land. At the end of each day, the wastes are covered with approximately six inches to 12 inches of cover. Ultimately, the wastes are covered with a layer of cover material that is at least four feet deep.

3. Landfill Emissions

Emissions of vinyl chloride from landfills mainly occur by two mechanisms: 1) direct vinyl chloride emissions from disposed wastes which contain vinyl chloride; and 2) the formation of vinyl chloride from the biodegradation of chlorinated hydrocarbons. Other minor mechanisms by which vinyl chloride emissions may occur include chemical reactions such as pyrolysis, surface photolysis, and hydrolysis of trichloroethylene and other chlorinated hydrocarbons, and off-gassing of PVC (Molton et al., 1987).

Direct Emissions. Direct emissions of vinyl chloride can only occur at landfill sites where vinyl chloride containing wastes were previously disposed. Because vinyl chloride containing wastes cannot be legally disposed in Class II or Class III landfills, Class I landfills (e.g., BKK) at which vinyl chloride has been disposed are probably the largest source of direct emissions of vinyl chloride. However, because vinyl-chloride-containing wastes may have been illegally disposed, Class II and Class III landfills may also emit vinyl chloride directly.

Formation of Vinyl Chloride. Because vinyl chloride can be formed from the biodegradation of chlorinated wastes, emissions of vinyl chloride may occur from any landfill site including Class II and Class III sites where no vinyl chloride has been disposed. Of the three landfill disposal methods, it appears that landcovering and landfarming are most likely to produce the conditions necessary for the formation of vinyl chloride.

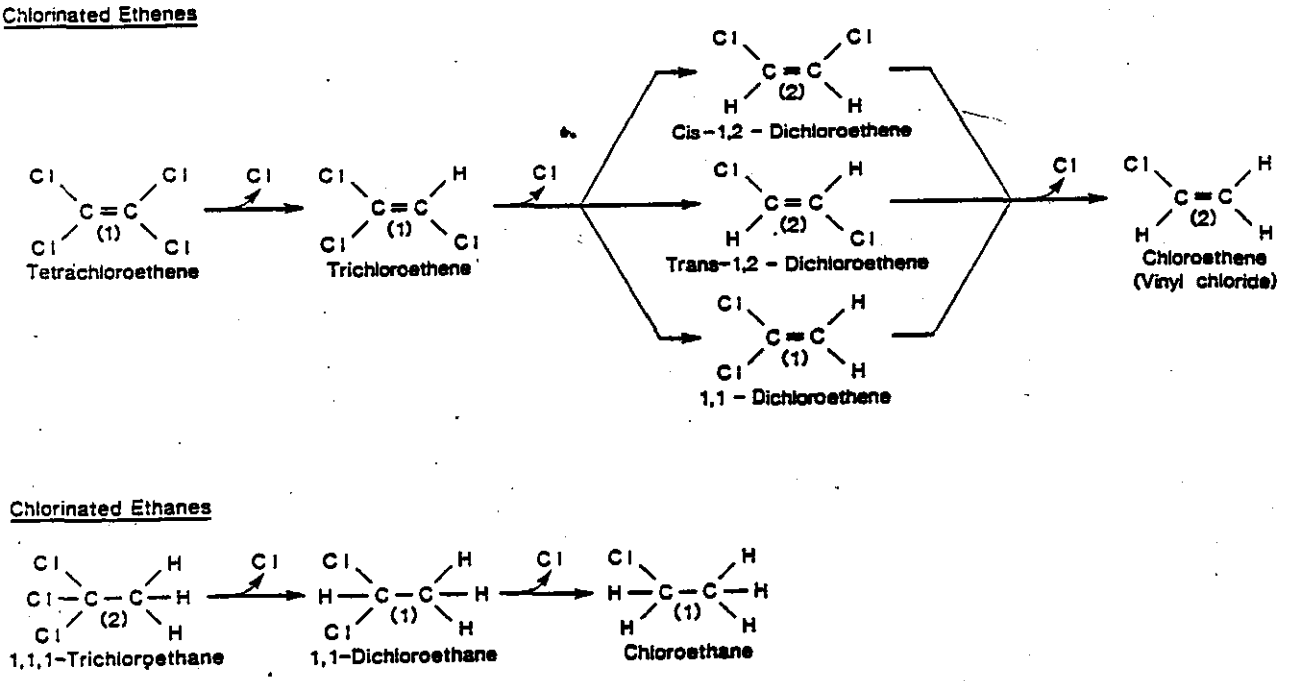
Results of an ARB sponsored study demonstrated the formation of vinyl chloride when soil samples from two municipal landfills were incubated with chlorinated hydrocarbons (Molten et al., 1987). Similar results were obtained when sludge samples were incubated with chlorinated hydrocarbons. The evaluation of the biological mechanism showed that vinyl chloride production occurred predominantly under anaerobic (without oxygen) conditions. Subsequent experimentation with carbon-13 labeled chloroethanes and chloroethenes yielded carbon-13 labeled vinyl chloride as well as other biodegradation products. These results are in agreement with other studies which evaluated the biodegradation of chlorinated hydrocarbons to produce vinyl chloride (Kleopfer, 1985; Beeman, et al., 1978; Wood et al., 1980; and Parsons et al., 1984). Figure II-2 illustrates the pathways by which vinyl chloride is formed from the dehalogenation (chlorine removal) of chlorinated ethenes and ethanes. In addition, the figure indicates the relative rate by which the various compounds are degraded. Not all of the compounds presented in this scheme have necessarily been unequivocally demonstrated to form vinyl chloride. However, given the current state of information, they should be regarded as vinyl chloride precursors.

Although the disposal of halogenated wastes from industrial operations is now substantially restricted, for decades these materials were disposed in Class I landfills as well as some Class II landfills throughout the state. The halogenated wastes are composed of many of the chlorinated compounds which can lead to the formation of vinyl chloride. However, the amount of halogenated wastes previously disposed in these facilities is unknown. Therefore, without monitoring data that shows otherwise, all Class I and Class II facilities (this includes open and closed facilities) should be regarded as potential vinyl chloride emission sources.

Industrially generated halogenated wastes were never permitted to be disposed in Class III facilities. However, many of the chlorinated compounds which can lead to the formation of vinyl chloride are used extensively in consumer products, which after use typically end up in Class III landfills. The amount of chlorinated compounds remaining in consumer products and disposed in landfills is not known. However, because of the widespread use of these compounds in consumer products, all Class III landfills (this includes open and closed facilities) should be regarded as potential vinyl chloride emission sources.

FIGURE II-2

ANAEROBIC BREAKDOWN SEQUENCE VIA REDUCTIVE DEHALOGENATION



(1) - Substantial degradation

(2) - Slow degradation

Source: Cline and Viste, 1984.

Methods of Estimating Landfill Emissions. Several models have been developed to estimate volatile organic gaseous emissions from hazardous waste landfills (Thidobeaux, 1981; Hwang, 1982; Shen, 1981; and Hartley, 1969). The models usually apply to specific landfill operations such as landfarming, surface impoundments, etc. However, these models are difficult to use because they require a number of input parameters such as waste composition, wind speed, and ambient conditions which are not commonly known. These models involve the use of Fick's Law (Fick's Law describes the diffusion of a species through a layer of fluid) and may be appropriate for estimating direct emissions of volatile compounds such as vinyl chloride. However, because the models do not consider factors such as formation, they may not be appropriate for estimating vinyl chloride emissions where formation is occurring.

A method to estimate vinyl chloride emissions where formation may be occurring is to establish monitoring stations around landfill sites to measure the 24-hour average ambient concentrations of the compounds of interest. The ambient concentrations along with appropriate meteorological data can then be used in dispersion models to back-calculate the emission rate from the landfill site. This is the method that was used to estimate vinyl chloride emissions from BKK and OII.

Landfill Emission Estimates. Table II-1 summarizes vinyl chloride emission estimates for the state's landfills. As indicated in the table, vinyl chloride emissions have been estimated for BKK and OII landfills. The vinyl chloride emission estimates for BKK and OII make several assumptions. These assumptions are: 1) vinyl chloride is emitted from an area of approximately 1,700,000 meters^2 for BKK and 330,000 meters^2 for OII; 2) annual average emission rates of vinyl chloride from BKK and OII are within the ranges estimated in Table III-4; and 3) emissions of vinyl chloride are uniform over the entire area of the landfill that is estimated to emit vinyl chloride.

Further testing is necessary to estimate emissions from landfills other than BKK and OII. Thus, for the 1987 and 1986 inventory years, total vinyl chloride emissions from California landfills were probably greater than those estimated in this report.

TABLE II-1

VINYL CHLORIDE LANDFILL EMISSION ESTIMATES*

Source	Source Type	Emissions (tons/year)	Inventory Year	Ref.
Class I Landfills				
BKK, West Covina	Area	44-197	1987	ARB,1988b
Other Sites	Area	NA		
Class II Landfills				
OII, Los Angeles	Area	4-51	1986	ARB, 1988b
Other Sites	Area	NA		
Class III Landfills	Area	NA		

* - These emission estimates assume that the vinyl chloride emission rates are uniform throughout the year over the area of the landfill that is estimated to emit vinyl chloride.
NA - Not Available

Based on 1987 monitoring data for BKK, ARB staff estimate a vinyl chloride emission rate ranging from 0.75 to 3.32 micrograms meter^{-2} second^{-1} (see Table III-4). For BKK landfill, this translates to estimated vinyl chloride emissions ranging from 44 and 197 tons per year. Over BKK's history it is not known how much vinyl-chloride-containing or halogenated wastes were disposed at the landfill. However, in 1984, BKK received approximately 136,000 tons of volatile or toxic wastes. An unknown portion of these wastes were halogenated solvents (ARB, 1982b). For OII, ARB staff estimated a vinyl chloride emission rate ranging from 0.31 to 4.42 micrograms meter^{-2} second^{-1} (Table III-4). For the OII landfill, this translates to estimated vinyl chloride emissions ranging from 4 to 51 tons per year. The amount of halogenated wastes disposed at OII over its history is unknown. However, in 1982, OII received 9,200 tons of volatile or toxic wastes. As with BKK, an unknown portion of these wastes were halogenated solvents (ARB, 1982a).

Monitoring results available for several other landfills are as follows: Flux measurements on the surface of the Scholl Canyon sanitary landfill (a former Class II landfill located in Glendale, California) showed vinyl chloride concentrations ranging from non-detectable to 180 ppbv (parts per billion by volume) at various locations (Todd and Propper, 1985). In addition, tests conducted by the SCAQMD at several other Class II landfill sites from 1981 to 1985 confirmed the presence of vinyl chloride in landfill surface gas or gas collection systems (Coy, 1985).

To partially address the lack of monitoring data from other landfills throughout the state, Health and Safety Code section 41805.5 (AB 3525 and subsequent amendments by AB 3374) required the development and implementation of landfill monitoring guidelines and the reporting of monitoring results. The law required the ARB to establish guidelines for landfill operators to monitor gas migration, gas constituency, and the ambient air at many of the hazardous and municipal waste landfills in California. The testing guidelines identified vinyl chloride as one of the compounds requiring monitoring. Although the choice of vinyl chloride analytical methods was left to individual laboratories performing the analysis, the guidelines specified an ambient vinyl chloride detection limit of 2 ppbv and provided an example of a vinyl chloride method with an achievable detection limit of 2 ppbv. In this example method, the limit of detection (LOD) is based on 3 standard deviations of runs near the method detection limit (within 10 standard deviations of the method detection limit) (ARB, 1986; ARB, 1987). This means of calculating the LOD is the same as that used by the ARB's Monitoring and Laboratory Division (see Appendix VII). 24-hour ambient vinyl chloride concentrations ranging from the detection limit of 2 ppbv to 15 ppbv were detected at 24 (10 per cent) out of the 251 landfills at which ambient monitoring was performed. Vinyl chloride concentrations ranging from the detection limit (see Appendix VI, Table 1 for the method of determining the detection limit) of 106 ppbv to 72,000 ppbv were detected in the internal gas of 160 (47 per cent) out of the 340 landfills at which internal gas testing was performed (Appendix VI).

4. Gas Collection Systems

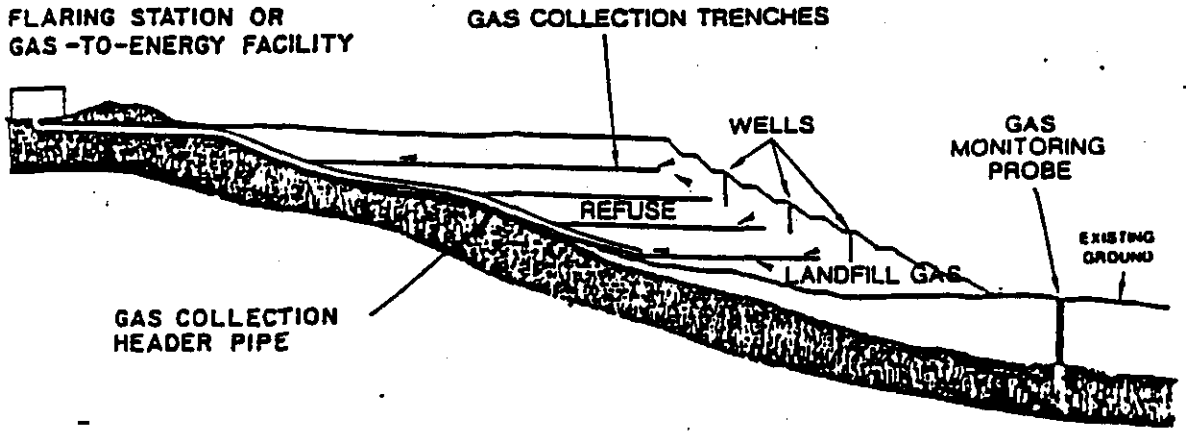
For some landfills, emissions are required to be controlled to reduce odors as well as emissions of methane and toxicants. However, gas

control systems have been installed at some landfills as a resource recovery and/or energy conservation measure. For example, BKK transmits collected landfill gases to either one of two flare stations and/or to a five megawatt gas turbine for use as a fuel in generating electricity. Both well (vertical piping) and trench systems (horizontal piping) are used to collect landfill gases. In 1983, BKK installed a number of wells and gas collection lines to help control gaseous emissions. Although there are still potential sources of gaseous emissions such as cracks at the landfill surface, pipe connections and valves, and burner exhaust, ambient concentrations of vinyl chloride near BKK have been declining. Since installing their gas collection system, BKK has continued to expand the system by adding wells and trenches. Since installing a gas collection system at OII, ambient concentrations at the perimeter of the facility have continued to decline. Due to the lack of violations of the state standard for vinyl chloride (10 ppbv), ambient monitoring at the perimeter of OII was discontinued by the SCAQMD in early 1987. Currently, OII Landfill is a federally listed superfund site managed by the EPA.

A well system consists of a network of wells drilled vertically into the refuse to collect the generated gases. These wells are connected to collection pipelines where gases are withdrawn from the buried layers of waste. In general, a vertical gas well is constructed by drilling a 30-inch diameter hole 50 to 100 feet deep into the wastes. Perforated PVC pipes are then placed inside the hole. The space between the pipe and the hole is backfilled with uncrushed gravel (Sanitation Districts of Los Angeles County, 1984). A typical gas control system showing both well and trench systems is shown in Figure II-3.

FIGURE II-3

LANDFILL GAS COLLECTION SYSTEM



Source: Sanitation Districts of Los Angeles, 1984.

In the trench system, a network of perforated pipelines is laid in trenches within the waste at approximately 200-foot intervals horizontally and 80-foot intervals vertically. To support the pipes and to allow the migration of the generated gases, approximately 2 feet of uncrushed gravel are packed around the pipelines. These pipelines are then connected to a main collection pipe where gases are withdrawn (Sanitation Districts of Los Angeles County, 1984).

D. OTHER KNOWN EMISSION SOURCES

Other than landfills, emissions of vinyl chloride occur from: PVC production and fabrication, publicly-owned treatment works (POTWs), ethylene dichloride production, vinyl chloride production, methyl chloroform production, caprolactam production, and incomplete incineration of chlorine containing materials (Sittig, M., 1981; Zwiacher, W., et al., 1983; and Lamorte, M., 1978). In California, the identified sources of vinyl chloride emissions that can be quantified are PVC production, PVC fabrication, and POTWs. Table II-2 provides estimates of vinyl chloride emissions for identified sources. Currently, there are no known vinyl chloride, ethylene dichloride, methyl chloroform (TCA) or caprolactam production facilities operating in the state.

TABLE II-2

SUMMARY OF VINYL CHLORIDE EMISSION ESTIMATES FOR OTHER SOURCES

Source	Source Type	Emissions Tons/Year	Inventory Year	Reference
PVC Production	Point	<0.5	1988	ARB, 1988b
PVC Fabrication	Point	0.75	1982	Zwiacher, et al., 1983
POTWs	Point	1.7	1985	Chang, et al., 1987
On-site Wastewater Treatment Plants	Point	NA		
Waste Incinerators	Point	NA		
Transportation and Accidental Spillage	Area	NA		

NA - Not Available

1. Polyvinyl Chloride (PVC) Production

Three PVC producers reported emitting a cumulative total of 3 tons of vinyl chloride in 1984 (Personal Communication, 1985a, 1985b, and 1985c). In 1982, vinyl chloride emissions from these producers were estimated to be 1.4 tons (Zwiacher, W., et al., 1983). All three producers reported that they were in compliance with the South Coast Air Quality Management District's (SCAQMD's) Rule 1163. This rule requires that vinyl chloride emissions from designated plants not cause ambient vinyl chloride levels to exceed 10 ppbv (parts per billion by volume) during any 24-hour period when measured beyond the plant's property line (Personal Communication, 1985a, 1985b, 1985c; and ARB, 1980). Rule 1163 was adopted by SCAQMD as part of their program to control vinyl chloride emissions to 10 ppbv.

In 1984, the PVC producers operating in the state reported using closed systems, incineration, routine leak surveys, and maintenance programs as control technologies to comply with existing standards for vinyl chloride emissions (Personal Communication, 1985a, 1985b, and 1985c). The primary control method used by the two PVC producers currently operating in California is incineration. One facility (facility A) uses an afterburner with an operating temperature of approximately 2000^{o}F while the other facility (facility B) uses a catalytic-type incinerator. Both facilities have a monitoring system that continuously measures the vinyl chloride concentration within various areas of the plant. Portable hydrocarbon (HC) detectors are used to pinpoint leaks detected by the area monitoring system. These plants are also inspected at least once a year by the SCAQMD Enforcement Division to ensure compliance with district rules (Personal Communication, 1985d).

The SCAQMD periodically conducts ambient monitoring for vinyl chloride near the two PVC producers in California. In addition to the SCAQMD's monitoring program, the SCAQMD requires one of the PVC producers (facility A) to monitor the ambient air for vinyl chloride at the perimeter of their facility on a daily basis. The other PVC producer (facility B) is not required by the SCAQMD to conduct ongoing offsite ambient monitoring for vinyl chloride. This is because: 1) historically, the facility has not exceeded the ambient air quality standard for vinyl chloride; and 2) the process that is used to manufacture latex emulsions is not expected to result in vinyl chloride emissions as great as those associated with the other facility which produces PVC resins. Generally, 24-hour average concentrations near these facilities are below the 10 ppbv standard. However, in October of 1988, the SCAQMD reported concentrations as high as 20 ppbv for facility A (Molita, 1989). As a result, the SCAQMD plans to conduct ambient monitoring more frequently at this facility to ensure compliance with the ambient air quality standard for vinyl chloride. The SCAQMD's monitoring results indicate that this PVC producer may contribute to the public's exposure to vinyl chloride. Therefore, this facility should be investigated in more detail when considering control measures to reduce the public's exposure to vinyl chloride.

Table II-2 lists the cumulative vinyl chloride emissions estimates from the two PVC producers in California at less than 0.5 tons for 1987. This estimate is substantially lower than the 1984 estimate of 3 tons when three PVC producers were operating in California (Personal Communication, 1985a, 1985b, 1985c).

2. Polyvinyl Chloride Fabrication

Polyvinyl chloride (PVC) can be fabricated into several products such as PVC pipes, pipe fittings, plastics, etc. Some major fabrication processes are extrusion (to shape by forcing through a die), calendering (to press between rolling cylinders), molding, and bonding. PVC contains the vinyl chloride monomer as a residual from the PVC production processes. Residual vinyl chloride (RVC) in PVC ranges from 0.002 ppmw (parts per million by weight) to 10 ppmw (U.S. EPA, 1982). When PVC is fabricated into final products, vinyl chloride is emitted.

The SCAQMD identified 33 PVC handling and fabrication facilities under its jurisdiction with an estimated usage of 75,000 tons of PVC in 1982. The SCAQMD staff assumed that all vinyl chloride is emitted from the fabrication processes. Using this assumption and a maximum RVC of 10 ppmw in PVC, the SCAQMD estimated that these handling and fabrication facilities emitted approximately 0.75 ton of vinyl chloride in 1982 (Zwiacher, 1983). This estimate represents an upper-bound condition because the maximum RVC was used to estimate emissions, and because all RVC from the incoming PVC was assumed to be emitted from the fabrication processes. The vinyl chloride migration studies conducted by the Environmental Protection Agency (EPA) indicated a much smaller percentage of monomer is released during fabrication (U.S. EPA, 1982). A typical release of vinyl chloride in the extrusion process was only 10 percent of that in the PVC (U.S. EPA, 1982).

3. Publicly-owned Treatment Works

Publicly-owned treatment works (POTWs) are wastewater treatment plants that are owned by public entities, and which consist of wastewater collection systems, wastewater and sludge treatment facilities, and effluent and sludge disposal systems. Users that discharge wastewater into POTWs are normally classified as commercial, industrial, and residential. The two primary mechanisms that result in emissions of organic gases are volatilization and biodegradation. Because POTWs treat wastewater which can contain vinyl chloride and halogenated compounds from industries, vinyl chloride can be volatilized during the treatment processes. In addition, chlorinated hydrocarbons such as trichloroethylene and 1,2-dichloroethane could be biodegraded to vinyl chloride.

Halogenated hydrocarbons including vinyl chloride have been measured at wastewater treatment plants throughout the nation, including California (U.S. EPA, 1980). A preliminary study of two wastewater treatment plants, one in Los Angeles and another in the Sacramento Valley, indicated that vinyl chloride was present in the anaerobic digester tanks. Concentrations of up to 2.6 ppmv have been measured (ARB, 1985). These digester tanks are equipped with pressure/vacuum (P/V) valves to equilibrate the inside and outside pressure of the tanks. These P/V valves are potential sources of vinyl chloride emissions along with fugitive emissions associated with pipe fittings and valves.

In a study performed by the University of California at Davis (UCD), researchers used a mass balance approach to estimate that approximately 1.7 tons of vinyl chloride were emitted by POTWs in California in 1985 (Chang et al., 1987). Specifically, the difference between the concentration of vinyl chloride in the POTW influent and effluent was assumed to be emitted to the atmosphere. This approach may be useful in assessing which POTWs constitute a threat to public health. However, because this approach does not take into account the formation or degradation of vinyl chloride within POTWs, the resulting emission estimates should only be considered rough approximations. In response to the need for more information concerning emissions of toxicants from POTWs, ARB is currently funding a research contract. When the research is complete, the resulting report will contain the most recent information concerning the estimation of emissions of toxicants from POTWs and POTW collection lines. The report will also address the efficacy of POTW odor control systems on reducing emissions of toxic compounds.

E. OTHER POTENTIAL EMISSION SOURCES

Along with the sources discussed in Section D, there are several other potential sources of vinyl chloride emissions in California. These include on-site wastewater treatment plants, incineration of PVC materials, and transportation of vinyl chloride.

1. On-site Wastewater Treatment Plants

As presented in the discussion on POTWs, wastewater treatment facilities are sources of vinyl chloride emissions. At several industrial facilities such as oil refineries, chemical manufacturers, etc., industrial wastewater is normally treated before being discharged. These wastewater treatment plants are also potential sources of vinyl chloride emissions.

2. Waste Incinerators

Vinyl chloride has been identified as a combustion product in the flue gas of an incinerator burning plastics (Boettner et al., 1973). It has also been hypothesized to form upon the combustion of PVC materials (Ahling et al., 1978). PVC materials are used extensively in automobile's upholstery, bumper parts and floor mats. When these materials are incinerated, vinyl chloride is a likely pollutant in the incinerator exhaust. Hospital waste incinerators are another potential source of vinyl chloride emissions since much of the hospital waste such as syringes and plastic bags are PVC-containing materials.

3. Transportation and Accidental Spillage

Another potential source of emissions is the accidental spillage and/or leakage of vinyl chloride that is being transported either by rail car, tank car, or marine vessel. Vinyl chloride is transported by rail cars to the two PVC producers currently operating in California. As far back as records are available, there have been no reported accidents involving vinyl chloride in the state (Office Of Emergency Services, 1985; California Highway Patrol, 1985).

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III.

EXPOSURE TO VINYL CHLORIDE

A. AMBIENT MONITORING IN CALIFORNIA

To date, vinyl chloride has not been detected in California's ambient air except near known emission sources. However, in order to assure that vinyl chloride continues to pose no significant health risk in the background atmosphere of the state, a vinyl chloride screening procedure is routinely performed by the ARB's laboratory on samples collected from the state's 20-station ambient toxic air contaminant network. Bimonthly through-the-probe audits by the ARB Quality Assurance Section using known concentrations of vinyl chloride confirm that the screening procedure detects the presence of the compound. The screening procedure has not detected vinyl chloride in samples collected at the monitoring stations of the toxic air contaminant ambient monitoring network since the implementation of the procedure in 1988. Should vinyl chloride be detected, the ARB's Monitoring and Laboratory Division would begin immediate monitoring and investigation of the cause. Appendix VII describes the method for calculating the limit of detection (LOD) used by the ARB's Monitoring and Laboratory Division.

California Health and Safety Code Section 41805.5 required hazardous and municipal landfills throughout the state to conduct monitoring for several contaminants including vinyl chloride. 24-hour averaged ambient vinyl chloride concentrations ranging from the detection limit of 2 ppbv (the Testing Guidelines for Active Solid Waste Disposal Sites example method for calculating the LOD is discussed in Section C of Chapter II under Landfill Emissions Estimates) to 15 ppbv were detected at 24 out of the 251 landfills tested for ambient concentrations (see Appendix VI).

In recent years, the South Coast Air Quality Management District (SCAQMD) frequently measured 24-hour average ambient vinyl chloride concentrations above the SCAQMD's LOD of 2 ppbv at two South Coast Area Basin (SCAB) landfills: BKK Landfill and Operating Industries Incorporated (OII) Landfill. The SCAQMD used the following method for determining the vinyl chloride LOD: 1) a 10 ppbv standard was analyzed a minimum of 10

times, 2) response was calculated as peak height (in millimeters) times the attenuation of the signal, 3) the precision of the measurement at 10 ppbv was 0.5 millimeters, 3) the minimum observable peak was taken to be four times 0.5 millimeters or 2 millimeters which corresponds to the reported LOD of 2 ppbv (Barbosa, 1990). The analysis in this report estimates ambient concentrations and population exposure to vinyl chloride near BKK and OII because they are the only landfills in the state where vinyl chloride has been routinely monitored on a long-term basis.

The SCAQMD's monitoring program for vinyl chloride at BKK and OII consisted of six monitoring stations. Three stations were located on the southern borders of each landfill. Previous monitoring around both landfills indicated that the southern borders were generally where the highest concentrations were detected. All samples were collected in Tedlar bags over 24-hour periods and subsequently analyzed by gas chromatography employing a flame ionization detector. Details of the SCAQMD's sampling and analysis procedures are provided in Appendix I.

BKK Landfill is located near West Covina while OII Landfill is located near Monterey Park. Figure III-1 shows the locations of the two landfills in relation to major freeways. In addition, topographical maps of the BKK and OII landfills are provided respectively in Figures III-2 and III-3. These figures show the approximate perimeter of the landfills, the approximate locations of the monitoring sites, and the proximity of streets to the landfills. As indicated by the maps, the southern borders of BKK and OII are adjacent to a network of streets. However, the maps do not show that the area served by these streets consists of single-family residential housing.

FIGURE III-1

THE LOCATION OF BKK AND OII LANDFILLS IN RELATION TO MAJOR FREEWAYS

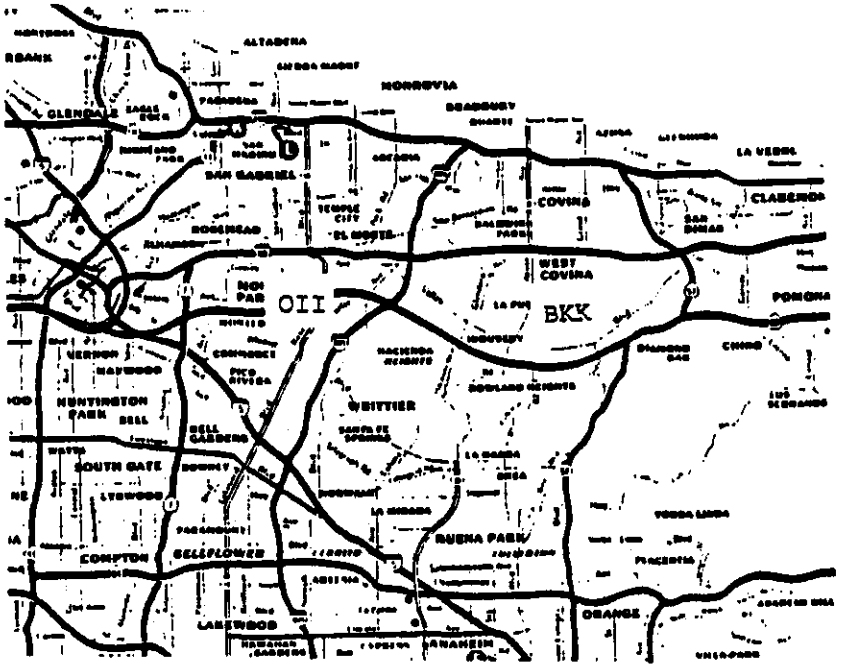
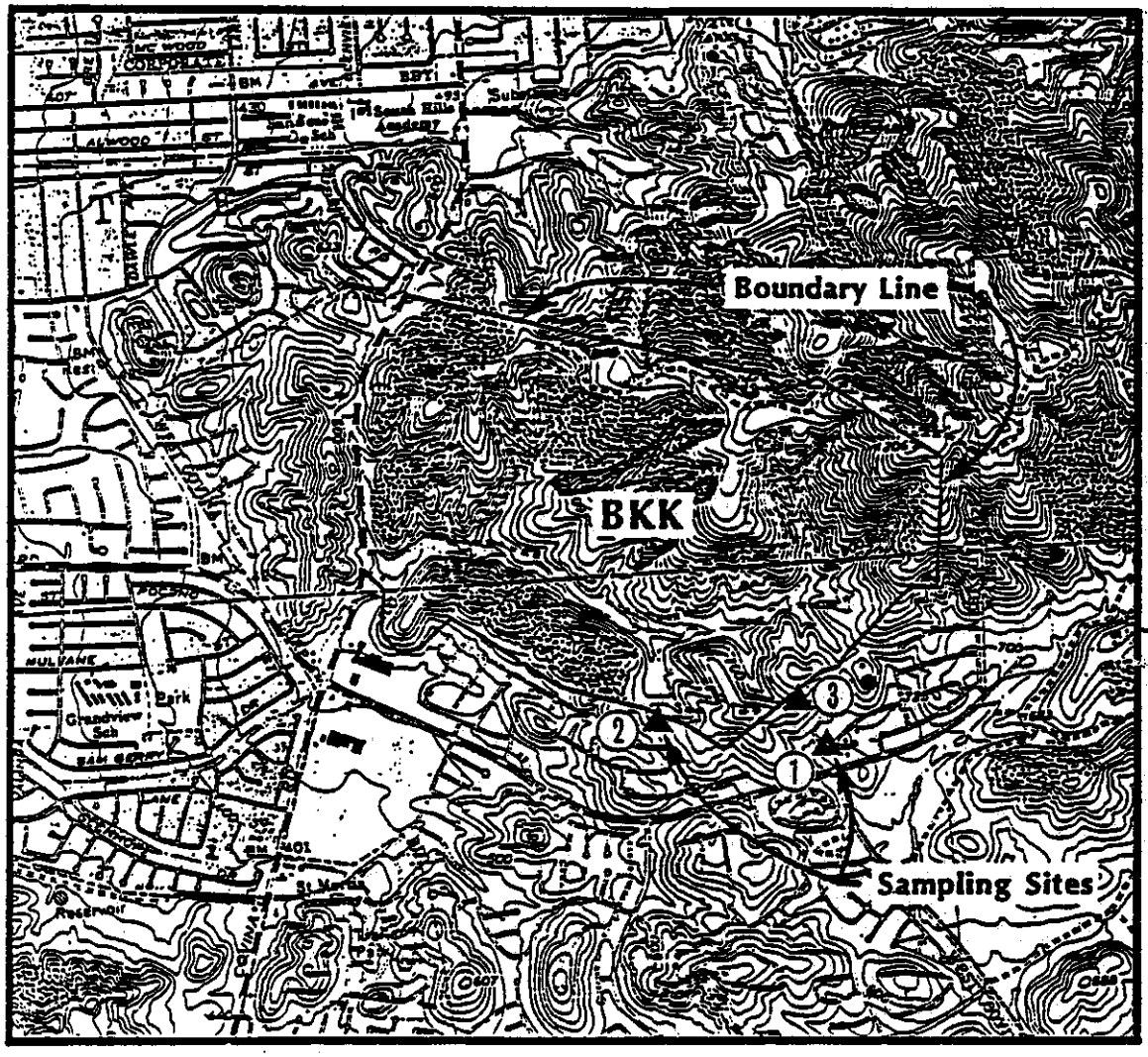
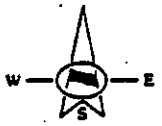


FIGURE III-2

BKK LANDFILL AND THE SURROUNDING AREA





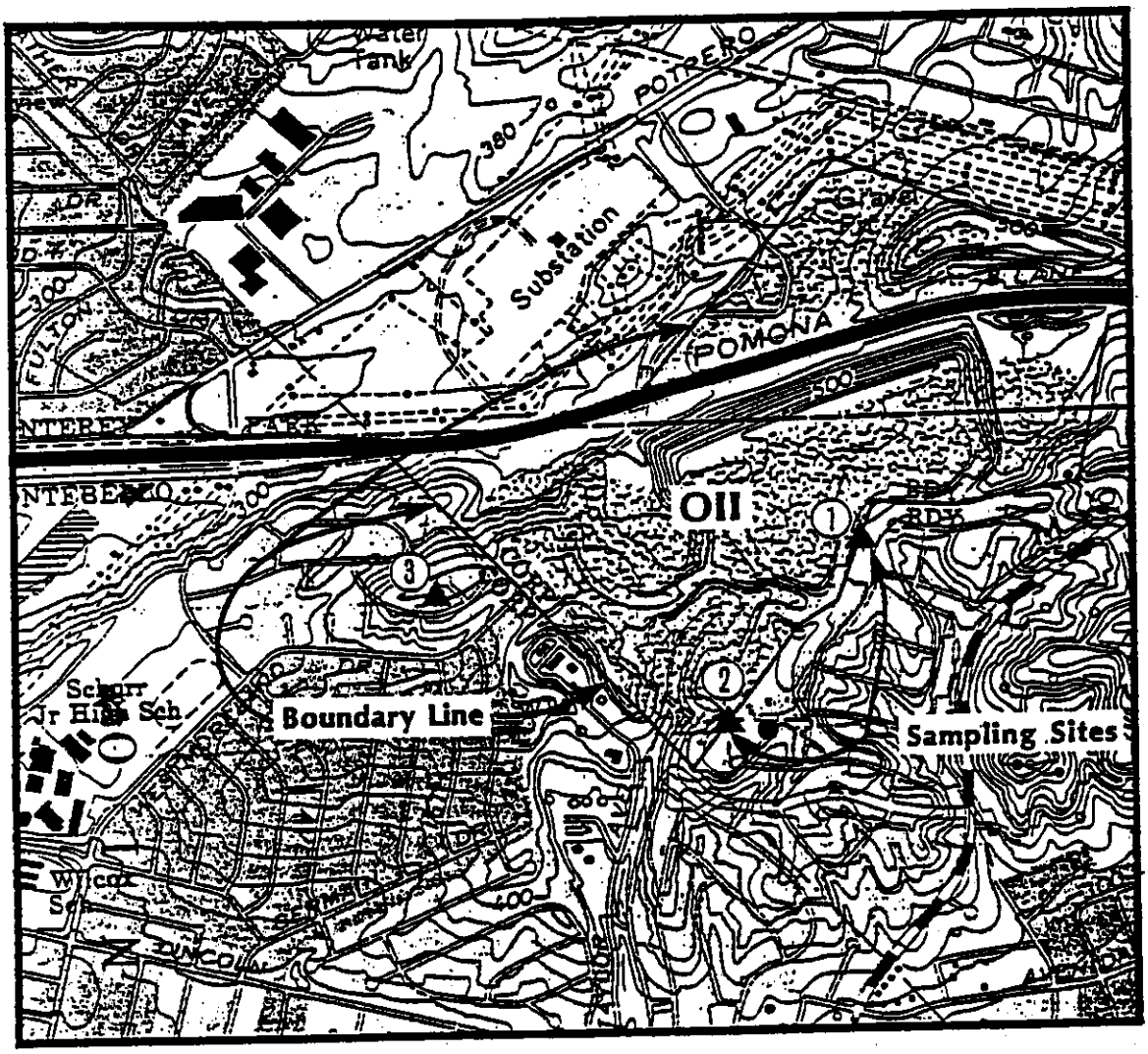
Boundary Line

① ▲ Sampling Sites

= 1 Kilometer

FIGURE III-3

OII LANDFILL AND THE SURROUNDING AREA



Boundary Line

① ▲ Sampling Sites

= 1 Kilometer

The 24-hour average ambient air samples used to estimate population exposure near BKK were collected from January through December of 1987. 24-hour ambient air samples used to estimate population exposure near OII were collected from January through December of 1986. When the exposure analysis was performed, these sampling periods represented the most recent calendar years of monitoring data that were available. For BKK, 337 to 345 samples were collected in 24-hour periods at each of the monitoring sites; of those sites, a range of 55 to 90 percent of the samples were below the LOD of 2 ppbv. For OII, 128 to 264 samples were taken at each of the monitoring sites; of those sites, 32 to 100 percent of the samples were below the LOD.

The ambient vinyl chloride monitoring data for BKK and OII are summarized in Tables III-1 and III-2, respectively. Each table provides the number of samples taken at each site, the percentage of samples below the LOD, an estimate for the values below the LOD, the estimated mean concentration, and the maximum 24-hour concentration that was measured.

Calculation of mean concentrations for stations was complicated by the presence of concentrations below the LOD. The concentrations below the LOD must be included in the calculation although their exact values were not known.

ARB staff has used a method proposed by Gleit (1985) to calculate the means. Gleit's method assumes that the sample of concentrations is a random sample from a normal distribution. Data that are judged not to be normally distributed may be transformed to approximate normality. Inspection of the vinyl chloride data suggested that they were lognormally distributed, and Gleit's method was applied to the logarithms of these data. The calculated means were then transformed back to the original units.

Gleit's method accounts for the concentrations below the LOD by setting them equal to the "below-LOD mean," the mean of the portion of the normal distribution below the LOD. Setting the unknown concentrations to their average value seems intuitively reasonable, and the simulations reported in Gleit's paper show that his method is more accurate than other commonly used approximations. A detailed description of the method used to estimate the concentration of data below the LOD is provided in Appendix II.

The estimated values for 24-hour averaged samples below the LOD ranged from 1.0 ppbv to 1.1 ppbv for BKK and from 1.0 ppbv to 1.2 ppbv for OII. As previously indicated, the specific value for each station is shown in Table III-1 for BKK and Table III-2 for OII. Because all samples for site 1 of OII were below the LOD, Gleit's method could not be used to estimate their concentration. A value of one-half the LOD (1.0 ppbv) was assumed for samples below the LOD based on the possibility that many contained between zero and 2 ppbv vinyl chloride.

TABLE III-1

SUMMARY STATISTICS FOR THE JANUARY 1987 THROUGH DECEMBER 1987 MONITORING DATA FOR VINYL CHLORIDE NEAR BKK LANDFILL
(Concentrations are reported in parts per billion by volume (ppbv) and are based on measurements averaged over a 24-hour sample collection period.)

	Station 1	Station 2	Station 3
Number of samples	337	337	345
Percent of Samples Below the LOD[a]	73	90	55
Estimated Concentration for[b] Samples Below the LOD	1.0	1.0	1.1
Estimated Mean[b] Concentration	1.7	1.2	2.6
Maximum 24-Hour Concentration[c]	7	8	15

a - The SCAQMD's limit of detection (LOD) for vinyl chloride is 2 ppbv.
b - Gleit's method was used to estimate the concentration of samples below the LOD.
c - California's Ambient Air Quality Standard for vinyl chloride is 10 ppbv for a 24-hour averaging period.

TABLE III-2

SUMMARY STATISTICS FOR THE JANUARY 1986 THROUGH DECEMBER 1986 MONITORING DATA FOR VINYL CHLORIDE NEAR OII LANDFILL
(Concentrations are reported in parts per billion by volume (ppbv) and are based on measurements averaged over a 24-hour sample collection period)

	Station 1	Station 2	Station 3
Number of Samples	264	220	128
Percent of Samples Below the LOD[a]	100	41	32
Estimated Concentration for[b] Samples Below the LOD	1.0	1.1	1.2
Estimated Mean[b] Concentration	1.0	2.0	2.0
Maximum 24-Hour Concentration[c]	d	8.3	9.8

a - The SCAQMD's limit of detection (LOD) for vinyl chloride is 2 ppbv.
b - Gleit's method was used to estimate the concentration of samples below the LOD, except for station 1.
c - California's Ambient Air Quality Standard for vinyl chloride is 10 ppbv for a 24-hour averaging period.
d - All samples are below 2 ppbv.

The estimated mean vinyl chloride concentrations using 24-hour averaged ambient measurements ranged from 1.2 ppbv to 2.6 ppbv for the monitoring stations at BKK and 1.0 ppbv to 2.0 ppbv for the monitoring stations at OII. The estimated mean vinyl chloride concentrations are shown in Tables III-1 and III-2. For all stations, except station 3 of BKK, the estimated annual mean concentration is equal to or less than the SCAQMD's LOD for vinyl chloride.

Tables III-1 and III-2 also list the maximum 24-hour average concentration of vinyl chloride for each monitoring station at BKK and OII, respectively. For BKK, the maximum 24-hour average concentration was 15 ppbv (measured at station 3); for OII the maximum 24-hour average concentration was 9.8 ppbv (measured at station 3). These concentrations can be compared to ARB's ambient air quality standard for vinyl chloride of 10 ppbv for a 24-hour averaging period. The standard was adopted in 1978 in response to information which associated vinyl chloride with the development

of cancer in humans. However, the standard is not necessarily health protective; it simply represented the LOD for vinyl chloride testing at the time it was adopted. According to the procedure specified in the standard, the LOD is based on 3 standard deviations of the method detection limit (within 10 standard deviations of the method detection limit). This method of calculating the LOD is the same as that used by the ARB Monitoring and Laboratory Division (see Appendix VII). For the monitoring periods presented in this report, BKK exceeded the state standard for vinyl chloride 11 times (all exceedances occurred at site 3) while OII did not exceed the standard. Based on previous years of monitoring data, the number of exceedances at BKK and OII has decreased substantially. In fact, due to the lack of exceedances of the standard, the SCAQMD discontinued routine monitoring for vinyl chloride at BKK in 1989 and at OII in early 1987. The reduction in ambient concentrations of vinyl chloride near BKK and OII landfills has been attributed to the installation of gas collection and flare systems.

In an effort to represent the uncertainties associated with the estimated mean concentrations of vinyl chloride, the staff developed a statistical treatment for calculating upper and lower bound estimates of the mean concentration at each monitoring station. This method takes into account factors such as sample size, variance of the data, and an estimate of the uncertainty associated with the sampling and analysis method. Table III-3 shows the estimated mean concentration as well as the upper and lower bound estimate of the mean concentration for each monitoring station at BKK and OII.

The following text discusses the statistical treatment that was used:

a) After reviewing the ambient vinyl chloride monitoring data for BKK and OII, the staff observed that the data appeared to be lognormally distributed. Because available software only analyze data that are normally distributed, vinyl chloride monitoring data were first converted from a lognormal distribution to a normal distribution. This was done by using the logarithms of the data for the analysis. The statistical analysis system (SAS, 1982) was used to calculate the standard error about the mean. The standard error calculated from the logarithms of the data is then converted back into concentration units by taking the antilogarithms.

b) The upper and lower bound estimates reported for the mean represented two standard errors. For the error associated with sampling and analysis, ARB staff used an overall uncertainty factor of $\pm$ 20 percent to calculate the upper and lower bound estimates of the mean. This was in agreement with the actual error which was estimated to be $\pm$ 1 ppbv in the range of 1 ppbv to 50 ppbv. The lower bound estimate represented two standard errors for the data with each sample concentration reduced by 20 percent. The upper bound estimate represented two standard errors for the data with each sample concentration increased by 20 percent. Upper and lower bound estimates for each station are shown in Table III-3. Because

all values for station 1 of OII are below the LOD, the upper and lower bound estimates represented ± 20 percent of one-half the LOD.

B. ESTIMATING AMBIENT CONCENTRATIONS

Annual average vinyl chloride concentrations were estimated for a 41 x 41 grid of one square kilometer cells surrounding each landfill with the use of the Industrial Source Complex Short Term (ISCST) Gaussian model. In order to predict the annual average concentration of vinyl chloride in each of the 1681 square kilometer cells, the ISCST model required the emission rates for each landfill as input. Emission rates were estimated for BKK and OII using the range of estimated annual mean concentrations at each of the monitoring stations.

The estimated emission rates were derived by ratioing estimated annual mean concentrations over modeled concentrations for each station.

TABLE III-3

UPPER AND LOWER BOUND ESTIMATES OF THE ANNUAL MEAN CONCENTRATIONS OF VINYL CHLORIDE AT BKK AND OII LANDFILLS

	Lower-bound Estimate	Annual Mean Concentration	Upper-bound Estimate
BKK Landfill			
Station 1	1.2	1.7	2.1
Station 2	0.9	1.2	1.4
Station 3	1.9	2.6	3.4
OII Landfill			
Station 1*	0.8	1.0	1.2
Station 2	1.4	2.0	2.8
Station 3	1.4	2.0	2.6

* - All samples were below the LOD of 2 ppbv.

The modeled concentrations were determined by assuming a landfill emission rate of 1 gram per square meter per second (gram meter^{-2} second^{-1}) in conjunction with historical meteorological data. Each landfill was represented as an area source. Based on review of topographical maps as well as information concerning the landfills disposal history, BKK was assumed to emit vinyl chloride from an area of approximately 1,700,000 meters^2 while OII was assumed to emit vinyl chloride from an area of approximately 330,000 meters^2. These assumed areas approximated the

area where wastes had been disposed. Meteorological data for 1981 at the SCAQMD's Walnut and Upland stations were used for BKK and OII, respectively. Meteorological data from these stations were used for this study because Walnut was considered the most representative station for BKK where processed data were avai$ble while Upland was considered the most representative station for OII where processed data were available. These data were entered into the ISCST model to calculate the annual average modeled concentration at each monitoring station. Because one year of meteorological data was used, one modeled concentration was obtained for each monitoring station at BKK and OII. For each site at BKK and OII the modeled concentration was divided into the estimated mean concentration (from Table III-3) of its respective monitoring station. The resulting factors or ratios were then multiplied by the assumed emission rate (1 gram $meter^{-2}$ $second^{-1}$) to estimate a landfill emission rate for each monitoring station that will result in an exact match between estimated and modeled concentrations. Equation (1) illustrates the procedure that was used:

$$\text{Estimated Emission Rate} = \text{Assumed Emission Rate} \times \left(\frac{\text{Estimated Concentration}}{\text{Modeled Concentration}}\right) \quad (1)$$

The estimated landfill emission rates for each monitoring station at BKK and OII are given in Table III-4. The emission rate derived from the estimated mean concentration for each monitoring station and the emission rates derived from the upper and lower bound estimates of the mean concentration for each monitoring station are listed. The greatest range of estimated emission rates for BKK was from 0.75 micrograms $meter^{-2}$ $second^{-1}$ (lower-bound at station 2) to 3.32 micrograms $meter^{-2}$ $second^{-1}$ (upper-bound at station 3). For OII, the estimated emission rates ranged from 0.31 micrograms $meter^{-2}$ $second^{-1}$ (lower-bound at station 1) to 4.42 micrograms $meter^{-2}$ $second^{-1}$ (upper-bound at station 3).

Using the full range of emission rate estimates (0.75 to 3.32 micrograms $meter^{-2}$ $second^{-1}$ for BKK and 0.31 to 4.42 micrograms $meter^{-2}$ $second^{-1}$ for OII), a range of estimated annual average vinyl chloride concentrations was derived for the 41 by 41 grid of one square kilometer cells. Each landfill was located in the center of the grid and was represented as an area source. As previously stated, BKK was assumed to emit vinyl chloride from an area of approximately 1,700,000 $meters^2$ while OII was assumed to emit vinyl chloride from an area of approximately 330,000 $meters^2$. These areas approximate the area where wastes were disposed at each landfill. However, because subsurface migration of landfill gases has been observed at BKK and OII, it is possible that emissions of vinyl chloride occur over an area substantially greater than where wastes were actually disposed. The ISCST model used the range of estimated emission rates assuming no plume rise in conjunction with historical meteorological data to predict a range of annual average concentrations for each of the 1681 one-square-kilometer-cells. The annual average concentrations of

TABLE III-4

ESTIMATED EMISSION RATES OF VINYL CHLORIDE FROM BKK AND OII LANDFILLS
(micrograms meters^{-2} second^{-1})

	Lower-bound[a]	Average[b]	Upper-bound[c]
BKK Landfill			
Station 1	1.36	1.80	2.30
Station 2	0.75	0.97	1.20
Station 3	1.88	2.55	3.32
OII Landfill			
Station 1	0.31	0.38	0.46
Station 2	0.52	0.74	1.04
Station 3	2.69	3.46	4.42

a - These emission rates were derived from the lower-bound annual mean concentration.
b - These emission rates were derived from the annual mean concentration.
c - These emission rates were derived from the upper-bound annual mean concentration.

vinyl chloride predicted for the one-square-kilometer cells within the grid centered on BKK, ranged from less than 0.1 ppbv to approximately 22 ppbv. For OII, the range was from less than 0.1 ppbv to approximately 3.8 ppbv.

In order to obtain these modeling results, several assumptions were made. These assumptions may act to elevate or reduce the estimated annual average concentrations of vinyl chloride predicted for the cells surrounding BKK and OII. The primary assumptions were as follows:

1) Vinyl chloride was assumed to be emitted from an area of approximately 1,700,000 meters^2 for BKK and 330,000 meters^2 for OII. Although these areas approximate the area where wastes were disposed, data were not available to demonstrate that these

areas actually represented where vinyl chloride emissions occurred. Emissions of vinyl chloride might have occurred over an area which is either larger or smaller than that assumed.

2) Emissions of vinyl chloride were assumed to occur continuously and uniformly over a given area of each landfill. In reality, vinyl chloride was not likely to emanate uniformly over the surface of the landfills. However, the data required by the model to take this into consideration were not available. If emissions of vinyl chloride vary over the surface of the landfills, the annual concentrations estimated for some cells would be expected to be underestimated while others would be overestimated.

3) This study did not use meteorology for the same year as the vinyl chloride measurements. Because there was not a great deal of variation in meteorological data from year to year, the degree of error from using a meteorological year different than the vinyl chloride measurement year was estimated to be less than ± 50 percent.

Because the emission rates were derived by model calibration to known vinyl chloride concentrations, the uncertainty was at a minimum near the monitoring sites. Alternatively, as the distance from each monitoring station increased, the uncertainty associated with the estimated concentration increased.

C. POPULATION EXPOSURE

The population exposure to vinyl chloride near BKK and OII was estimated by using the grid cell concentrations estimated from the ISCST model in conjunction with 1985 updated census data. Estimates of the cumulative population exposed to various concentration levels of vinyl chloride near BKK and OII landfills are shown in Tables III-5 and III-6. The 1985 residential population estimates were determined for each one kilometer grid cell with the concentration determined at the center of each cell by the ISCST model. The 1681 grid cells, with their associated populations, were sorted from high to low by concentration. The grid cell populations were then summed to determine the cumulative population exposed at or above certain levels of vinyl chloride. For Tables III-5 and III-6, a lower bound of exposure was estimated. This range of exposure is based on upper and lower bound estimates of the vinyl chloride emission rate from each of the two landfills. Table III-5 shows that approximately 730,000 to 2,000,000 people were exposed to an annual average concentration of at least 0.05 ppbv of vinyl chloride from the BKK landfill. Approximately 17,000 to 130,000 of these people were exposed to an annual average concentration of at least 1.0 ppbv from this facility. Table III-6 shows that approximately 33,000 to 1,100,000 people were exposed to an annual average concentration of at least 0.05 ppbv of vinyl chloride from the OII landfill. Approximately 0 to 22,000 of these people were exposed to an annual average concentration of at least 1.0 ppbv vinyl chloride from OII.

TABLE III-5

RANGE OF CUMULATIVE POPULATION EXPOSED TO VINYL CHLORIDE NEAR BKK

Range of Cumulative Population			Exposed to Vinyl Chloride Concentrations (ppbv) at or above:
Lower-bound Estimate[a]		Upper-bound Estimate[b]	
2,154,000	-	2,154,000	>0 but <0.01[c]
2,026,000	-	2,154,000	0.01
732,000	-	1,970,000	0.05
374,000	-	1,431,000	0.1
17,000	-	131,000	1.0
0	-	54,000	2.0
0	-	28,000	3.0
0	-	20,000	4.0
0	-	14,000	5.0
0	-	7,000	6.0
0	-	2,500	7.0

a - The exposure estimate is based on an emission rate of 0.75 ug/m^2s^{-1}.

b - The exposure estimate is based on an emission rate of 3.32 ug/m^2s^{-1}.

c - According to the model, the entire population was at least exposed to vinyl chloride concentrations between 0 and 0.01 ppbv. In addition, the calculated population-weighted exposure for this population was estimated to range from an annual average of 0.08 to 0.34 ppbv vinyl chloride.

In addition to estimating the cumulative population exposure to vinyl chloride for people living near BKK and OII, the population-weighted exposure results were calculated. The population-weighted exposure was calculated by multiplying the estimated annual average concentration for each cell by the population represented by the cell. The exposure results for the 1681 cells were subsequently summed and divided by the total population represented by the 1681 cells. For BKK, the population-weighted

TABLE III-6

RANGE OF CUMULATIVE POPULATION EXPOSED TO VINYL CHLORIDE NEAR OII

Range of Cumulative Population Exposed to Vinyl Chloride			
Lower-bound Estimate[a]		Upper-bound Estimate[b]	Concentrations (ppbv) at or above:
4,287,300	-	4,287,300	>0 but <0.01[c]
272,000	-	3,111,000	0.01
33,000	-	1,073,000	0.05
12,000	-	445,000	0.10
0	-	22,000	1.0
0	-	12,000	1.5
0	-	6,000	2.0
0	-	6,000	3.0

a - The exposure estimate is based on an emission rate of 0.31 ug/m^2s^{-1}.

b - The exposure estimate is based on an emission rate of 4.42 ug/m^2s^{-1}.

c - According to the model, the entire cumulative population studied was at least exposed to vinyl chloride concentrations between 0 and less than 0.01 ppbv. In addition, calculated population-weighted exposure for this population was estimated to range from an annual average of 0.004 to 0.06 ppbv vinyl chloride.

exposure estimates showed that approximately 2,000,000 people were exposed to an annual average vinyl chloride concentration ranging from 0.08 ppbv to 0.34 ppbv. For OII the population-weighted exposure estimates showed that approximately 4,000,000 people were exposed to an annual average vinyl chloride concentration ranging from 0.004 ppbv to 0.06 ppbv.

The model was also used to estimate the annual average concentrations for the maximum exposed individual at each landfill. For BKK, the maximum exposed individual was estimated to be exposed to an annual average concentration ranging from 2.3 ppbv to 10.3 ppbv. For OII, the maximum exposed individual was estimated to be exposed to an annual average concentration ranging from 0.6 to 8.7 ppbv.

The population exposure estimates for BKK and OII suggest that other landfills in California that emit vinyl chloride may expose the nearby population to elevated concentrations. Chapter II of this report discusses other vinyl chloride monitoring data that are available as well as preliminary data on the Landfill Gas Testing Program (see Appendix VI).

D. INDOOR EXPOSURE TO VINYL CHLORIDE

With the exception of some homes located near landfills, indoor concentrations of vinyl chloride are not expected to be substantially greater than outdoor concentrations. Although data are limited, the above statement is supported by the following facts: 1) few indoor sources of vinyl chloride have been identified; and 2) most studies that have monitored for indoor concentrations of vinyl chloride fail to detect it. However, landfills have been identified as a source of emissions that contributes to elevated indoor levels of vinyl chloride in nearby residences. Grab samples from some houses located near landfills have shown vinyl chloride at concentrations up to 100 ppbv.

We estimate that people living near landfills may be inhaling up to 2600 micrograms of vinyl chloride a day (see Appendix III for assumptions). For these individuals, inhalation of vinyl chloride indoors is expected to represent the most significant source of exposure. A more detailed discussion of indoor exposure to air contaminants is presented in Appendix III.

1. Potential Sources of Indoor Vinyl Chloride

There are several potential sources that can contribute to indoor concentrations of vinyl chloride. These sources include landfills, polyvinyl chloride (PVC) products containing residues of vinyl chloride, water that contains residues of vinyl chloride and cigarette smoke. For most homes, these sources are not expected to result in substantially elevated indoor levels of vinyl chloride. However, for some homes located near landfills, staff believe that landfills may represent the most significant contribution to indoor levels of vinyl chloride.

Vinyl Chloride From Landfill Gas. There are at least two ways that vinyl chloride emissions from landfills may contribute to indoor concentrations of vinyl chloride in nearby residences: 1) homes that are located downwind from landfills can receive vinyl chloride through direct outdoor air influx into indoor environments; and 2) landfill gases containing vinyl chloride can migrate underground and enter homes through substructures. The rate of accumulation of vinyl chloride indoors depends on several factors including soil permeability, source strength, air exchange rate and structure of the home. In addition, higher indoor concentrations may occur because vinyl chloride is more rapidly destroyed in outdoor air than indoor air. Outdoor destruction proceeds more quickly because vinyl chloride's reaction with hydroxyl radicals is the compound's dominant atmospheric removal mechanism and because hydroxyl radicals are formed in the presence of direct sunlight.

Plastic Materials and Consumer Products. Plastic products made of PVC and other vinyl chloride polymers are ubiquitous in most homes. Because vinyl chloride monomer can remain in the PVC resin for an extended period of time, an indirect source of indoor vinyl chloride emissions may come from the release of unreacted vinyl chloride monomer from these plastic products.

Emissions of unreacted vinyl chloride monomer have been substantially reduced due to improvements in monomer stripping technology (Wheeler, 1981). In the past, residual vinyl chloride concentrations in PVC resins at the time of shipment, were as high as 2000 ppm. Currently, PVC resins contain about 10 ppm residual vinyl chloride at the time of shipment and may lose vinyl chloride at a rate of 20 to 50 percent per month during storage. In addition, most of the vinyl chloride will vaporize and escape during the high temperature processes in which PVC resins are melted and made into final products. Thus, consumer products made of PVC resins no longer contain elevated residual levels of vinyl chloride monomer and, therefore, are not expected to be an important contributor of indoor levels of vinyl chloride.

Vaporization from Water Sources. Because activities such as using water for cooking, heating and showering can promote rapid vaporization of vinyl chloride from water, contaminated surface or ground water may increase indoor vinyl chloride levels.

In California, surface water is generally free of vinyl chloride (Sharrp, 1987). In assessing ground water quality, the California Department of Health Services reported, based on a limit of detection of 0.5 micrograms/liter, that one out of the 2,947 wells for large public water systems that were sampled had detectable levels of vinyl chloride (DHS, 1986). The maximum concentration found in that well was 23 micrograms/liter with a median value of 20 micrograms/liter. Vinyl chloride has not been detected in wells used for small public water systems (DHS, 1987). Therefore, vinyl chloride in the water supply is not believed to significantly impact indoor air concentrations of vinyl chloride.

Cigarette Smoke. Vinyl chloride has been identified in the smoke of cigarettes (1.3 to 16 nanograms/cigarette) and of little cigars (14 to 27 nanograms/cigar). (IARC, 1985; Hoffmann, Patrianakos and Brunnemann, 1976). The vinyl chloride level in the mainstream smoke may be estimated by the total inorganic chloride content of the tobacco. However, the contribution from tobacco smoke does not appear to have a significant impact on the indoor concentration of vinyl chloride.

2. Indoor Monitoring Data

Indoor air data can be obtained either by personal air sampling or by fixed-site air sampling. In personal sampling, the sampling equipment is carried by an individual and air samples are taken wherever the individual

may be. In contrast, fixed-site air sampling refers to air samples taken at fixed locations. Personal air sampling data generally provide a more realistic estimate of individual exposure. Because most people spend 80 to 90 percent of their time in indoor environments, personal air sampling data are strongly weighted by indoor air exposure data.

Personal Sampling Data. Based on limited personal sampling data, it appears that indoor air exposure to vinyl chloride is low. Nine subjects in New Jersey and three subjects in North Carolina were monitored for 5 to 10 hours on three separate occasions for several days over a 6-month period. All of the 138 air samples taken were below the LOD which was reported to be 20 ppbv (Wallace et al., 1984). The procedure for calculating the LOD was not described in the paper and the subcontractor who conducted the monitoring could not be located for his definition of LOD.

Fixed-site Sampling Data. For a study conducted in California, fixed-site monitoring stations were installed to monitor indoor and outdoor air concentrations of vinyl chloride. Based on the analysis of 32 indoor samples taken in eight homes during the summer season for two twelve-hour sampling periods (daytime and nighttime), concentrations of vinyl chloride were all below the LOD. The samples were analyzed by two analytical methods with LODs ranging from about 0.2 ppbv to 58 ppbv (0.55 and 148 micrograms $meter^{-3}$)(Pellizzari et al., 1989). The LOD as defined by Pellizari et al. is a value where the measured signal of the analyte is three times that of the noise of the instrument. Therefore, values below the LOD are not reported.

A similar study was conducted in Baltimore where indoor air concentrations of vinyl chloride in about 160 homes were monitored by fixed-site sampling stations. Based on partially analyzed results, vinyl chloride was not detected in indoor air environments. The LOD was reported to range from 10.2 ppbv to 15.7 ppbv (26 to 40 micrograms $meter^{-3}$) (Pellizzari, 1987).

Special Situation Air Monitoring. In 1981, the SCAQMD collected 24-hour bag samples in the vicinity of BKK landfill. Over 500 air samples were taken at two outdoor sites and at four indoor sites downwind of the landfill (SCAQMD, 1982). All of the samples (approximately 120 samples) that equaled or exceeded the state vinyl chloride standard of 10 ppbv (26 micrograms $meter^{-3}$) were taken inside the residences. The highest recorded indoor vinyl chloride concentration was 50 ppbv (130 micrograms/$meter^{-3}$). The LOD was reported to be 2 ppbv (5.2 micrograms $meter^{-3}$) (see section A, Chpater III for the SCAQMD method for calculating the LOD).

In 1984, the South Coast Air Quality Management District (SCAQMD) sampled water meter boxes at the property lines of a few homes adjacent to OII Landfill as a screen for landfill gas migration. Water meter boxes are below-ground enclosed boxes containing an apparatus which measures the amount of water used by a household. The 10 grab samples taken from inside water meter boxes of homes adjacent to OII Landfill showed vinyl chloride levels ranging from 13 to 36000 ppbv. This finding prompted a 1985 South

Coast Air Quality Management District indoor air grab-sample study which showed vinyl chloride concentrations ranging from 8 to 100 ppbv (20.8 - 260 micrograms meter^{-3}) in some homes near the landfill (SCAQMD, 1986). Presently, indoor concentrations of vinyl chloride in these residences are believed to be substantially lower because of the installation of gas collectors and flares (Coy, 1987). OII is now a federal superfund site managed by the EPA.

E. EXPOSURE THROUGH OTHER ROUTES

While the main objective of this report is to estimate exposure through the air, exposure to vinyl chloride may also occur from the ingestion of food and water that contain residues of vinyl chloride. The Health and Safety Code specifies that the ARB shall identify the relative contribution to total exposure to the contaminant from indoor concentrations, taking into account both ambient and indoor environments (California Health and Safety Code, 1989). The inclusion of these data provide a useful perspective of the overall exposure to vinyl chloride through environmental media. The estimated daily dose of vinyl chloride from different environmental media are presented in Table III-7. From the table, exposure to vinyl chloride from the indoor air of homes not located near landfills, food, and water appears to be minor. However, for people living in houses located near landfills, indoor exposure to vinyl chloride may represent the major source of total vinyl chloride exposure. The need for total exposure assessment and some of the issues and concepts involved in total exposure estimates are discussed in Appendix III.

1. Water Ingestion

The major source of drinking water for California is surface water which is not expected to have detectable levels of the highly volatile vinyl chloride. Ground water used for public water systems is also relatively free of vinyl chloride with concentrations typically below 0.5 ug liter^{-1} (DHS, 1987, 1986). Based on this information, staff believe that exposure to vinyl chloride through drinking water is not important under ordinary situations.

2. Food Ingestion

Vinyl chloride is not routinely monitored for in U.S. food products. However, before 1973 vinyl chloride was found in food and beverages packaged in vinyl chloride polymer materials (IARC, 1979). At that time, levels as high as 20 mg^{-1} kg^{-1} (ppm) of vinyl chloride monomer were present in alcoholic beverages packaged in this material. Vinyl chloride was also found in edible oils, butter, and margarine at concentrations ranging from 0.05 - 14.8 mg kg^{-1}. In 1986, the Food and Drug Administration (FDA) proposed to limit the maximum amount of residual vinyl chloride monomer in rigid and semi-rigid food containers to 10 ppbw and the maximum amount of vinyl chloride monomer allowed in polymeric coatings and films which contact food to 5 ppbw. The regulation was not promulgated because the FDA believed that monomer stripping processes leave no residue of vinyl chloride monomer. In 1986, the Food and Drug Administration (FDA) estimated that the lifetime-averaged individual exposure to vinyl chloride

from food and beverages packaged with vinyl chloride polymer materials would not exceed 25 nanograms per day (FDA, 1986). An estimate of today's potential for vinyl chloride exposure from food ingestion is not possible because, to the ARB's knowledge, current information on the levels in food and food packaging are not available.

TABLE III-7

ESTIMATED VINYL CHLORIDE EXPOSURE THROUGH DIFFERENT MEDIA[a]

Media	Daily Dose	Reference
AIR		
Ambient Air		
not near landfills	b	
near landfills	<104 to 780 ug	Table III-1, 1990
Indoor Air		
homes not near landfills	<11 ug	Pellizari et al., 1989
homes near landfills	up to 2600 ug	SCAQMD, 1982
INGESTION		
Drinking water: Surface/Ground Water	<1 ug	DHS, 1986; 1987
Food including beverages	< 0.025 ug	FDA, 1986

a - The assumptions that were used for Table II-7 are provided in Appendix III.

b - The dose from exposure to ambient air not near landfills was not calculated because the ARB's ambient monitoring network has not detected vinyl chloride.

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IV.

PERSISTENCE IN THE ATMOSPHERE

A. PHYSICAL PROPERTIES

The chemical structure of vinyl chloride (chloroethene, chloroethylene) is CH_2= CHCl. Vinyl chloride is a sweet smelling, colorless gas at ambient temperature and pressure. It polymerizes in light or in the presence of a catalyst. Vinyl chloride is readily flammable and forms explosive mixtures in air. Upon combustion, it is degraded mainly to hydrogen chloride gas (HCl), carbon monoxide (CO), carbon dioxide (CO_2) and traces of phosgene (Cl_2C=O). Vinyl chloride is expected to volatilize rapidly from water (H_2O) systems. Experimental data indicate that for an initial concentration of 1 ppm at a solution depth of 6.5 cm and a stirring rate of 200 rpm, the average evaporative half-life of vinyl chloride at a temperature of approximately 25°C is 27.6 minutes (Dilling, 1977). Another study determined that distilled water spiked with 16 ppm vinyl chloride lost 96 percent of the vinyl chloride within two hours (U.S. EPA, 1974). Although it is soluble in ethanol (CH_3CH_2OH), industrial solvents, and a number of organic liquids, vinyl chloride is only slightly soluble in water. Vinyl chloride's physical properties are shown in Table IV-1.

B. ATMOSPHERIC PERSISTENCE

Reaction with hydroxyl radicals (OH) is the dominant mechanism removing vinyl chloride from the troposphere (Cupitt, 1980; Atkinson, 1986a). Estimates of vinyl chloride's tropospheric lifetime range from 0.5 to 5.8 days. However, for reasons provided later in this section, ARB staff believe that a tropospheric lifetime ranging from 1.6 to 3.9 days is representative of typical atmospheric conditions. The rate at which this reaction proceeds depends on the temperature and the tropospheric concentration of both vinyl chloride and hydroxyl radicals. The temperature dependence of the reaction rate is incorporated in the rate constant for the reaction of vinyl chloride with hydroxyl radicals. The product of the rate constant and both species concentrations gives the rate at which vinyl chloride is being degraded (Finlayson-Pitts and Pitts, 1986).

TABLE IV-1

PHYSICAL PROPERTIES OF VINYL CHLORIDE

Properties	Value	Reference
Boiling point, 1 Atm	-13.37 ^{o}C	Merck Index, 1983
Molecular weight	62.5	Merck Index, 1983
Vapor Pressure, 20 ^{o}C	2530 mm Hg	Merck Index, 1983
Sol. in water, 25^{o}C	0.11g/100g H_2O	Kirk-Othmer, 1980
Partition Coeff. H_2O/Air 10^{o}C	0.02	McConnell, G., et al., 1975
Octanol/H_2O Partition Coeff.	20.7	Withey, 1976
Specific gravity, 20/4 ^{o}C	0.912	Kirk-Othmer, 1980
Flash pt. open cup	-77.8^{o}C	Kirk-Othmer, 1980
Liq. Dens. -14.2 Cg/cm^3	0.969	CRC Handbook, 1985
Heat capacity, 27^{o}C	16.1	CRC Handbook, 1985

The tropospheric lifetime of a compound is an estimate of the time required for a given amount of the compound to decrease to 1/e (0.368) of its original concentration (at time zero). The tropospheric lifetime (τ) of vinyl chloride is related to the rate constant (k) and the hydroxyl radical concentration ([OH]) by the equation (1):

$$\tau = (k[OH])^{-1} \qquad (1)$$

In deriving the above equation, it is assumed that hydroxyl radicals are at a constant steady state concentration in the troposphere.

Estimates have been made for the rate constant resulting from vinyl chloride's reaction with hydroxyl radicals. Perry et al. (1977) estimated the absolute rate constants over the temperature range of 299 Kelvin (^{o}K) to 426^{o}K. The limiting high pressure rate constant for a temperature of 299^{o}K is estimated to be 6.60 $\pm$ 0.66 x 10^{-12} cm^3 molecule^{-1} second^{-1}. Howard determined rate constants for the reaction of vinyl chloride with hydroxyl radicals at 296^{o}K over a range of pressure where the highest pressure employed had not reached the limiting high pressure regime (Howard et al., 1976). However, when data obtained by Howard are extrapolated to the high pressure limit, the resulting rate constant of

approximately 7 x 10^{-12} cm^3 $molecules^{-1}$ $second^{-1}$ is in good agreement with the value reported by Perry (Perry et al., 1976). Using a different technique, a study by Liu, et al. over the temperature range of 313 to 423°K was also in good agreement with Perry, et al. (Perry, et al.,1976; Liu, et al., 1989). Table IV-2 summarizes the rate constant estimates, atmospheric lifetime estimates, average temperature assumed, and the method used to estimate the rate constant for the reaction of vinyl chloride with hydroxyl radicals and ozone (O_3).

The 24-hour average hydroxyl radical concentration in the troposphere has been estimated to range from 3 x 10^5 to 3 x 10^6 molecules cm^{-3} (Hewitt & Harrison, 1985). Because hydroxyl radicals are only present during daylight, the actual range for daytime concentrations is twice the 24-hour averages given above while nighttime concentrations are essentially zero. Prinn, et al. derived the most reliable average hydroxyl radical concentration of 7.7 X 10^5 molecules cm^{-3} using the ambient tropospheric concentration and emission inventory of methyl chloroform (CH_3CCl_3) (Prinn, et al., 1987). Daytime hydroxyl radical concentrations vary depending on many factors including photolytic activity and the concentration of ozone as well as other pollutants in the troposphere.

Using the rate constant determined by Perry et al. for an average tropospheric temperature of 299°K and a range of hydroxyl radical concentrations ranging from 3 x 10^5 to 3 x 10^6 molecules cm^{-3}, the estimated tropospheric lifetime for vinyl chloride ranges from:

0.6 days for [OH] = 3 x 10^6 molecules cm^{-3}

to

5.8 days for [OH] = 3 x 10^5 molecules cm^{-3}

As previously indicated, the concentration of hydroxyl radicals in the troposphere can vary considerably. However, several researchers recommend 24-hour average hydroxyl radical concentrations which are between 0.5 x 10^6 and 1 x 10^6 molecules cm^{-3} (Prinn et al., 1987; Winer, 1978; Singh et al., 1983; Cupitt, 1980; Cox et al., 1976; Davis et al., 1976). By using this range of 24-hour average hydroxyl radical concentrations (0.5 x 10^6 to 1 x 10^6 molecules cm^{-3}) in conjunction with the rate constants determined from Perry's rate constant at 299°K, the resulting range in atmospheric lifetimes is from 1.6 days to 3.9 days. Using Howard's adjusted rate constant derived by extrapolating to the high pressure limit, and the same range of hydroxyl radical concentrations (0.5 x 10^6 to to 1 x 10^6 molecules cm^{-3}), the atmospheric lifetime estimates for vinyl chloride ranges from 1.6 to 3.3 days.

TABLE IV-2

ATMOSPHERIC LIFETIME AND REACTION
RATE CONSTANT ESTIMATES FOR VINYL CHLORIDE

Reactant	Rate[a] Constant	Temperature (Kelvin)	Method	Atmospheric[b] Lifetime (days)	References
OH	$6.6 \pm 0.66 \times 10^{-12}$	299	FP-RF[c]	1.6 - 3.9[d]	Perry et al., 1977
O_3	$2.45 \pm 0.45 \times 10^{-19}$	298	S-FTIR[e]	47[f]	Zhang et al., 1983
O_3	2.3×10^{-19}	NR	S-FTIR[e]	50	Gay et al., 1976
O_3	6.5×10^{-21}	295	S-UV[g]	4.9 years	Sanhueza et al. 1976

a - Rate constant units are cm^3 $molecule^{-1}$ $second^{-1}$.

b - The atmospheric lifetime is defined as the time required for a given amount of the compound to decrease to 1/e (0.368) of its original concentration (at time zero).

c - FP-RF = Flash photolysis, resonance fluorescence.

d - Assumes a 24-hour average hydroxyl radical concentration ranging from 0.5×10^5 to 1×10^6 molecules cm^{-3} (Cupitt, 1980).

e - S-FTIR = Static system, Fourier transform infrared absorption spectroscopy.

f - Assumes a 24-hour average O_3 concentration of 1×10^{12} molecules cm^{-3} (Singh et al., 1978).

g - S-UV = Static system, ultraviolet absorption.

NR- Not Reported

The initial step in the reaction of vinyl chloride with hydroxyl radicals proceeds by the addition of hydroxyl radical to the carbon-carbon double bond. Although subsequent steps in the reaction mechanism are unknown, reaction products have been identified (Atkinson, 1986b). The major product resulting from hydroxyl radical attack on vinyl chloride is formyl chloride (HCOCl). Within the experimental error of two independent studies, the reaction of one molecule of vinyl chloride with hydroxyl radicals was demonstrated to yield one molecule of formyl chloride (Pitts et al., 1984, Tuazon et al., 1988). An ARB sponsored study demonstrated that the yield of formyl chloride from the reaction of hydroxyl radicals with vinyl chloride is unity (one molecule of formyl chloride for each molecule of vinyl chloride) within the experimental error of the study (Pitts et al., 1984). The

observed unit yield of formyl chloride implies a corresponding unit yield of formaldehyde (HCHO) and shows that the reaction of vinyl chloride with hydroxyl radicals proceeds by essentially 100 percent cleavage of the double bond. Equations (2) through (6) summarize the overall reaction scheme which seems most likely (Pitts et al., 1984).

$$OH + CH_2{=}CHCl \longrightarrow HOCH_2CHCl \quad (2)$$

$$HOCH_2\overset{\overset{O}{\overset{2}{|}}}{C}HCl \longrightarrow HOCH_2\overset{\overset{O\text{-}O^{\cdot}}{|}}{C}HCl \quad (3)$$

$$HOCH_2\overset{\overset{O\text{-}O^{\cdot}}{|}}{C}HCl + NO \longrightarrow HOCH_2\overset{\overset{O\text{-}O^{\cdot}}{|}}{C}HCl + NO_2 \quad 4)$$

$$HOCH_2\overset{\overset{O^{\cdot}}{|}}{C}HCl \longrightarrow CH_2OH + HCOCl \quad (5)$$

$$CH_2OH + O_2 \longrightarrow HCHO + HO_2 \quad (6)$$

This reaction was confirmed in a study by Tuazon, et al. (Tuazon, et al., 1988).

Under atmospheric conditions, the reaction of vinyl chloride with ozone is not expected to be important compared to its reaction with hydroxyl radicals (Atkinson, 1986a; Atkinson and Carter, 1984). Several rate constant estimates have been made for the reaction of vinyl chloride with ozone. Based on these rate constants, atmospheric lifetime estimates range from about 47 days to approximately 5 years (Zhang et al., 1983; Sanhueza et al., 1976). Table IV-2 summarizes the atmospheric lifetime and rate constant estimates along with other pertinent information for vinyl chloride's reaction with ozone. Due to the variability among the estimated rate constants, a review publication made no recommendations as to the rate constant for the reaction of vinyl chloride with ozone (Atkinson and Carter, 1984). Furthermore, because the reaction of ozone with vinyl chloride can be complicated by secondary reactions, the rate constants provided in Table IV-2 should be considered to be upper bound limits.

Products resulting from the reaction of ozone with vinyl chloride in the absence of scavengers are formyl chloride and formic acid (HCOOH) (Zhang et. al., 1983). Other products resulting from the reaction of ozone with vinyl chloride include carbon monoxide, carbon dioxide, formaldehyde, and hydrochloric acid (Gay et al., 1976; Zhang et al., 1983).

A relative rate technique was recently employed to obtain a rate constant for the gas-phase reaction of vinyl chloride and the nitrate (NO_3) radical (Atkinson, et al., 1987). The rate constant ratio of k(NO_3+ vinyl chloride)/ k(NO_3+ ethene) at 298 $\pm$ 2°K is 2.08 $\pm$ 0.09 with the room temperature rate constant for the reaction of the nitrate radical with ethene

(CH_2=CH_2) is 2.1 X 10^{-16} cm^3 $molecule^{-1}$ $second^{-1}$. Combining the two measured rates leads to a rate constant of 4.4 X 10^{-16} cm^3 $molecule^{-1}$ $second^{-1}$ at 298 ± 2°K for k(NO_3+ vinyl chloride). The measured average lower tropospheric nitrate radical concentration over continental areas ranges from less than 1 ppt to 430 ppt (Atkinson, et al., 1986). Assuming an average value of 10 ppt (2.4 X 18^8 molecule cm^{-3}) ± 10 would give a vinyl chloride lifetime of 220 days with respect to reactions with the nitrate radical.

As stated, the most important atmospheric removal mechanism for vinyl chloride is its daytime reaction with hydroxyl radicals. Vinyl chloride does not absorb in the actinic ultraviolet region, hence photolysis need not be considered.

Little is known about the formation of vinyl chloride in the atmosphere. However, under experimental conditions, vinyl chloride has been shown to be a photodissociation product of 1,2-dichloroethane (CH_2ClCH_2Cl) (Yano and Tschulkaw-Roux, 1980). In the study, 1,2-dichloroethane photodissociated when irradiated with ultraviolet light at 147 nanometers (nm) under pressure and in the presence of nitrous oxide (NO) and carbon tetrafluoride (CF_4) additives. Although it was not the purpose of the study to identify vinyl chloride formation pathways, vinyl chloride was one of the photodissociation products. Since wavelengths of ultraviolet light below 290 nm do not reach the troposphere, this formation pathway is not important for vinyl chloride in the atmosphere.

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APPENDICES

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APPENDIX I

SCAQMD'S ANALYTICAL METHOD FOR SAMPLING AND ANALYSIS OF ATMOSPHERIC VINYL CHLORIDE

AMBIENT AIR SAMPLES AT LANDFILL PARIMETER

(REQUIRED BY SUBPARAGRAPH (c)(4)(D) OF RULE 1150.1)

SAMPLING FREQUENCY

Once per month or at less frequent intervals to be determined by the Executive Officer. The landfill owner/operator must file a written request with the Executive Officer if he wants to sample at intervals less frequent than monthly. Such a request must be supported with previous sampling results and other documentation. In determining if the requested sampling frequency is appropriate, the Executive Officer will consider previous ambient air sampling results, landfill surface sampling results, landfill gas composition and other pertinent data. The Executive Officer will notify the landfill owner/operator of his decision in writing.

NUMBER OF SAMPLES

The number of ambient air samples required will depend upon the topography and the size of the landfill. At a minimum, samplers will be sited to provide good meteorological exposure to the predominant offshore (drainage land breeze) and onshore (sea breeze) wind flow patterns. In areas with significant slopes,

local nightly drainage patterns will also be sampled. All sampling locations must be approved by the Executive Officer prior to sampling.

SAMPLING CONDITIONS

Ambient air sampling will be conducted on days when stable (offshore drainage) and unstable (onshore sea breeze) meteorological conditions are representative for the season. Preferable sampling conditions are characterized by the following meteorological conditions:

1. Clear cool nights with wind speeds two (2) miles per hour or less.
2. Onshore sea breezes with wind speeds 10 miles per hour or less.

No sampling will be conducted if the following adverse meteorological conditions exist:

1. Rain
2. Average wind speeds greater than 15 miles per hour for any 30 minute period.
3. Instantaneous wind speeds greater than 25 miles per hour.

Continuously recorded on site wind speed and direction measurements will characterize the micrometeorology of the site and serve to verify that the meteorological criteria have been

met during sampling.

EQUIPMENT DESCRIPTION

An ambient air sampling unit consists of a 10-liter Tedlar (Dupont trade name for polyvinyl fluoride) bag, a DC operated pump, stainless steel capillary tubing to control the sample rate to the bag, a bypass valve to control the sample flow rate (and mimimize back pressure on the pump), a rotameter for flow indication to aid in setting the flow, a 24-hour clock timer to shut off the sampler at the end of the 24-hour sampling period, and associated tubing and connections (made of stainless steel, teflon, or borosilicate glass to minimize contamination and reactivity). The physical layout of the sampler is shown in Figure 5 (see Appendix A).

EQUIPMENT SPECIFICATIONS

A. Power -- one 12V DC marine battery

   The marine battery provides 12V DC to the pump and the clock.

B. Pump -- one 12V DC pump

   The diaphragm is made of non-lubricated Viton (Dupont trade name for co-polymer of hexafluoropropylene and vinylidene fluoride) rubber. The maximum pump unloaded flow rate is

4.5 liters per minute.

C. Bag -- one 10-liter Tedlar bag with a valve

TEDLAR BAG IS ENCLOSED IN A LIGHT-SEALED CARDBOARD BOX TO PREVENT PHOTOCHEMICAL REACTIONS FROM OCCURING DURING SAMPLING AND TRANSPORTATION. The valve is a push-pull type constructed of aluminum and stainless steel, with a Viton o-ring seal.

D. Rotameter

Rotameter is made of borosilicate glass and has a flow range of 3 to 50 cubic centimeters per minute. The scale is in millimeters with major graduations (labeled) every 5 mm and minor graduations every 1 mm.

E. Air flow control orifice -- 316 stainless steel capillary tubing

F. Bypass valve

G. Fittings, tubing, and connectors -- 316 stainless steel or teflon

H. Clock timer

Accuracy should be better than 1%.

I. Wind speed and direction monitor with continuous recorder

1. Wind speed -- 3 cup assembly, range 0 - 50 miles per hour with a threshold of 0.75 mile per hour or less.
2. Wind direction -- Vane, range 0 - 540 degrees with a threshold of 0.75 mile per hour or less.

SAMPLING PROCEDURES

Ambient air samples will be collected at the perimeter of the landfill over a 24-hour period beginning between 10 A.M. and 11 A.M. using the above described self-contained portable sampling units. The samplers will be placed at the approved locations as described previously. One or more wind speed and direction monitors with continuous recorders will be installed and operated in areas approved by the Executive Officer to measure wind speed and direction throughout the entire sampling period. The wind direction transmitter must be oriented to true north using a compass.

QUALITY CONTROL PROCEDURE

The following quality control procedure is required for the ambient air sampling operation:

A. Assign an identification number to each sampling bag.

B. Clearly mark sampling locations on a landfill topographic map which is drawn to scale.

C. Document the date and time that the bag was put into operation, the sampling location, and the date and time that it was pulled from service.

D. Check the clock timer. The clock time and the actual time should agree within ± 3 minutes.

E. Check whether or not the pump is running.

F. Check the rotameter reading. The float (measured at the middle) should be within +3 and -6 minor graduations of the marked setting for 6.0 cubic centimeters per minute. If the rotameter setting exceeds the above limits adjust the bypass valve to correct the flow rate. Make sure that the flow has stabilized (at least three minutes at constant flow) since there may be a lag time between the adjustment and final flow.

G. Check whether the bag valve is in the open position. If the valve is in the closed position open the valve and and record the time on the quality control sheet.

H. Remove the bag for analyses at the end of the 24-hour period. KEEP THE BAG IN A LIGHT-SEALED CONTAINER AT ALL TIMES.

Data for each sample collected must be entered on a quality control sheet as shown in Figure 3 (see Appendix A). Prior to use, the Tedlar bags should be evacuated and filled with purified nitrogen three times to flush out the old sample. Before sending the bags into the field, they should be checked to make sure that the vacuum has been maintained. Remove from service any bag that has experienced any leakage.

ANALYTICAL PROCEDURES

Bag samples collected must be analyzed within 72 hours of collection, or shorter period if notified by the Executive Officer, for total organic compounds and toxic air contaminants using analytical methods identified in Table 1 (see Appendix A) or equivalent methods approved by the Executive Officer. NOTE THAT ALL BAG SAMPLES MUST BE KEPT IN LIGHT-SEALED CONTAINERS TO AVOID PHOTOCHEMICAL REACTIONS.

REPORTING OF THE RESULTS

The following data must be submitted to the Director of Engineering within 45 days after the end of the quarterly reporting period for the landfill or 45 days after the analytical results are available whichever is sooner. A different submittal time may be implemented upon approval of the Executive Officer.

A. Volume concentration of total organic compounds (reported as methane and total non-methane hydrocarbons).

B. Volume concentration of toxic air contaminants identified in these guidelines.

C. Barometric sea level pressure (inches of mercury) on the days the samples were collected. If a barometer is not available at the landfill site, use the National Weather Service data at the nearest station.

D. Wind speed and direction data.

E. A drawn to scale landfill topographic map with sampling locations clearly marked and numbered.

F. Quality control data sheets.

VINYL CHLORIDE ANALYSIS -- METHOD A

Instrument: Hewlett Packard 5700A Gas Chromatograph

Detector: Flame Ionization

Injection System: Two Carle valves, a 10-port and a 4-port, are plumbed to contain a 4 ml, 1/4" stainless steel sample loop with pre-column, back-flush and pressure balance. See Figure D for valve plumbing.

GC Conditions:

Detector Temp. -	200°C
Oven Temp. -	60°C
Analytical Column -	6' x 1/4" ss, Chromosil 310, 60/80 mesh
Pre-Column -	6' x 1/8" ss, Durapack n-octane/Porasil C, 100/120 mesh
Carrier Gas -	80/100 ml/min nitrogen

Data Gathering: A Hewlett Packard 3388A Integrator is used to calculate concentration by peak area comparison to an external standard.

Valve Timing: Timing and switching events are performed by the integrator. 1.4 minutes after injection both valv are switched to the back-flush or initial position

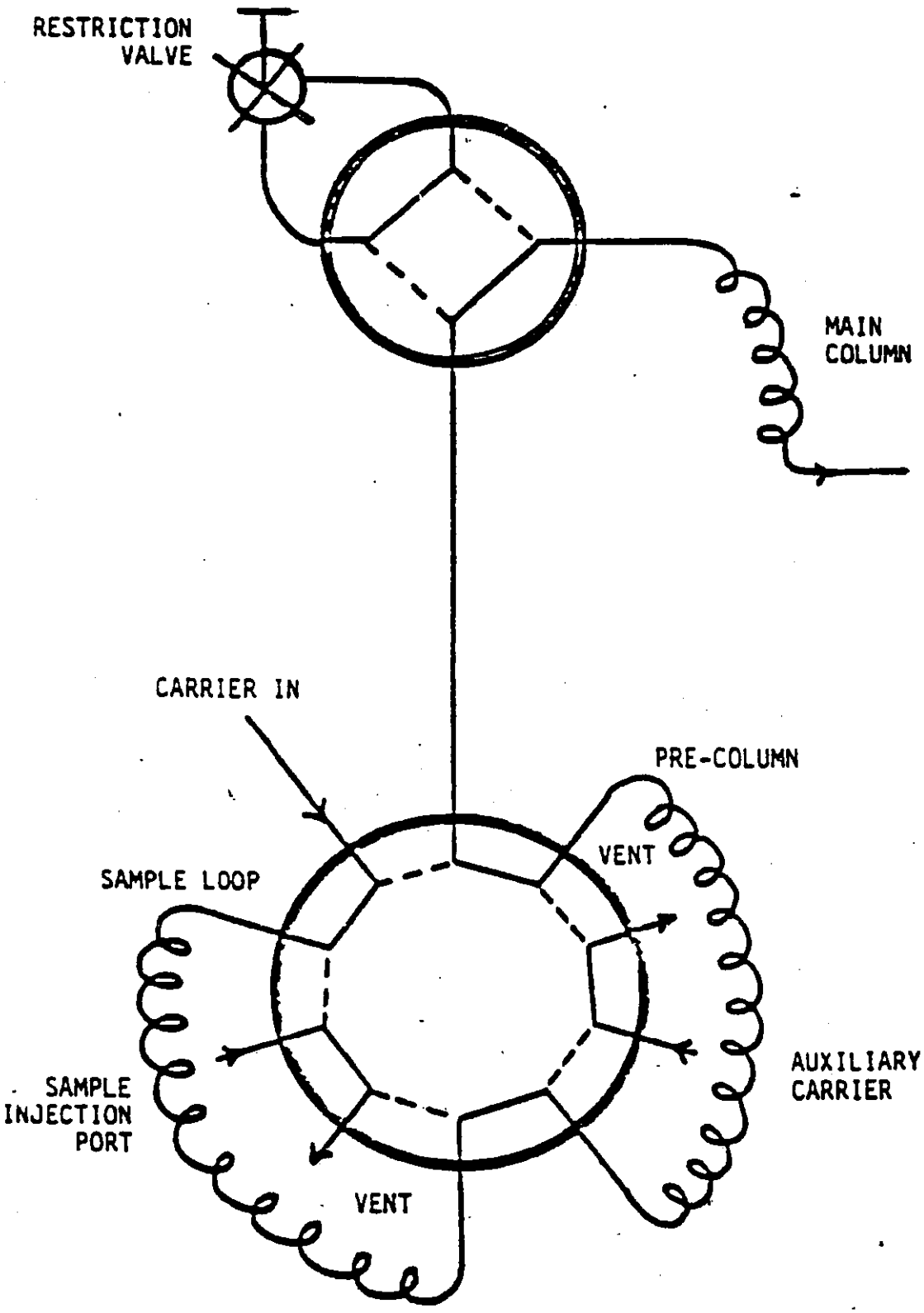

FIGURE D:
Valve Plumbing

Standard: Approximately 1 ppm vinyl chloride is prepared by Scott Environmental Technology and certified to ±2% analysis.

Range: 2 ppb to 1% vinyl chloride

Accuracy: ± 1 ppb in the range 2 - 50 ppb, ± 2 in the range 50 ppb to 1%

APPENDIX II

DESCRIPTION OF GLEIT'S METHOD

DESCRIPTION OF GLEIT'S METHOD

Gleit's method accounts for the concentrations below the LOD* by setting them equal to the 'below-LOD mean" μ_{BLOD}, the mean of the portion of the normal distribution below the LOD. Setting the unknown concentrations to their average value seems intuitively reasonable, and the simulations reported in Gleit's paper show that his method is more accurate than other commonly used approximations.

The below-LOD mean of a normal distribution of a variable with a limit of detection L is given, in terms of L and the mean μ and the standard deviation σ of the distribution, by equation 1:

$$\mu_{BLOD} = \mu - \sigma^{*}[f((L-\mu)/\sigma)/F((L-\mu)/\sigma)] \qquad (1)$$

In equation (1), f and F are, respectively, the probability density function and cumulative distribution function of the standard normal distribution. The "Estimated Concentrations for Samples Below the LOD" reported in Table II-2 are the below-LOD means of the assumed lognormal distributions of the concentrations. These below-LOD means are computed from equation (2) in terms of parameters of the associated normal distribution: the LOD L, the mean concentration from Table II-2, and the estimated standard deviation (which is not tabulated).

$$\exp(\mu+0.5^{*}\sigma^{2})^{*}F((L-\mu-\sigma^{2})/\sigma)/F(L-\mu/\sigma) \qquad (2)$$

We now describe how Gleit's method estimates the mean and variance of the assumed normal distribution. The mean and variance cannot be estimated by merely substituting into standard formulas, if below-LOD concentrations are to be set to the below-LOD mean. On the one hand, the mean and variance must be known in order to calculate the below-LOD mean from (1); on the other hand, the below-LOD mean must be known if it is to be used in the calculation of the mean and variance. Statistical theory, by asserting that a "best-fitting" mean and variance for the distribution exist, provides a way out of this dilemma. Gleit uses a simple iterative

procedure to compute these best-fitting parameters. Since his procedure can be simply described in words, a written description is given, supplemented where necessary by equations written in a notation more convenient than Gleit's.

Starting with initial guesses $\mu(0)$ and $\sigma^2(0)$ for the mean and variance, the procedure repeatedly generates new estimates of the mean and variance by the two-step computation described below until successive estimates of the mean and variance converge sufficiently (The K-th pair of estimates are denoted by $\mu(K)$ and $\sigma^2(K)$.). The two steps are:

(a) the K+1-st below-LOD mean $\mu_{BLOD}(K+1)$ is computed by substituting $\mu(K)$ and $\sigma(K)$ (the square root of $\sigma^2(K)$) into equation (1).

(b) The K+1-st estimate of the mean, $\mu(K+1)$, is computed in the usual way with $\mu_{BLOD}(K+1)$ substituted for the sample values below the LOD. The K+1-st estimate of the variance, $\sigma^2(K+1)$, is also computed in the usual way, with an analogous substitution for sample values below the LOD: the squared deviations from the mean of concentrations below the LOD are set equal to the average squared deviation from the mean of the below-LOD portion of the distribution.

Let the N sample items be X(1),.....,X(N), and let p be the number of sample items below the LOD. $\mu(K+1)$ is computed by:

$$\mu(K+1) = (1/N)\ \Sigma\ Y(J), \text{ where } Y(J)=X(J) \text{ if } X(J)\ R\ L$$
$$\text{and } Y(J)= \mu_{BLOD}(K+1) \text{ otherwise}$$

$\sigma^2(K+1)$ is computed by:

$$\sigma^2(K+1)=(1/N)\ \Sigma\ D^2(J), \text{ where } D^2(J)=(X(J) - \mu(K+1))^2$$
$$\text{if } X(J)\ \ L, \text{ and } D^2(J)= \sigma^2_{BLOD}(K+1) \text{ otherwise.}$$

The quantitiy $^2\sigma_{BLOD}(K+1)$, the average squared deviation of the below-LOD portion of the distribution, is computed from the following equation:

$$^2\sigma_{BLOD}(K+1) = \sigma^2(K)^*[1-Z(K)^*(f(Z(K))/F(Z(K)))],$$
$$\text{where } Z(K)=((L-\mu(K))/\sigma(K)).$$

Gleit's method nearly always converges in a few steps unless there are only a few distinct values above the detection limit, in which case it may converge very slowly. Gleit's method and closely related methods appear to be the best available estimators of the mean when the sample includes values below the LOD, as is demonstrated by the simulations reported in Gleit's paper.

* See Appendix VII for the ARB Monitoring and Laboratory Division's method for calculating the LOD and Section A. Chapter III for the South Coast Air Quality Management District laboratory's method for calculating the LOD.

APPENDIX III

ESTIMATE OF TOTAL EXPOSURE TO VINYL CHLORIDE FROM INDOOR AIR

INDOOR AIR EXPOSURE/OTHER ROUTES OF EXPOSURE ASSESSMENT FOR VINYL CHLORIDE

I. BACKGROUND

Health and Safety Code Section 39660.5 directs the Board, in its toxic air contaminants identification process, to assess exposures to toxic air contaminants in indoor as well as outdoor environments. Indoor exposure assessment has become increasingly important as an integral part of air exposure assessment because (ARB 1987, 1989):

1. people spend a predominant proportion of their time indoors; and

2. personal and indoor air monitoring data indicate that some pollutant concentrations are regularly higher indoors than outdoors.

Indoor air exposure data, combined with outdoor air exposure data, can provide a realistic estimate of personal exposure through the air environment. A more detailed discussion of indoor air exposure is contained in Appendix A.

Indoor air data can be obtained either by personal air sampling or by fixed-site air sampling. In personal sampling, the sampling equipment is carried by an individual and air samples are taken wherever the individual may be. In contrast, fixed-site air samplings refer to air samples taken at a fixed location. Personal air sampling data generally provide a more realistic estimate of individual exposure. Since most people spend 80-90% of their time in indoor environments, personal air sampling data are strongly weighted by indoor air exposure data.

While the main objective of this report is to define exposure through the air, this report also presents personal exposure data through other media. The inclusion of these data will provide an useful perspective of the overall exposures to toxic air contaminants through environmental media. The need for total exposure assessment and some of the issues and concepts involved in total exposure estimates are discussed in Appendix B.

II. INDOOR AIR EXPOSURE TO VINYL CHLORIDE

A. PERSONAL AIR SAMPLING

Personal air sampling data for most organic compounds come from the Total Exposure Assessment Methodology (TEAM) studies conducted by the Environmental Protection Agency (EPA) during 1980-85 (Wallace, 1987; USEPA 1987a,b; Wallace & Clayton, 1987; Wallace et al., 1986; Pellizzari et al., 1986). Although vinyl chloride was included in the initial pilot study (Phase I) of the TEAM project, vinyl chloride was deleted from the subsequent main studies (Phase II and III). The deletion of vinyl chloride was due to two factors (Pellizzari, 1987). First, Tenax, the most cost-effective sampling

medium which could collect a number of compounds of concern, was not suitable for vinyl chloride collection. In addition, the alternative sampling method used to collect vinyl chloride in the pilot study did not provide the required reliability for detecting low vinyl chloride concentrations.

Consequently, the pilot study provides the only available personal air sampling data for vinyl chloride. Based on this limited information, indoor air exposure to vinyl chloride is apparently low. In monitoring nine subjects in New Jersey and three from North Carolina for several days on three separate visits over a 6-month period, all of the 138 air samples (collected in 5 to 10 hour sampling periods) were below the reported limit of detection (LOD) of 20 ppb (51 ug/m^3)[1] (Wallace et al. 1984). The procedure for calculating the LOD was not described in the paper and the subcontractor who conducted the monitoring could not be located for the information.

B. FIXED-SITE AIR SAMPLING

As part of a recent follow-up TEAM study in California, fixed-site monitoring stations were installed to monitor indoor and outdoor air concentrations of a number of organic compounds (Pellizzari, et al., 1989). Specially designed stainless steel canisters were used for collecting vinyl chloride air samples from homes in the Los Angeles area for two seasons. Ten homes were sampled in the Winter season and eight of the original homes were sampled in the Summer season. Canister air samples were collected indoors and outdoors at each home during two, 12-hour periods. Samples obtained in the Winter season did not provide reliable data due to technical problems. All outdoor or indoor samples, a total of 32 samples, obtained in the Summer season indicated that vinyl chloride air concentrations were below the limit of detection. The samples were analyzed by two analytical methods with limits of detection at about 0.2 and 58 ppb (0.55 and 148 ug/m^3), respectively. The LOD, as defined by Pellazzari, et al., is a value where the measured signal of the analyte is three times that of the noise of the instrument. Therefore, values below the LOD are not reported.

A similar TEAM study was conducted in Baltimore. Indoor air concentrations of vinyl chloride in about 160 homes were monitored by fixed-site sampling stations. Based on partially analyzed results, vinyl chloride was not detected in indoor air environments. The limit of detection was quoted by the researcher as 10 to 16 ppb (26 to 40 ug/m^3) (Pellizzari, 1987).

C. SPECIAL SITUATION AIR MONITORING

In 1981, the South Coast Air Quality Management District (SCAQMD) collected 24-hour bag samples in the vicinity of the BKK landfill (a Class I site) in West Covina. A total of more than 500 air samples were taken at two outdoor sites and at four sites inside downwind residences (SCAQMD, 1982). All the samples (24% of the total sampled) that equaled or exceeded the state vinyl chloride air quality standard of 10 ppb (26 ug/m^3) were taken inside the

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1 1 ug/m^3 x (0.0245/MW) x 10^3 = 1 ppb

residences. The highest recorded indoor vinyl chloride concentration was 50 ppb (130 ug/m^3). The limit of detection was 2 ppb (5.2 ug/m^3) (see Section A, Chapter III for the SCAQMD method of calculating the LOD).

In late 1984, the SCAQMD staff screened for landfill gas migration from Operating Industrial, Inc. (OII) Landfill by taking about ten grab-samples inside the water meter boxes of residences adjacent to the landfill (SCAQMD, 1985a). A water meter box is a below-ground, enclosed box containing an apparatus which measures the amount of water used by a household. Grab samples from the water meter boxes showed vinyl chloride concentrations ranging from 13 to 36000 ppb (31.2-93600 ug/m^3). In 1985, the SCAQMD (1985b) conducted further monitoring by grab-samples inside some of the residences and found indoor vinyl chloride air concentrations at 8 to 100 ppb (20.8 to 260 ug/m^3). Present indoor concentrations of vinyl chloride in these residences near OII landfill may be lower since monitoring of water meter boxes has not detected significant levels of landfill gases due to improvements in OII's landfill gas collection system (Coy, 1987).

C. SUMMARY

Except for houses near landfills, the vinyl chloride concentration in indoor air appears to be low. However, this conclusion is based on the evaluation of a very limited database. In addition, the sampling and analytical procedures for vinyl chloride indoor air monitoring are less than satisfactory as evidenced by the wide range for reported limits of detection. The limit of detection, 0.2 ppb (0.55 ug/m^3), reported in the latest California TEAM study appears to be the most reliable. This limit of detection will be used to estimate the upper limit exposure for houses not adjacent to landfills (see Section II B of this appendix for the TEAM study method of calculating the LOD).

For houses near landfills, the measured high indoor vinyl chloride air concentrations may indicate the potential impact of nearby emission sources to indoor environments. A more detailed discussion of landfill emissions as a source of indoor vinyl chloride is presented in section III(C).

III. POTENTIAL SOURCES OF INDOOR VINYL CHLORIDE

A. PLASTIC MATERIALS AND CONSUMER PRODUCTS

Vinyl chloride has not been used in any consumer products since 1974 when vinyl chloride was banned as a propellant in household aerosol products and as an ingredient of drug and cosmetic products (IARC, 1979).

Because of its versatility, plastic products made of polyvinyl chloride (PVC) and other vinyl chloride polymers are ubiquitous in any household. Before being made into different products, PVC polymer is in the form of a resin that is made by chemically linking the vinyl chloride molecules. Individual vinyl chloride molecules are also called vinyl chloride monomer (VCM). Unreacted VCM can remain in the PVC resin for some time depending on the initial amount of the unreacted VCM. Therefore, an indirect source of vinyl chloride indoors may come from the release of unreacted VCM from these plastic products. For example, during 1975 to 1976, VCM concentrations

ranging from below 2 ppb to 1.2 ppm (5.2 to 3,077 ug/m^3) were measured in automobile interior air space under experimental conditions (U.S.EPA, 1976; 1977).

Emissions of unreacted VCM have been greatly reduced due to improvements in monomer stripping technology (Wheeler, 1981). In the past, residual VCM concentrations in the PVC resins at the time of shipment ranged as high as 2000 ppm (5,128 mg/m^3). Currently, PVC resins contain about 10 ppm (26 mg/m^3) residual VCM at the time of shipment and may lose VCM at a rate of 20 to 50% per month during storage. In addition, most of the VCM will vaporize and escape during the high temperature processes in which PVC resins are melted and made into final products. Thus, commercial products made of PVC resins do not now contain significant residual vinyl chloride for later emission.

B. VAPORIZATION FROM WATER SOURCES

Water can serve as a medium to carry pollutants from outdoor to indoor environments. Once in contact with air indoors, volatile chemicals such as vinyl chloride can leave the water and enter the air. Human activities such as using water for cooking, heating or showering can promote rapid vaporization of vinyl chloride from water. Industrial solvent contaminated surface or ground water may, therefore, bring outdoor vinyl chloride indoors via the water supply.

In California, surface water is generally free of vinyl chloride (Sharrp, 1987). In assessing ground water quality, the California Department of Health Services (CDHS, 1986) reported that only one out of the 2,947 wells for large public water systems was contaminated with vinyl chloride. The maximum concentration found in that well was 23 ug/l with a median value of 20 ug/l. Vinyl chloride has not been detected in wells used for small public water systems (CDHS, 1987). The limit of detection of vinyl chloride in water is 0.5 ug/l. Based on this information, vinyl chloride in the water supply will have an insignificant impact on the indoor vinyl chloride air concentration.

C. VINYL CHLORIDE FROM LANDFILL GAS

Homes built on or near landfills containing vinyl chloride or related chlorinated hydrocarbons may have high indoor air concentrations of vinyl chloride. Vinyl chloride emission from landfills can be caused by the vaporization of vinyl chloride that was originally disposed there. Class I landfills that are designated for toxic waste are likely to contain vinyl chloride waste. In addition, microbiological conversion of chlorinated hydrocarbons can produce and emit vinyl chloride <u>in situ</u> (Molton, Hallen and Pyne, 1987).

Wood and Porter (1987) reported their evaluations of over 20 Class II landfills that are designated only for municipal waste. Ninety percent of these landfills contained measurable amounts of vinyl chloride and the concentrations at half of these landfills were above 1000 ppb (2,564) ug/m^3). These high concentrations were measured by grab-sampling, an instant filling of a two-liter evacuated flask, at ground levels or at landfill gas collection points. For five of the landfills, 24-hour bag sampling was also conducted. Only one of these five landfills produced measurable 24-hour concentrations of vinyl chloride off-site.

There are at least two ways that vinyl chloride from landfills may contribute to indoor vinyl chloride concentrations of nearby residential houses. First, houses that are located downwind from landfills can receive vinyl chloride through direct outdoor air influx into indoor environments. Secondly, landfill gases, carrying vinyl chloride, can migrate underground and enter houses through substructures. The rate of accumulation of vinyl chloride indoors depends heavily on the soil permeability, source strength, air exchange rate and structure of the house. Higher indoor than outdoor vinyl chloride concentrations may occur because vinyl chloride is more rapidly destroyed in outdoor than indoor air. Outdoor destruction proceeds more quickly because vinyl chloride's reaction with hydroxyl radicals is the compound's dominant atmospheric removal mechanism and because hydroxyl radicals are formed in the presence of direct sunlight.

As discussed in Section II(B), houses located near Class I landfills had higher indoor than outdoor air concentrations of vinyl chloride. The accumulation of high vinyl chloride concentrations in the water meter boxes indicated that landfill gas containing vinyl chloride can migrate underground and enter nearby indoor environments. Controlled release or combustion of landfill gas on site may slow down vinyl chloride subterranean migration.

D. OTHER FACTORS THAT MAY INFLUENCE INDOOR CONCENTRATIONS

A minute amount of vinyl chloride has been identified in the smoke of cigarettes (1.3-16 ng/cigarette) and of little cigars (14-27 ng/cigar) (IARC, 1985; Hoffmann, Patrianakos and Brunnemann, 1976). The vinyl chloride level in the mainstream smoke may be determined by the total inorganic chloride content of the tobacco. The contribution from tobacco smoke appears to have insignificant impact on the indoor air concentration of vinyl chloride.

E. SUMMARY

In general, there are very few, minor emission sources of vinyl chloride indoors. However, houses that are situated near landfills may accumulate vinyl chloride in the indoor environment due to subterranean gas migration and direct air infiltration. Some of these houses may have indoor air levels of vinyl chloride higher than the State of California Ambient Air Quality Standard for outdoor vinyl chloride.

The results from the SCAQMD's five hundred 24-hour bag samples (the highest measured 24-hour averaged concentration was 50 ppb or 130 ug/m^3) can be used to estimate the upper limit of indoor air exposure to vinyl chloride in houses near landfills (SCAQMD, 1982). The results obtained by grab-sample monitoring, however, are not necessarily reflective of long-term indoor exposure to vinyl chloride.

IV. OTHER ROUTES OF VINYL CHLORIDE EXPOSURE

A. WATER INGESTION

The major source of drinking water for California is surface water which does not have detectable vinyl chloride concentrations. Ground water used for public water systems is also relatively free of vinyl chloride (CDHS, 1987, 1986). The detectable limit of vinyl chloride in water is 0.5 ug/l (0.5 ppb). Based on this information, vinyl chloride exposure through drinking water is judged to be insignificant under ordinary situations.

B. FOOD INGESTION

Vinyl chloride is not one of the compounds that have been monitored routinely in U.S. food and food products. However, before 1973, vinyl chloride was found in food and beverages marketed in vinyl chloride polymer containers or packaging materials (IARC, 1979). At that time, levels as high as 20 mg/kg (ppm) of vinyl chloride monomer were present in alcoholic beverages packaged in this material. Vinyl chloride was also found in edible oils, butter and margarine at 0.05-14.8 mg/kg.

When cleaner PVC resins became available after 1975, vinyl chloride polymer containers contained only about 10 ppb of residual vinyl chloride monomer. In its recent rule-making proposal, the Food and Drug administration (FDA) (1986) estimated vinyl chloride exposure from food and beverages packaged with vinyl chloride polymer materials. These materials include liquor bottles, wine bottles, oil bottles, vinyl chloride homopolymer film, and materials made with vinyl chloride-vinylidene chloride copolymers. Based on a conservative approach, the FDA's estimated lifetime-averaged individual exposure to vinyl chloride would not exceed 25 nanograms per day.

V. ESTIMATES OF TOTAL EXPOSURE FROM INDOOR AIR AND OTHER ROUTES

The estimated daily dose of vinyl chloride from different environmental media are presented in Table 1. From the Table, exposure to vinyl chloride in general indoor air, food and water appears to be insignificant. However, exposure to vinyl chloride indoors in homes near landfills may be the major portion of total vinyl chloride exposure.

A. INDOOR AIR EXPOSURE

The average concentration of vinyl chloride indoors in houses not near landfills is estimated to be below the limit of detection (0.2 ppb or 0.55 ug/m^3). For homes that are located near landfills, the highest observed daily average measurement, 50 ppb or 130 ug/m^3, is used for a conservative estimate.

B. FOOD INGESTION

The estimate of daily dose reported by FDA (1986) is directly used.

C. DRINKING WATER

The relative contribution of drinking water to daily exposures of vinyl chloride appears to be insignificant. The average concentration of vinyl chloride in drinking water is estimated to be below the limit of detection (0.5 ppb or 0.5 ug/l).

D. ASSUMPTIONS

Some of the assumptions used for making the daily dose estimates from different environmental media are:

1. The average person ingests 2 liters of drinking water per day;

2. The average person inhales an average of 20 cubic meters of air daily;

3. Dermal exposure is negligible; and

4. 100% of the pollutant ingested or inhaled is absorbed.

Table 1: Estimated Doses Of Vinyl Chloride Exposure Through Different Media

Media	Daily Dose	Refs.
AIR		
Indoor Air		
Homes not near landfills	less than 11 ug	Pellizzari et al., 1989
Homes near landfills	up to 2600 ug	SCAQMD, 1982
FOOD		
Including beverages	less than 0.025 ug	FDA, 1986
WATER-DRINKING PURPOSES		
Surface/Ground Water	less than 1 ug	CDHS, 1986; 1987

APPENDIX A - INDOOR AIR EXPOSURE

Prediction of health risk from pollutants depends upon knowledge of total personal exposure to the pollutants. For direct exposure to air pollutants, the dose of pollutant received through the respiratory system is the basic quantity needed for risk assessment. In general, that dose depends on: a) the pollutant concentration in the environment occupied by an individual (exposure concentration); b) the length of time spent in that environment (exposure duration); c) the rate of breathing in that environment; and d) other physiological factors. Exposure through air can be estimated by using only the first two parameters, exposure concentration and exposure duration.

Historically, outdoor air concentrations of an air pollutant have been used as a surrogate for estimating personal air exposure. However, studies of indoor environments and of personal exposures to pollutants have revealed that indoor concentrations of some pollutants are regularly higher than outdoor concentrations of those pollutants. In addition, human time-activity pattern studies show that people spend most of their time in non-outdoor microenvironments such as in their homes, work places, transportation vehicles and public buildings. On the average, people spend 80-90 percent of their time indoors.

The California Legislature recognizes the importance to risk assessment of considering both indoor air exposure and outdoor air exposure. The current statute requires the Board, when identifying toxic air contaminants, to assess exposures in indoor, as well as outdoor, environments (H&SC Sec. 39660.5). This combined indoor plus outdoor, or total air, exposure assessment permits more accurate public health risk estimates for airborne toxics. Indoor air exposure information can also provide direction for the control of many toxic air contaminants.

An even more realistic estimate of total air exposure would be the sum of the products of the pollutant concentration in each microenvironment and the fraction of time people spend in that microenvironment. However, time-activity and indoor/personal monitoring data are limited and insufficient at this time for quantifying the concentration of most air pollutants in each microenvironment. Based on this limited database, indoor air exposure assessment of most of the toxic air contaminants will be crude estimates.

Risk assessments, based only on outdoor air concentrations, may greatly underestimate health risk to the public. For some pollutants present in high concentrations indoors, ventilation with clean air is the only feasible method of reducing exposure. The Board must, therefore, manage outdoor concentrations of toxic air contaminants, not only to reduce significant outdoor exposures where they occur, but also to preserve a clean air supply for controlling indoor exposure to these substances.

APPENDIX B - TOTAL EXPOSURE FROM ALL MEDIA

The concentrations of some pollutants have been measured in different environmental media such as air, water, food, pesticides and drugs. Ideally, these measurements can be integrated to estimate the total exposure to those pollutants through all the environmental media. Total exposure data are critical for setting priorities and formulating regulatory actions that can best achieve overall personal risk reduction.

While one of the main objectives of this report is to define exposure through the air medium, personal exposure data through other media are also included. Exposure data are presented according to three basic routes of exposure which are inhalation, ingestion, and skin absorption.

The combination of exposure data from all media will allow the determination of the total human exposure to a toxic air contaminant through the environment. To determine the added risk caused by a particular exposure, both the shape of the dose-response curve and the previously existing exposure level must be known. Although the exposure through a particular medium may be small, its addition to exposures through other media could provide a total dose in excess of a postulated "safe level".

In addition, the pathway of pollutants in the environment is dynamic and complex. Pollutants emitted into the environment in one medium can remain in that medium, transfer to another medium, and/or disperse into a number of media. This results in different routes of exposure. For example, solvents emitted as water pollutants can become airborne and cause exposure through inhalation. Airborne lead particles can be deposit onto food and result in exposure through ingestion. Thus, inclusion of exposure through all media will provide a more accurate exposure estimate for each route of exposure, including inhalation, which is the Board's primary concern.

Documenting exposure to toxic substances through different media can serve as a stimulus for coordinated risk reduction efforts among different regulatory agencies. Other regulatory agencies are more likely to increase their efforts in reducing the overall exposure through other media if they are made aware of such exposure data.

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APPENDIX IV

INFORMATION REQUEST LETTER WITH ATTACHMENTS AND RESPONSES

STATE OF CALIFORNIA GEORGE DEUKMEJIAN, Governor

AIR RESOURCES BOARD
1102 Q STREET
P.O. BOX 2815
SACRAMENTO, CA 95812

April 4, 1985

Dear Sir or Madam:

Subject: Request for Information Regarding Vinyl Chloride

I am writing to request information on the health effects of vinyl chloride as part of our toxic air contaminant program. This program is based on Health and Safety Code Sections 39650, et seq. which require the ARB to identify compounds as toxic air contaminants and once identified to develop and adopt control measures for such compounds. After consultation with the staff of the Department of Health Services (DHS), we have selected vinyl chloride as a candidate toxic air contaminant to be evaluated in accordance with the provisions of Health and Safety Code Sections 39650, et seq. During our evaluation of vinyl chloride, we will consider all available health information regarding this compound. Additionally, we are soliciting information regarding possible biological production of vinyl chloride.

Before the ARB can formally identify a compound as a toxic air contaminant, several steps must be taken. First, the ARB must request the Department of Health Services to evaluate the health effects of candidate compounds. Second, the ARB staff must prepare a report which includes the health effects evaluation and then submit the report to a Scientific Review Panel for its review. The report submitted to the Panel will be made available to the public. Information submitted in response to this request will be considered in the ARB report to the Panel. Although any person may also submit information directly to the Panel for its consideration, I urge you to submit all information at this time for our consideration in the development of the report for the Panel. The Panel reviews the sufficiency of the information, methods, and data used by the DHS in its evaluation. Last, after review by the Scientific Review Panel, the report with the written findings of the Panel will be considered by the Air Resources Board and will be the basis for any regulatory action by the Board officially to identify a compound as a toxic air contaminant.

Prior to formally requesting the DHS to prepare a health effects evaluation of vinyl chloride, we are providing, pursuant to the provisions of

Section 39660(e) of the Health and Safety Code, an opportunity to interested parties to submit information on the health effects of vinyl chloride which he or she believes would be important in DHS's evaluation of vinyl chloride as a candidate toxic air contaminant.

In March 1985, we received a reference search on vinyl chloride health effects using the MEDLINE and TOXLINE Information Services. These information services include material available to the public in late 1984. The attached bibliography lists the references from this information search. We are requesting pertinent information on vinyl chloride health effects, including any material that may not be available to the public, that is not included in the attached bibliography.

Pursuant to the provisions of the Public Records Act (Government Code Sections 6280 et seq.), the information you provide will be a public record and subject to public disclosure, except for trade secrets which are not emission data or other information which is exempt from disclosure or the disclosure of which is prohibited by law. The information may also be released to the Environmental Protection Agency, which protects trade secrets and confidential information in accordance with federal law, and to other public agencies, which are also required to protect such information.

To expedite the review process, we ask that any information which you believe should be regarded as "trade secret" be clearly marked and separated from other information. You may identify portions of the information you submit as "trade secret" in accordance with Health and Safety Code Section 39660(e). The claim of trade secrecy must be supported upon the request of the Air Resources Board. Other information claimed to be trade secret and information otherwise claimed to be exempt from disclosure may be identified as confidential in accordance with Section 91011, Title 17, California Administrative Code. Section 91011 requires that the claim of confidentiality be accompanied by specified supporting information.

I would appreciate receiving any relevant information you wish to submit by May 19, 1985. Your help in expediting our review will be greatly appreciated. Please send the information to the attention of:

William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Vinyl Chloride
California Air Resources Board
P. O. Box 2815
Sacramento, CA 95812

If you have any further questions regarding health effects information, please contact Mr. John Batchelder at (916) 323-1505. For any other questions, please contact Mr. Don Ames at (916) 322-8285.

If you are not the person to whom this request should be addressed, please forward it to the appropriate person in your organization. Also, please let us know whether you would like to continue to receive information inquiries for other candidate compounds, and if not, if there is anyone in your organization to whom such requests should be sent.

Sincerely,

for Peter D. Venturini, Chief
Stationary Source Division

cc: Alex Kelter, DHS
Lori Johnston, DFA
Wayne Morgan, President, CAPCOA
Jan Bush, Executive Secretary, CAPCOA
David Howekamp, EPA Region IX
Assemblywoman Sally Tanner, Chairwoman, Committee on Toxic Materials
Senator Ralph Dills, Chairman, Committee on Governmental Organization
Senator Art Torres, Chairman, Committee on Toxics and Public Safety Management
Emil Mrak, Chairman and Scientific Review Panel Members
APCOs

Attachment

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To: William V. Loscutoff

From: David D. Doniger

Date: April 10, 1985

Subject: Vinyl Chloride

The attached is in answer to your recent inquiry.

TCM

The Law and Policy of Toxic Substances Control

A Case Study of Vinyl Chloride

DAVID D. DONIGER

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dards, however, give virtually no guidance on the relative weight to be accorded health and environmental interests in comparison to economic ones, and to date, the agencies' actions reflect Congress's lack of consensus on the issue of acceptable risk.

A measure of guidance to decision makers in the face of uncertainty and lack of consensus is provided by the observation that regulatory decisions involve moral as well as economic values. We may begin with the observation that the sacrifice of an individual for the benefit of a group is acceptable if the benefit served is the group's survival or the fulfillment of some other basic need. The sacrifice is morally unacceptable, however, if it is for no more important benefit than the provision of the luxuries of our consuming society. That some must die so that all can eat is one thing; that some must die so that all can have see-through food packaging is another.[80b] Particularly where non-essential products are concerned, the long-term goal of toxic substances control and the long-term effect of each regulation should be to channel economic growth away from industries hazardous to health and towards safer products and forms of employment.

As the case study in Part II shows, the problems of deciding under uncertainty and of balancing incommensurable interests have pervaded the regulation of VC. Reluctance to face these difficult problems accounts in part for the fact that in more than four years the agencies have set standards for only two of the major sources of exposure to VC. While the standards that have been set and the proposals that have been put forward are by no means totally deficient, there have been significant instances in which the agencies have made factually or logically insupportable conclusions, or in which they have ignored evidence and failed to draw conclusions the evidence virtually demands. In all instances, the agencies have held back from imposing standards that would require any significant economic change in the regulated industries. The industries' profits, volume of production of the regulated substances, and future growth prospects have been virtually unaffected. One may question whether the benefits of VC production and use are substantial enough to justify such extreme deference to the

80b. Many value judgments are not so easily made as the distinction between food and food packaging. Typically, economists take the position that neutrality is required at all times in this regard, because of the difficulty of making so many of these value judgments. *See, e.g., Evaluation of Life and Limb*, *supra* note 74, at 695-96, 703. But the difficulty of making the hard decisions does not require us to avoid making the clearest ones. And one need not accept the view that the values of a society must be regarded as inviolate. They change in a manner not fully understood, but certainly not free from the influences of groups, such as the business community, with strong financial interests in promoting the materialistic, consuming behavior of the public. As Professor Tribe puts it: "[W]e cannot simply assume that we must stand mute when confronting the ultimate question of whether we want our children, and their children's children, to live in—and *enjoy*—a plastic world." Tribe, *Ways Not to Think About Plastic Trees* in When Values Conflict 61, 70 (L. Tribe, C. Schelling, & J. Voss eds. 1976) (emphasis in original). *See also* Dorfman, *An Afterword: Humane Values and Environmental Decisions*, in *id.* at 153-71.

industries' market position. This question is pursued in the sections that follow, each detailing the regulatory situation with regard to one or more of the many sources of human exposure to VC.

II

VINYL CHLORIDE CASE STUDY

A. *An Introduction to Vinyl Chloride*

In the previous section the problems of deciding under uncertainty and determining socially acceptable risks were discussed as they apply to toxic substances regulation generally. In this case study, these problems are considered as they have affected agency decision making with respect to the regulation of vinyl chloride. The case study begins with essential background material on the applications of VC, on the industries which create, transform, and use it, and on its deadly properties. Subsection 1 surveys the chemical's uses and the industries associated with them. Subsection 2 surveys VC's toxicity and the sources of human exposure to it.

1. *Vinyl Chloride's Uses and the Associated Industries*

The carcinogen vinyl chloride is the basis of the second most widely used plastic in the United States.[81] VC, a gas, is made from petrochemicals and chlorine. When polymerized into polyvinyl chloride, a solid, it is fabricated into a phenomenal array of products. VC was first manufactured commercially in the United States in 1939;[82] by 1976, VC production exceeded 5.5 billion pounds.[83]

The wide variety of uses of PVC is testimony to its adaptability. The major use of PVC is in construction products; other important uses are packaging and consumer products of all kinds. Figure 1 summarizes the applications of PVC in 1974. Woods, metals, glass, other plastics, and other materials can substitute for nearly all of PVC's uses, but PVC is preferred because of better performance or lower cost.[84] However, there are only a few uses for which no direct substitutes exist.[85]

Several direct uses of VC gas itself once existed, but these have been discontinued. In the late 1940s, VC was tested for use as an anaesthetic, but

81. *See Thermoplastics Poised For a Good Five Years*, Chem. & Eng'g News, Nov. 8, 1976, at 15. Polyethylene is the highest volume plastic. *Id.*

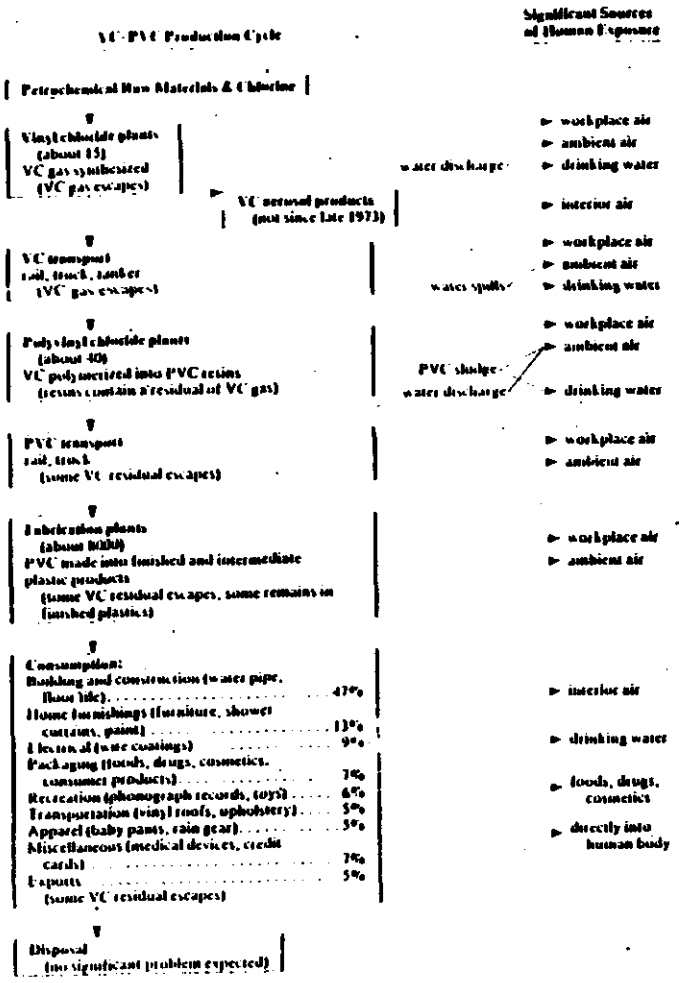
82. *OSHA Permanent Standard for VC*, *supra* note 1, at 15,890.

83. *Key Chemicals: Vinyl Chloride*, Chem. & Eng'g News, Aug. 23, 1976, at 13.

84. *Second Thoughts On Using PVC*, Chemical Week, July 31, 1974, at 19.

85. U.S. Environmental Protection Agency, Standard Support and Environmental Impact Statement: Emission Standard for Vinyl Chloride 2-4 (Oct. 1975) (hereinafter cited as EPA EIS); U.S. Environmental Protection Agency, Preliminary Assessment of the Environmental Problems Associated With Vinyl Chloride and Polyvinyl Chloride, Report on the Activities and Findings of the Vinyl Chloride Task Force at app. 1, table 2 (Sept. 1974) (hereinafter cited as EPA Task Force Report).

FIGURE 1
THE LIFE CYCLE OF
VINYL CHLORIDE POLYVINYL CHLORIDE



it was rejected because it upset cardiac function.[86] There are also indications that VC was once used as a refrigerant in cooling equipment.[87] Until late 1973, a small percentage of the VC produced was used as an aerosol propellant in some cosmetics, drugs, pesticides, and other consumer products. In 1974, when VC's carcinogenicity became generally known, millions of VC-propelled aerosols were still on the market or in consumer hands. The use of VC in aerosols has since been prohibited.[88]

Figure 1 illustrates the cycle of VC's creation, transformation, use, and disposal, and the routes of human exposure. There are three industries of central importance. The VC industry produces VC from petrochemicals. The PVC industry polymerizes the gas into the solid plastic, known in its raw form as resin. The fabrication industry converts PVC resins into finished products ready for consumer use or for incorporation into products of other industries. It is in these three industries that the workers are most heavily exposed to VC, and that the known human cancers have occurred.

Outside of these plants, additional people are exposed to VC in two major ways. First, VC emissions escape from factories to the surrounding air.[89] Second, since the polymerization process is imperfect, some VC remains a gas trapped in PVC materials. This residual escapes from the plastic in later production in fabrication plants and beyond, and in subsequent use and disposal.

As one moves through the production cycle of VC and PVC, the number of plants and companies increases, and plants become smaller and more labor intensive. The VC industry is composed of 10 companies operating 15 plants; in 1973, Shell, Dow, and Goodrich together held 56 percent of capacity.[90] In 1975, 23 companies operating 37 plants comprised the PVC industry. Goodrich is the major PVC producer with 15 percent; Firestone, Conoco, Union Carbide, Borden, Diamond Shamrock, and Tenneco each produce between five and nine percent.[91] There are about 8,000 fabrication companies of all sizes.[92] Beyond this point industries cease to be identified primarily by their use of VC.

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86 Oster, Carr, Kramer, & Sasmovich, *Anesthesia XXVII Nurses with Vinyl Chloride*, 8 Anesthesiology 359, 361 (1947).

87 2 I. Patty, Industrial Hygiene and Toxicology 811 (1963).

88 The fate of these aerosols is discussed in the text accompanying notes §§1-556 *infra*.

89 Most VC escapes directly into the air. Some leaves the plants in effluent water; most of this VC evaporates into the air. Some, however, enters drinking water. *See* EPA Task Force Report, *supra* note 85, at 5, 10. Appendices at 11; Environmental Protection Agency, Preliminary Assessment of Suspected Carcinogens in Drinking Water (Report to Congress) 2, 26-30, 35-39 (1975) [hereinafter cited as EPA Preliminary Report on Drinking Water Contaminants].

90 Office of Research and Development, U.S. Environmental Protection Agency, Scientific and Technical Assessment Report on Vinyl Chloride and Polyvinyl Chloride 20 (June 1975) [hereinafter cited as EPA Scientific and Technical Report].

91 *Id.* at 21-23.

92 EPA TIS, *supra* note 85, at 1-23.

As the above figures indicate, the VC and PVC industries are substantially concentrated. They are also vertically integrated. In 1972, 34 percent of VC produced was sold to PVC plants owned by VC companies, although this figure had dropped from 61 percent in 1962 as the industries grew.[93]

The fact that the VC and PVC industries contain only a small number of relatively concentrated and integrated firms has made it easy for the industries to speak with one voice in regulatory proceedings regarding the limits of their technological and economic capabilities to control VC exposures. The industrial market structure also makes it difficult to analyze the true costs of control measures and to determine the incidence of those costs on product prices, profits, wages, and other inputs.

Most VC plants are open-air, resembling oil refineries. They are located in populated areas in warm states—principally Louisiana, Texas, Kentucky, and California.[94] PVC plants are enclosed, but still emit VC, and they too are located mostly in populated areas. In addition to the above states, New Jersey, Ohio, and Massachusetts are major PVC-producing states.[95]

Only about one-third of total VC production is polymerized at the site at which it is produced. Most VC must be transported between VC and PVC plants, mainly by rail tank car, and also by tank truck and barge. In addition, PVC resin must be transported between the PVC and fabrication plants; this is done primarily by train and truck.[96]

VC and PVC plants are highly mechanized and employ a relatively small number of workers. At any one time, there are only about 1,000 employees in the VC industry, and only about 5,500 in the PVC industry.[97] Taking into account the normal turnover of workers, about 30,000 employees are estimated to have worked in these industries since 1939.[98] Fabrication plants are more labor intensive. The number of fabrication workers is estimated at 350,000.[99] These workers are subject to much lower exposures of VC than the workers in VC and PVC plants, as the exposure of the fabrication workers comes only from escaping VC residual.[100] The size of the group, however, gives rise to fears that even a low incidence of cancers may claim a large number of lives.

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93 Booze-Allen Stacl, Inc., Draft Final Report, Economic Impact Studies of the Effects of Proposed OSHA Standards for Vinyl Chloride, at III-3 (Sept. 11, 1974) [hereinafter cited as Stacl Economic Impact Study].

94 *Id.* at III-2; EPA EIS, *supra* note 85, at 3-31, -39.

95 EPA Standards and Technical Report, *supra* note 90, at 21-23.

96 EPA Task Force Report, *supra* note 85, at 7.

97 Stacl Economic Impact Study, *supra* note 93, at III-4, 8.

98 N. Ashford *et al.*, *supra* note 5, at 36-41, -45 (appendix concerning VC regulation in the workplace). The number of cancers found in these workers, while not large in absolute terms, represents an extremely high incidence in the small population.

99 EPA Task Force Report, *supra* note 85, at 11.

100 OSHA *Permanent Standard for VC*, *supra* note 1, at 55392-93.

The VC and PVC workers are represented primarily by three unions: the United Rubber Workers, the United Steelworkers, and the Oil, Chemical and Atomic Workers. On their own and through the Industrial Union Department of the AFL-CIO, these unions were major participants in setting the VC occupational exposure standard. The fabrication workers are represented by a variety of unions, and some are not unionized at all.

The technological and economic capabilities of the VC, PVC, and fabrication industries to lower the release of VC—in plants, to the surrounding air, and through later-escaping residual in PVC—have been constantly at issue in the regulatory actions described in subsequent sections. The difficulty of predicting the future technological and economic limits to changes in these industries is discussed elsewhere,[101] but here it is useful to give some indication of their clearly demonstrated past and present capabilities.

Without need for any significant technological breakthroughs, in the four years since VC's carcinogenicity became clear, the VC, PVC, and fabrication industries have significantly reduced their releases of VC. In response the regulations or the threat of regulations, the VC and PVC industries have been shown to be able to reduce workplace airborne concentrations of VC from about 250 parts per million (ppm) to about one ppm,[102] to reduce VC emissions to the outside by about 95 percent,[103] and to reduce the VC residual content of food packaging by several orders of magnitude.[104] New plants face no difficulties meeting these lowered levels.[105] Furthermore, there is no sign that the limits of current technologies have been reached and the possibility remains that fundamental technological breakthroughs could occur.

These reductions have been achieved without any significant economic strain on the companies or damage to PVC's market position and future growth prospects. Throughout the 1960s and early 1970s, PVC consumption grew at a staggering rate as prices declined and the number of uses increased.[106] In 1973, the business analysts forecast uninterrupted growth; the major problem experienced at that time was the tight supply of petrochemical raw materials.[107]

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101 See notes 61-72 *supra*.

102 See text accompanying notes 227, 329-331 *infra*.

103 EPA EIS, *supra* note 85, at 1-4; EPA Standards and Technical Report, *supra* note 90, at 111-14.

104 See text accompanying note 535 *infra*.

105 *PVC Rolls Out of Jeopardy, Into Jubilation*, Chemical Week, Sept. 15, 1976, at 14, 16.

106 *See* Stacl Economic Impact Study, *supra* note 93, at III-3, exhibit III-2; EPA EIS, *supra* note 85, at 2-1.

107 *Tight Monomer Supply Plagues PVC Producers*, Chem. & Eng'g News, May 28, 1973, at 6.

In 1974 and 1975, VC and PVC consumption dropped sharply, marking the first break in the trend of phenomenal growth. The slump, however, was not caused by a consumer response to the revelation of VC's carcinogenicity and to the costs of complying with subsequently imposed standards. Rather, the slump had two extrinsic causes: (1) attempts in the early 1970s to pass on to consumers the rising costs of petrochemical feedstocks; and (2) the general economic recession in 1974 and 1975, which was particularly severe in the building construction industry, a major user of plastics.[108] The slump was shared equally by all the major plastics.

With the end of the recession and the revival of the housing industry, plastics generally and PVC in particular returned to the prior trend of profitable growth. Presently, new plants are being built to meet anticipated demand, without any apparent hindrance from current and proposed regulations.[109] Hence, it appears that somewhat greater reductions in VC exposure could be demanded of the industries without rendering either current operations or expansion unprofitable.

As the following survey of VC's toxicity will show, there is no basis for thinking the VC hazard has been eliminated by the measures already taken, or that it will be eliminated by the additional measures conceded by the industries to be both technologically and economically within their reach. Further reductions are justified by the medical evidence. The point at which the industries would be seriously burdened economically is, however, impossible to predict reliably.

2 *Health Risks and Sources of Exposure to Vinyl Chloride*

The toxic effects of VC are now known better than those of nearly any other industrial chemical. VC's carcinogenicity has been well established by human experience, animal experiments, and other laboratory tests. The bitter experience of occupational exposure has confirmed VC's ability to cause cancers and a host of lesser effects in humans. The risks extend beyond the workers; millions of other people are exposed to VC. This subsection surveys the evidence of VC's toxicity and the extent of human exposure to the chemical.

a Acute and chronic human toxicity

Before 1974, when VC had not yet been connected to human cancer, other dangers of the chemical were well known. VC is extremely flammable, and concentrations in air exceeding 40,000 ppm are explosive.[110]

108 Ingalls, *1976 Outlook Brighter in U.S. and European Chemical Industries*, Chem. Engr. News, Apr. 1976, at 14; Greek, *Vinyl Chloride May Face Shortages By 1977*, Chem. & Eng. News, Aug. 11, 1975, at 8.

109 *PVC Rolls Out of Jeopardy, Into Inhibition*, *supra* note 105, at 41.

110 Doty, *Vinyl Chloride: How Many Unknown Problems?*, 11 Toxicology & Env't Health 17, 19 (1975) [hereinafter cited as *VC: How Many Unknown Problems?*].

Several workers have died from inhaling extremely high concentrations.[111] Exposure to concentrations greater than 8,000 ppm VC causes one to become dizzy, drowsy, disoriented, and eventually unconscious.[112] Lower doses inhaled over a period of a work shift make one prone to deep, dreamless sleep.[113]

Before 1974, VC concentrations of 250 to 300 ppm were common for many job categories in the VC and PVC industries.[114] Among workers exposed to these concentrations for periods of months or years a number of effects had been identified. Many workers suffered enlargement and fibrosis of the liver and spleen.[115] Many exhibited Reynaud's syndrome, characterized by circulatory degeneration in the extremities and by a cold feeling or a feeling of pins and needles in the hands and feet.[116] Many developed scleroderma, a skin condition, and acroosteolysis, a rare disease characterized by the shrinking of the last bones in the fingers and toes.[117] Although workers who suffered one of these effects did not always suffer the others, the effects were grouped under the name "vinyl chloride disease."[118]

When VC's capacity to cause cancer in humans became clear in 1974, close medical examination of VC and PVC workers identified numerous acute and chronic effects of VC that previously had gone unnoticed. Impaired arterial circulation and fibrosis of the liver and spleen were found to occur at a microscopic level long before they were clinically observable.[119]

111 Spotas, McMichael, Gamble, & Van Ert, *The Association of Vinyl Chloride Exposures With Morbidity Symptoms*, 36 Am. Indus. Hygiene Ass'n J. 779 (1975).

112 EPA Standards and Technical Report, *supra* note 90, at 85; *VC: How Many Unknown Problems?*, *supra* note 110, at 61.

113 *VC Hearings*, *supra* note 1, at 40 (testimony of Dr. Marcus Key, Director, National Institute for Occupational Safety and Health).

114 Kramer & Mutchler, *The Correlation of Clinical and Environmental Measurements For Workers Exposed to Vinyl Chloride*, 33 Am. Indus. Hygiene Ass'n J. 19 (1972) [hereinafter cited as *Worker Exposure to VC*]; EPA Standards and Technical Report, *supra* note 90, at 91-94.

115 Lilis, Anderson, Nicholson, Daum, Fischbein, & Schkuff, *Prevalence of Disease Among Vinyl Chloride and Polyvinyl Chloride Workers*, 246 Annals N.Y. Acad. Sci. 22 (1975) [hereinafter cited as *Prevalence of Disease*]; Lange, Infec. Stein, & Veltman, *Further Results on Polyvinyl Chloride Production Workers*, 246 Annals N.Y. Acad. Sci. 48 (1975) [hereinafter cited as *Further Results*].

116 *Prevalence of Disease*, *supra* note 115, at 22; *Further Results*, *supra* note 115, at 48.

117 *Prevalence of Disease*, *supra* note 115, at 22; *Further Results*, *supra* note 115, at 48; Suhube, *Bone Lesions Among Polyvinyl Chloride Production Workers in Japan*, 246 Annals N.Y. Acad. Sci. 78 (1975); *VC: How Many Unknown Problems?*, *supra* note 110, at 62-63.

118 Veltman, Lange, Infec, Stein, & Rochner, *Clinical Manifestations and Course of Vinyl Chloride Disease*, 246 Annals N.Y. Acad. Sci. 8 (1975).

119 Marsch, Johnson, Whetstone, & DeRoy, *Capillary Abnormalities in Polyvinyl Chloride Production Workers Examination By In Vivo Microscopy*, 236 J. Am. Med. Ass'n 1968 (1976); Popper & Thomas, *Alterations of Liver and Spleen Among Workers Exposed to Vinyl Chloride*, 246 Annals N.Y. Acad. Sci. 172 (1975); Thomas & Popper, *Pathology of Angiosarcoma of the Liver Among Vinyl Chloride-Polyvinyl Chloride Workers*, 246 Annals

Microscopic examination revealed other changes in liver cells.[120] Some workers displayed a wide variety of abnormal liver function and blood tests.[121] Some suffered impairment of lung function.[122]

The medical researchers had hoped that these observable effects could be used to indicate who is at increased risk of developing cancer and how VC produces its carcinogenic effect. To their disappointment, however, none of these effects correlated well enough with observed cases of liver angiosarcoma or of other cancers to indicate clearly which workers are at increased risk, nor has the knowledge of these effects enabled researchers to explain how VC causes cancer.[123]

b. Human cancers

Throughout the spring of 1974, after the deaths of the four Goodrich workers became known, other companies reported additional angiosarcoma deaths among VC and PVC workers.[124] The toll has risen steadily since then. Thirteen cases of angiosarcoma of the liver had been counted among American workers by July 1974,[125] and a total of 25 cases were known

N.Y. ACAD. SCI. 268 (1975). Gedigk, Muller, & Bechtelsheimer, *Morphology of Liver Damage Among Polyvinyl Chloride Production Workers: A Report on 31 Cases*, 246 ANNALS N.Y. ACAD. SCI. 278 (1975).

120. *See* sources cited in note 119 *supra*.

121. Blood platelet count has been down in some, but not all, victims of liver angiosarcoma. Liver function tests show a wide variety of abnormalities. *Prevalence of Disease*, *supra* note 115; *Further Results*, *supra* note 115; Veltman, Lange, Jobe, Stein, & Bachner, *supra* note 118; Marsteller, Leibsch, Shiller, & Gedigk, *Unusual Splenomegalic Liver Disease as Evidenced by Peritoneoscopy and Guided Liver Biopsy Among Polyvinyl Chloride Production Workers*, 246 ANNALS N.Y. ACAD. SCI. 95 (1975).

122. Gamble, Liu, McMichael, & Wasweiler, *Effects of Occupational and Nonoccupational Factors on the Respiratory System of Vinyl Chloride and Other Workers*, 18 J. OCCUPATIONAL MED. 659 (1976).

123. In promulgating the permanent standard for workplaces, OSHA noted the failure to find in blood tests, liver function tests, and certain other examinations a reliable indicator of increased cancer risk. *OSHA Permanent Standard For VC*, *supra* note 1, at 35,895. Since the fall of 1974, when OSHA's observation was made, there has been little change in the state of knowledge of VC's carcinogenesis. Some hope of identifying VC-induced changes at an early stage is promised by two recent techniques. The first is ultrasonography, a technique for "taking a picture" of internal organs such as the liver and spleen without surgery through a sort of "sonar." This technique can reveal lesions and other gross changes in these organs. Taylor, Barrett, Williams, Smith, & Buck, *Preliminary Results of Grey-scale Ultrasonography in the Detection of Vinyl Chloride Related Liver and Spleen Disease*, 69 PROC. ROY. SOC'Y MED. 392 (1976). Another technique detects microscopic changes in the circulatory system in the fingers. It was reasoned that the Reynaud's syndrome and acroosteolysis would be preceded by subclinical changes, which, if they could be found, would be an indication of increased risk. Marsh, Johnson, Whetstone, LeRoy, *supra* note 119. Neither of these techniques, however, offers any assurance of detecting VC-induced changes before the changes at the cellular level which eventually lead to cancer have already occurred.

124. Occupational Safety and Health Administration, *Vinyl Chloride: Proposed Standard*, 39 Fed. Reg. 16,896 (1974) [hereinafter cited as *OSHA Proposed Standard for VC*].

125. *OSHA Permanent Standard for VC*, *supra* note 1, at 35,891.

worldwide one month later.[126] Although the majority of cases involved workers at PVC plants, other workers were also affected.[127] There were 38 cases worldwide by June 1975,[128] at least 51 cases by December 1976,[129] and at least 68 cases by the spring of 1978.[130]

Although in absolute terms these numbers are not large, the rate of liver angiosarcoma in various groups of PVC workers studied ranges from 400 to 3,000 times the expected incidence in the general population.[131] Since the latency period—the time between initial exposure to VC and the clinical appearance of liver cancer—has been averaging about 20 years, more cases can be expected as the result of high exposures in the 1950s, 1960s, and early 1970s.[132]

Many of the employees who have died of liver angiosarcoma worked as cleaners of the polymerization reactor (the chamber in which VC is converted to PVC) or worked in areas with similarly high VC exposures.[133] These workers probably were the most heavily exposed, in terms of both momentary peaks and sustained averages. The precise levels are not known but are estimated to have included peak exposures of several thousand ppm and an average of 250 to 300 ppm.[134] Other liver angiosarcoma victims presumably had lower, less sustained exposures to VC.[135]

126. *VC Hearings*, *supra* note 1, at 60-62 (statement of Dr. Joseph K. Wagoner).

127. Those affected included a worker at a VC plant, a worker who filled pesticide cans with VC propellant, and an accountant and a worker at PVC cloth fabricating plants. *Id.* These last two are assumed to have had very low exposures.

128. *Id.* at 60; EPA SUBSTITUTE AND TECHNICAL REPORT, *supra* note 90, at 72, 82.

129. Reported Cases of Angiosarcoma of the Liver Among Vinyl Chloride Polymerization Workers (Dec. 6, 1976) (typewritten tabular data enclosed with letter to the author from Rose Kaminski, Statistician (Health), Illness Effects Section, Division of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health (Dec. 6, 1976)).

130. Personal communication with Rose M. Kaminski, Statistician (Health), Illness Effects Section, Division of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, Apr. 18, 1978.

131. The lower figure is from Heath, Falk, & Creech, *Characteristics of Cases of Angiosarcoma of the Liver Among Vinyl Chloride Workers in the United States*, 246 ANNALS N.Y. ACAD. SCI. 231, 233 (1975). The higher figure is from EPA, EPA, *National Emission Standards for Hazardous Air Pollutants, Proposed Standard for Vinyl Chloride*, 40 Fed. Reg. 59,532 (1975) (hereinafter cited as *EPA Proposed Standard for VC*). These figures are the result of comparing the rates of angiosarcoma of the liver in workers with the rates in the general population, assuming that the general population cases are not caused by VC. But if, as the remainder of this section suggests, some of the cases in the general population may be caused by nonoccupational VC exposure, then these figures understate the potency of the chemical.

132. *VC Hearings*, *supra* note 1, at 25 (statement of Dr. Irving J. Selikoff); Heath, Falk, & Creech, *supra* note 131, at 241.

133. Nicholson, Hammond, Seidman, & Selikoff, *Mortality Experience of a Cohort of Vinyl Chloride Polyvinyl Chloride Workers*, 246 ANNALS N.Y. ACAD. SCI. 225, 226-27 (1975). *See also* Fox & Collier, *Mortality Experience of Workers Exposed to Vinyl Chloride Monomer in the Manufacture of Polyvinyl Chloride in Great Britain*, 34 BRIT. J. INDUS. MED. 1 (1977).

134. *Worker Exposure to VC*, *supra* note 114.

135. EPA SUBSTITUTE AND TECHNICAL REPORT, *supra* note 90, at 72, 82.

The long term risks of VC exposure may not be limited to liver angiosarcoma. One study of PVC workers identified a statistically significant excess incidence of cancers of the brain and the respiratory system.[136] A study of the causes of death among fabrication workers identified no liver angiosarcomas, but it did suggest an increased incidence of cancers of the digestive system among both men and women and the breast and the urinary system in women.[137] Evidence of other effects has appeared in populations other than workers. One study has shown a statistically significant excess of birth defects and central nervous system tumors among the children of families in three Ohio communities that have hosted PVC plants for as long as 28 years.[138]

c. Animal bioassays and other tests for carcinogenicity

VC's carcinogenicity has been confirmed by the results of experiments on animals and by other laboratory tests. VC has been shown to cause cancer in animals both when inhaled and when ingested. The first hint that the chemical causes cancer came from the publication in 1971 of the results of Italian experiments sponsored by the European and American VC and PVC producers. In these tests rats inhaling high concentrations of VC (30,000 ppm) developed cancerous tumors of the skin, lung, and bone.[139] Further tests sponsored by the producers were concluded and reported to the Occupational Safety and Health Administration (OSHA) in early 1974. As shall be seen in the case study of OSHA's standard setting, the data arrived at a crucial time. On February 15, at OSHA's fact-finding hearing on VC, the Italian researchers reported their then-unpublished findings that VC had induced angiosarcoma of the liver in rats inhaling concentrations as low as 250 ppm.[140] On April 15, OSHA received reports from tests conducted in

136 Tabershaw & Gaffey, *Mortality Study of Workers in the Manufacture of Vinyl Chloride and its Polymers*, 16 J. Occupational Med. 509 (1974). *See also* Monson, Peters, & Johnson, *Proportional Mortality Among Vinyl Chloride Workers*, Ind. Hygiene, Aug. 17, 1974, at 397.

137 Chairse, Nichols, & Wong, *Mortality Among Employees of PVC Fabricators*, 19 J. Occupational Med. 623, 628 (1977) [hereinafter cited as *Mortality Among Fabrication Employees*]. The authors noted a number of flaws in the design and data for their study that precluded drawing firm conclusions from it. In particular, it would be useful to follow the population of fabrication workers for some years into the future, as a sufficient latency period may not yet have elapsed for some effects to manifest themselves.

138 Infante, *Oncogenic and Mutagenic Risk in Communities with Polyvinyl Chloride Production Facilities*, 271 Annals N.Y. Acad. Sci. 49, 58 (1976). *Cf.* Infante, McMichael, Wagoner, Wanweiler, & Falk, *Genetic Risks of Vinyl Chloride*, The Lancet, Apr. 3, 1976, at 734 (finding increased fetal loss among wives of workers exposed to VC).

139 Viola, Bigotti, & Caputo, *Oncogenic Response of Rat Skin, Lungs, and Bones to Vinyl Chloride*, 31 Cancer Research 516 (1971) [hereinafter cited as *Oncogenic Response of Rats to VC*].

140 Occupational Safety and Health Administration, *Emergency Temporary Standard for Exposure to Vinyl Chloride*, 39 Fed. Reg. 12,342 (1974) [hereinafter cited as *OSHA Emergency Temporary Standard for VC*].

Illinois of similar results in mice at the lowest level of exposure then being tested, 50 ppm.[141]

Animal test results stood at this point until early 1975. It was reported then that *ingestion* by rats of as little as approximately 17 milligrams per kilogram of body weight produces liver angiosarcoma and other cancers.[142] Most recently, in September 1976, the results of another round of inhalation experiments demonstrated that VC causes liver angiosarcoma in rats at 25 ppm, and that it causes mammary tumors at one ppm, the lowest concentration yet tested.[143]

The animal experiments also support suspicions that VC causes human cancers other than angiosarcoma of the liver. The experiments have shown increased cancer incidence at many sites other than the liver, including the lungs, spleen, brain, and, as already noted, breast.[144] Experiments demonstrating the induction of cancer in two species other than rats—mice and hamsters—further confirm that VC is carcinogenic.[145] In the animal experiments the subjects were exposed to constant, prolonged doses of VC. The need was noted in mid-1974 for studies of the effects of single and sporadic doses, typical of many humans' exposure.[146] Such a study is now nearing completion, but the results are not yet available.[147]

d. Additional humans at risk

In addition to several hundred thousand workers in VC and PVC production and in PVC fabrication, millions of American have been, and continue to be, exposed to VC. First, about 4.6 million people live within

141 *OSHA Proposed Standard for VC*, *supra* note 124, at 16,898. The results of the Italian tests, also showing the induction of liver angiosarcoma at 50 ppm, were published in early 1975. Maltoni & Telemine, *Carcinogenicity Bioassays of Vinyl Chloride: Current Results*, 246 Annals N.Y. Acad. Sci. 195 (1975).

142 Maltoni, Cilberti, Gianni, & Chieco, *Gli Effetti Oncogeni Del Cloruro Di Vinile Somministrato Per Via Orale Nel Ratto (Oncogenic Effects of VC Administered Orally to Rats: Preliminary Report)*, Gli Ospedali Vita, Dec., 1975 (unpaginated reprint on file in offices of the *Ecology Law Quarterly*).

143 Memorandum from Cesare Maltoni to the Members of the European Cooperative Group for the Experimental Bio assays on Vinyl Chloride Carcinogenicity (undated) [hereinafter cited as Maltoni Memorandum].

144 See sources cited in notes 140-143 *supra*.

145 EPA Scientific and Technical Review, *supra* note 90, at 56. In addition, metabolites of VC have been shown to mutate bacteria and yeasts in the "quick tests" for mutagenicity. *See, e.g.*, Lupiens, Basile, Bassucelli, Batsch, Bonatetti, Cannellini, Corsi, Freese, Nieti, Leporini, Rovellini, & Russo, *Induction of Gene Mutations and Gene Conversions by Vinyl Chloride Metabolites in Yeast*, 37 Cancer Research 253 (1977), and sources cited therein. Mutagenicity as revealed in these tests correlates well with carcinogenicity. *See* McCann & Ames, *supra* note 57; *From Microbes to Men*, *supra* note 57.

146 *VC Hearings*, *supra* note 1, at 80-81 (statement of Dr. Theodore R. Torkelson).

147 Telephone interview with Dr. Joseph McLaughlin, Director, Division of Toxicology and Medicine, Consumer Product Safety Commission, Feb. 27, 1978.

five miles of a VC or PVC plant.[148] In 1974, about 220 million pounds of VC escaped into the air surrounding such plants.[149] The plant neighbors appear to have been exposed to more than one ppm less than 10 percent of the time.[150] One air sample, however, measured 33 ppm near a plant,[151] suggesting that there may be short term, localized peak exposures.[152] In addition, VC is found in the sludge waste and water effluent of these plants. VC has been found in sludge at levels as high as 3,000 ppm,[153] and in water effluent as high as 20 ppm.[154] Most of this VC escapes into the air surrounding the plants;[155] some, however, makes its way into drinking water.[156] These VC emissions to the ambient air have been implicated as the cause of increased rates of cancer and birth defects in the surrounding communities.[157]

Another large group of persons is exposed to VC released in transportation. Only about one-third of VC production is polymerized at the site where it is produced. The rest must be shipped between VC and PVC factories under pressure as a liquified gas. About 95 percent of this is shipped in rail tank cars, and the rest in tank trucks, tank vessels, and barges.[158] These tanks may leak, puncture, or explode, sometimes in heavily populated areas.[159] Between 1971 and 1974, there were at least 24 accidental releases of VC from rail tank cars alone.[160] As VC diffuses from the site of a spill or an accident, transportation workers, nearby residents, travellers, and other bystanders can receive short-term exposures to VC concentrations ranging from a few parts per billion (ppb) to thousands of ppm. Transportation workers and emergency personnel such as firemen and police officers may

148. EPA, *National Emission Standard for Hazardous Air Pollutants, Standard for Vinyl Chloride*, 41 Fed. Reg. 46,560 (1976) [hereinafter cited as *EPA Standard for VC*].
149. EPA Scientific and Technical Report, *supra* note 90, at 18.
150. *Id.*
151. *Id.*
152. *See id.*
153. EPA Task Force Report, *supra* note 85, at 2.
154. *Id.*
155. *Id.*, apps., at 31-32.
156. See note 89 *supra*.
157. See sources cited in note 138 *supra*.
158. EPA Task Force Report, *supra* note 85, at 7.
159. See note 160 *infra*.
160. Most of these accidents occurred in Texas and Louisiana, the states with the highest concentrations of VC and PVC facilities. There were several other accidents involving no release. Data on accidents and releases is derived from a computer file of the Department of Transportation which records incidents of hazardous materials leakage or accidents during this period, and from the appendix to H.R. Rep. No. 1083, 93d Cong., 2d Sess. 30-34 (1974). One accident, in Fort Wayne, Indiana, necessitated the evacuation of 4,500 people. *Id.* at 32. When a rail tank car ruptures, as much as 30,000 gallons of VC escapes and returns to its gaseous state. The figure for the maximum tank size comes from an interview with Mary Williams, Chemical Engineer, Department of Transportation, Office of Hazardous Materials Operations, Oct. 28, 1976.

be subject to repeated exposures and, if accidents occur in the same places, so may residents and bystanders. These exposures are less sustained than those suffered by VC and PVC workers and plant neighbors, but peak concentrations may reach those experienced by PVC polymerization reactor cleaners, the most heavily exposed occupational group.

Third, people are exposed to VC through consumer products, food, and drinking water. Until late 1973 or early 1974, VC was used as an aerosol propellant in drug, cosmetic, pesticide, and other consumer products.[161] The user of a VC-propelled aerosol product such as a hair spray or a pesticide in a small, enclosed space such as a bathroom may have been exposed to short term concentrations approaching 400 ppm, and persons may have had an average exposure from all VC-propelled products in their homes equivalent to an average exposure of about 16 ppm in the factories.[162] VC has been detected in the air of rooms freshly painted with certain latex paints, but no VC emissions have been detected in a limited sampling of other new PVC products or from automobile interiors.[163] VC leaches into food and beverages from the more than 300 million pounds of PVC packaging and other PVC food-contact materials used annually.[164] VC also enters drinking water from raw water supplies and by leaching from increasingly common PVC pipe.[165] One study estimates average American daily human intake of VC through food, water, and air at 34 micrograms.[166]

The hazardousness of non-occupational exposures to VC is even less well understood than the risk to the workers. The danger from inhaling extremely low concentrations of VC, or from single or sporadic exposures to high VC concentrations, is unknown. Similarly, the relative risks of ingesting and inhaling VC are unknown.[167] Thus the urgency of reducing or eliminating these sources of exposure is impossible to assess.

161. See text accompanying notes 551-556 *infra*.
162. Gay, Lounsman, Bridburd, & Moran, *Measurement of Vinyl Chloride from Aerosol Sprays*, 246 Annals N.Y. Acad. Sci. 286, 294-95 (1975).
163. Environmental Protection Agency, Office of Toxic Substances, Sampling and Analysis of Selected Toxic Substances: Task III—Vinyl Chloride, Secondary Sources, table 6, at 21 (Apr. 1976).
164. Food and Drug Administration, *Vinyl Chloride Polymers in Contact with Food, Notice of Proposed Rulemaking*, 40 Fed. Reg. 40,529, 40,530 (1975) [hereinafter cited as *FDA Proposed Rules for Food-Contact PVC*].
165. EPA Scientific and Technical Report, *supra* note 90, at 38-39; EPA Preliminary Report on Drinking Water Carcinogens, *supra* note 89, at 40-44; EPA Task Force Report, *supra* note 85, at 7, 19.
166. EPA Scientific and Technical Report, *supra* note 90, at 43.
167. *See* Withey & Collins, *A Statistical Assessment of the Quantitative Uptake of Vinyl Chloride Monomer from Aqueous Solution*, 2 J. Toxicology & Env't Health 311 (1976); Withey, *The Pharmacodynamics and Uptake of Vinyl Chloride Monomer Administered by Various Routes to Rats*, 1 J. Toxicology & Env't Health 381 (1976). In the former article, the authors estimate that for rats, 0.9 micrograms of VC in the daily intake of water is approximately equal to inhaling 6 ppm for eight hours. They caution against any direct extrapolation to human equivalences. Withey & Collins, *supra*, at 319-20.

South Coast
AIR QUALITY MANAGEMENT DISTRICT
9150 FLAIR DRIVE, EL MONTE, CA 91731 (818) 572-6200

April 22, 1985

Mr. William V. Loscutoff, Chief
Toxic Pollutants Branch
California Air Resources Board
P.O. Box 2815
Sacramento, California 95812

Dear Mr. ~~Loscutoff~~ Bill:

Vinyl Chloride

In response to Mr. Venturini's request for information on the health effects of vinyl chloride, we are submitting several references which could be of use in your toxic air contaminant program. These references were not included in your bibliography dated March 1, 1985. Also, information regarding possible biological production of vinyl chloride may be obtained from Dr. Freeman Allen of Pomona College.

We would like to continue to receive information inquiries for other candidate compounds and to be kept informed of your program's progress.

Very truly yours,

Jo Anne Aplet
Director of Planning

JAA:cas

Enclosure

PACIFIC GAS AND ELECTRIC COMPANY

PG&E — 77 BEALE STREET • SAN FRANCISCO, CALIFORNIA 94106 • (415) 781-4211 • TWX 910-372-6587

May 7, 1985

Mr. William V. Locustoff, Chief
Toxic Pollutants Branch
Re: Vinyl Chloride.
California Air Resources Board
P.O. Box 2815
Sacramento, California 95812

Dear Mr. Loscutoff:

Request for Public Health
Information Regarding Vinyl Chloride

Pacific Gas and Electric Company received your April 4, 1985 request for additional public health information regarding Vinyl Chloride. We have reviewed the bibliography attached to your request and concluded that we are unaware of any additional information which would be of use to you.

Sincerely,

J. F. McKenzie

Chemicals PPG

Chlor-Alkali Business Unit
PPG Chemicals
One PPG Place
Pittsburgh, Pennsylvania 15272

May 13, 1985

Mr. William V. Loscutoff, Chief
Toxic Pollutants Branch
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Re: Vinyl Chloride

Dear Sirs:

Relative to your request for health information on vinyl chloride, we have no information which was not covered in the MEDLINE and TOXLINE information services.

We thank you for asking for our input.

Sincerely yours,

Clete M. Smith

Clete M. Smith
Technical Service

CMS/rs

Additional References on Vinyl Chloride Health Effects

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AIR PRODUCTS

16 April 1985

W. V. Loscutoff
Chief, Toxics Pollutant Branch
CARB
Box 2815
Sacramento, CA 95812

Re: Vinyl Chloride

Dear Mr. Loscutoff:

We are happy to provide some information relative to vinyl chloride in response to the April request of P. D. Venturini. This includes:

1. A paper by me given at the APCA, New Orleans meeting.

2. A paper presented at a CMA seminar in Washington, 9 December 1983.

3. An unpublished review by me on the safety and health aspects of vinyl chloride, which contains several references not in the bibliography with the Venturini letter.

4. A report from Br. J. Surg. 71 322 (1984) of an apparently successful liver resection on an ASL patient.

5. An article from EST 19 277 (1985) on biodegradation of TCE to VC. Note especially reference 10, Parsons, et. al., for corroborating evidence. You may want to get the Dade County report "An Investigation into the Source of Vinyl Chloride Detected at the Preston and Hialiah Water Treatment Plants" by J. C. Balter, 1983, for more details.

6. A summary of a report on a 1984 bioassay by CIVO.

I hope that these are useful to you in your evaluation of this substance.

Very truly yours,

John T. Barr, Manager
Regulatory Response

JTB:csb

Air Products

ASL
VC

3650

Date 21 August 1984

INTEROFFICE
MEMORANDUM

Subject Surgical Removal of Angiosarcoma

To L. B. Tepper — Corporate Medical Department
(Location, Organization, or Department)

From J. T. Barr — Regulatory Response
(Location, Organization, or Department)

cc: G. Bays
H. L. Watson

The attached article is a report of an apparently successful surgical removal of an angiosarcoma from the liver of a PVC worker. It appeared at Br. J. Surg. 71 322 (1984).

J. T. Barr

JTB:csb

Vinylchloride induced hepatic angiosarcoma

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Correspondence to: Dr Y.A. Louagie, 20 Avenue d'Huart (bte 3), 1150 Brussels, Belgium

The relationship between vinylchloride exposure and human angiosarcoma of the liver (ASL) received attention in 1973 when a case of this rare tumour was diagnosed at autopsy[1].

Case report

A 39-year old man was first seen in July 1979 with pain in the right upper quadrant. The liver was palpated at the right costal margin. Oral cholecystography and barium swallow were normal and liver function tests were in normal limits. From 1965 to 1970 he had cleaned reactors used for the polymerization of the vinylchloride monomer in PVC and was thus exposed to high amounts.

He was admitted 3 months later with persisting right upper quadrant pain, loss of appetite and fatigue. The liver edge was by then hard and 4 cm below the costal margin. The ESR was accelerated (80 mm/h) and alkaline phosphatases and GGTP were elevated. Carcino-embryonic antigen (CEA) and α fetoglobulin were normal. Ultrasound showed a large dense tumour of the right hepatic lobe with areas of necrosis. The ^{99m}Tc hepatic scan confirmed the presence of an ill-defined filling defect in the inferior part of the right lobe with two small defects at the level of the hilum.

The liver computerized tomography confirmed the integrity of the left lobe.

A selective angiography of the celiac artery revealed a hypervascularized tumour of the right hepatic lobe (*Figure 1*).

At a right thoracophrenolaparotomy the tumour was found to be confined to the right lobe. An extended right lobectomy was then performed. The postoperative recovery was uneventful and the patient was sent home with a monthly administration of Vincristine (1 mg IV) and Adriamycin (150 mg/m² IV) which was discontinued in June 1981.

Repeated controls up to September 1981 by liver scan and computerized tomography remained normal. The patient is still in good health 38 months after the resection.

Pathology

The resected specimen was 2050 g. On macroscopical examination, the main tumour (13·5 × 8 cm) was yellowish and spongy and contained cystic and haemorrhagic zones. A second smaller haemorrhagic mass was found at the inferior aspect of the right lobe surrounded by numerous purple masses.

Macroscopically, the main tumour showed large areas of necrosis and haemorrhagic pseudocystic spaces (*Figure 2*). These spaces were surrounded by areas of dense vascular proliferation. The sinusoids were lined with variably sized irregular sarcomatous cells with hyperchromatic nuclei. Elsewhere, blood-filled spaces were surrounded by sarcomatous cells. The sarcoma cells encompassed adjacent liver cells and bile ductules and infiltrated the parenchyma. The pathological diagnosis of multicentric angiosarcoma was made. The rest of the liver was normal except for some moderately enlarged portal tracts. Progressive fibrosis separated hepatocytes at the margins of the portal tracts from adjacent hepatic

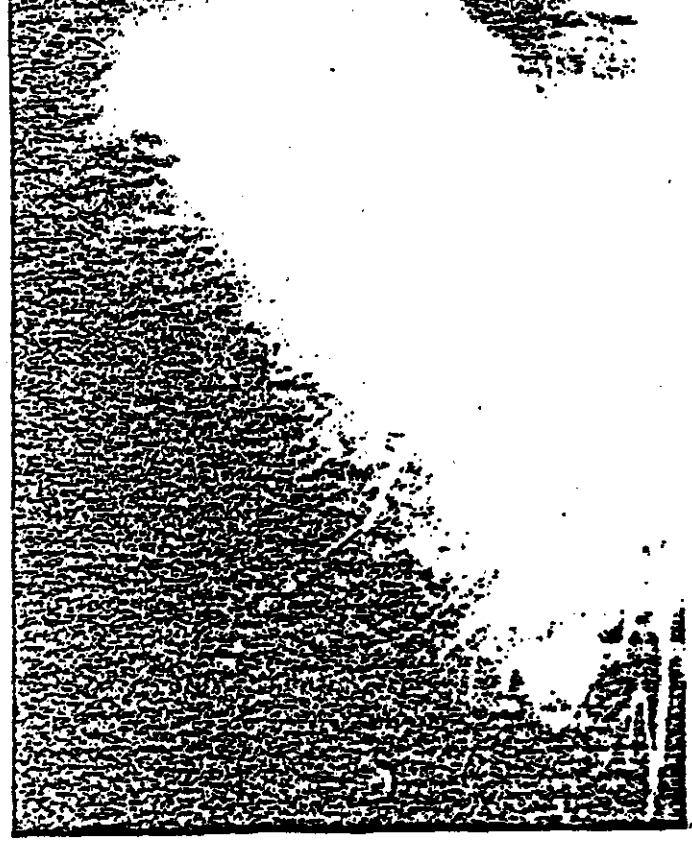

Figure 1 *Selective angiography of the coeliac artery. The hypervascularized tumour is supplied by an anterior branch (arrow) of the right hepatic artery*

Figure 2 *Photomicrography of the main tumour showing blood filled spaces surrounded by sarcomatous cells. Haematoxylin eosin. × 100*

cord cells. Anisocaryosis and anisocytosis were frequent. A cross-section biopsy of the left lobe showed normal tissue with slight hepatocytic anisocaryosis. The lymph nodes taken from the liver hilum were hyperplastic. The main features of this tumour were its multicentricity and the presence of mild fibrosis.

Discussion

The occurrence of liver angiosarcoma in vinylchloride polymerization workers was reported in 1974[1,2]. Prolonged exposure and long interval from initial exposure is required before liver disease becomes apparent. The average interval is 12 years (range 6–29)[3]. Our patient was exposed for 5 years and became symptomatic 14 years later.

It is a rapidly progressing fatal disease, especially in adults. The clinical features include rapid liver enlargement with haemorrhagic ascites, fast deterioration and cachexia usually with death within 6 months.

The treatment is disappointing and chemotherapy and radiation of palliative value only. If the diagnosis is made early, the disease is localized and there is no associated liver fibrosis or portal hypertension, resective operation might prove to be curative. However, there are few reported cases of successful operative removal of hepatic angiosarcoma and the longest survival has been 16 months[4].

In our case the tumour was confined to the right lobe and there was no sign of the extensive fibrosis. So far the patient is apparently free of disease after 38 months. This is, to our knowledge, the longest published survival.

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Paper accepted 27 July 1983

morphology, there was no evidence that the surface nodules were composed of any special, unique element. These particles from these particular collections seem to be quite similar to the micrometer size particles emitted in the ash (2). It is not clear, therefore, why the collected ash shows a bimodal distribution of micrometer size particles centered around 5 μm and submicrometer size particles centered around 0.5 μm. X-ray photoelectron spectroscopy (XPS) and depth profile XPS have been applied to these samples to determine surface composition. Results of these analyses will be presented in the near future.

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TCE Biodeg JC.

Anaerobic Degradation of Trichloroethylene in Soil

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■ When trichloroethylene (TCE) isotopically labeled with one ^{13}C atom is used and gas chromatography/mass spectrometry is employed to monitor the production of 1,2-dichloroethylene-$^{13}C_1$ (DCE), it has been demonstrated that reductive dechlorination of TCE takes place in the soil. Microbial involvement in this process is indicated since unsterilized soil samples yielded up to 78 ppb of labeled DCE while sterilized soil samples produced none. Isomer specificity was also found; only 1,2-DCE was produced—no 1,1-DCE was observed.

Introduction

Since trichloroethylene (TCE) is a major industrial solvent (234 000 metric tons produced annually, worldwide (*1*)) used for degreasing and cleaning metal parts and electronic components, it is perhaps not surprising that TCE has found its way into the environment. In fact, TCE appears to be widely distributed in the aquatic environment (*1*).

However, the environmental fate of TCE has not been well documented, and considerable controversy still exists concerning its behavior in environmental matrices. Early literature references have concluded that C_1 and C_2 halogenated hydrocarbons are not metabolized by microorganisms (*2*, *3*). More recent studies, however, are split on the issue of whether TCE is biodegraded (*4*–*7*), with one research group reporting both "no appreciable anaerobic degradation" and 40% degradation of TCE in similar methanogenic cultures (*8*, *9*).

In a very recent publication, Parsons et al. have demonstrated that tetrachloroethylene (herein referred to as perchloroethylene, PCE) is reductively dechlorinated to TCE, dichloroethylene (DCE), and vinyl chloride in Florida muck/surface water microcosms (*10*). Whether TCE, which was present as a 1.6% impurity in the PCE study, was similarly biotransformed was not directly investigated but was implied by the results for PCE (*10*).

Therefore, in order to determine whether TCE itself undergoes biodegradation, we have undertaken a study using TCE with single atom ^{13}C isotopic labeling, soil from a TCE spill site in Des Moines (*11*), and very sensitive gas chromatography/mass spectrometry (GC/MS) analytical techniques. Since DCE-$^{13}C_1$ could only arise via a soil or soil–microbe-induced reductive dechlorination of TCE-$^{13}C_1$, this experimental method should provide concrete evidence in support of such a pathway. The results of our investigation of this problem are reported herein.

Experimental Section

Materials. Since any microbes present had probably adapted to TCE, soil samples were collected at the Des Moines site, at depths of 1–2 ("A" samples), 6–8 ("B" samples), and 15–17 ft ("C" samples), by using an 18 in. long by 2 in. o.d. split barrel sampler (*11*). These soil samples were analyzed by GC/MS for the presence of TCE and DCE. In spite of the TCE sludge application having been discontinued in 1979 (*11*), all soil samples contained 6 ppb of "native" (unlabeled) TCE. No DCE's were found in any soil sample. (An analysis of the Des Moines TCE sludge itself by this laboratory and by an independent testing laboratory showed very high levels of TCE (3000 ppm), but no DCE was detected.)

TCE-$^{13}C_1$ was purchased from Merck Sharp & Dohme Isotopes. Single ^{13}C labeling was used to produce molecular and fragment ion peaks which did not have the same m/z values as the $^{35}Cl/^{37}Cl$ natural isotopic mixtures. GC/MS analysis showed the TCE-^{13}C [illegible] ically pure and [illegible]

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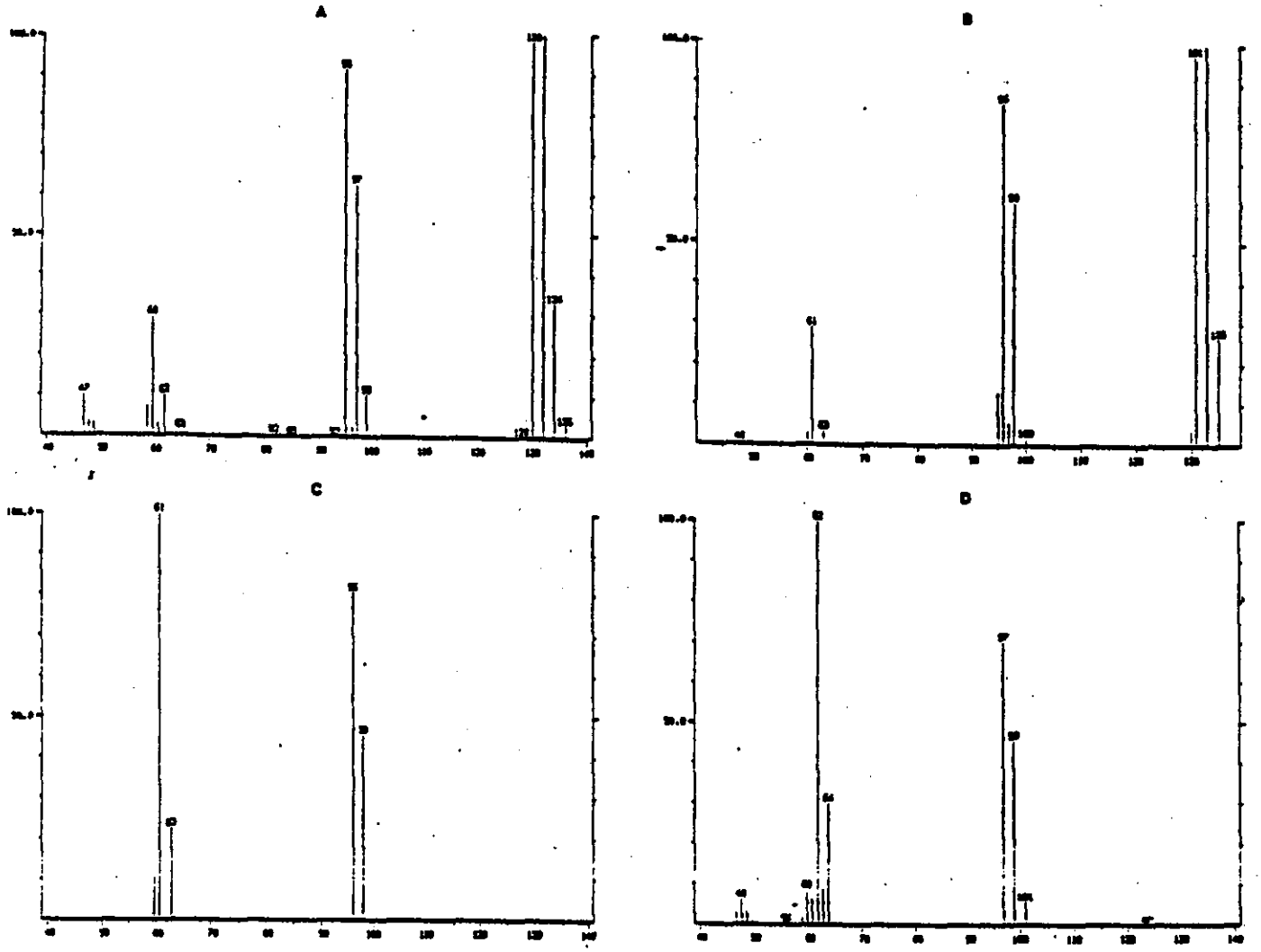

Figure 1. Representative mass spectra of trichloroethylene and 1,2-dichloroethylene: (A) trichloroethylene; (B) trichloroethylene-$^{13}C_1$; (C) 1,2-dichloroethylene; (D) 1,2-dichloroethylene-$^{13}C_1$.

contamination. Volatile organic standards were purchased from Supelco, Inc., and were diluted appropriately with methanol to contain 200 ppb of TCE and DCE. Soybean meal was obtained commercially.

Methods. Five grams of soil from each depth was placed in 5-mL amber vials which had been baked at 150 °C for 3 h to remove any adhering volatile organic compounds. One gram of soybean meal was added to each vial to ensure anaerobic conditions, and the vials were then filled with "organic-free" distilled water (which had been purged with nitrogen). (Organic-free water is distilled, passed through a carbon column, and checked for organics by using GC/MS methods.) The vials were sealed with Teflon septa. Samples to be sterilized were placed in an autoclave for 30 min at 15 psi. Each vial was then injected with 10 μg of TCE-$^{13}C_1$ (2000 ppb, or μg/kg). Duplicates of each sample were prepared. The sealed vials were transferred into CO_2/H_2 Anaerobic-Paks (BBL, Division of Bioquest), which were then placed in an incubator at 23 °C. As much as possible, the samples were kept in the dark to avoid photolytic degradation of the TCE. Subsets of the vials were removed for analyses at 6, 17, and 41 weeks.

Control and method blank samples were prepared as follows: (1) Vials containing only organic-free water, both with and without the TCE-$^{13}C_1$ spike, were prepared to monitor volatilization losses and to check for cross-contamination throughout the procedure. (2) Vials containing only soybean meal (both sterilized and unsterilized) and water were prepared to test for any possible conversion of TCE to DCE by the soybean meal itself or any "foreign" microbes and to monitor adsorption of the TCE onto this organic matter. (3) For the 6-week samples only, vials containing soils A–C, soybean meal, and water without the TCE-$^{13}C_1$ spike were prepared as method blanks.

For the analyses, the contents of each vial were transferred to a 25-mL vial by using organic-free water to eliminate headspace again, sealed with a Teflon septum, mixed, and allowed to settle. Five milliliters of the supernatant liquid was then removed for volatile organics analysis using a Finnigan Model OWA GC/MS and standard purge and trap methodology (*12*, *13*). Detection limits for TCE and DCE by this method are estimated to be 1 ppb.

Results and Discussion

All compounds involved in this study were identified by their characteristic GC elution times and mass spectra. (Figure 1 shows the observed mass spectra of labeled and unlabeled TCE and DCE.) Both qualitative and quantitative identifications were effected from several selected ion mass chromatograms for each substance. For example, the ions at the listed m/z values were used for the analyses of the following compounds: TCE (m/z (95, 97, 130, 132, 134); TCE-$^{13}C_1$ (m/z 96, 98, 131, 133, 135); DCE (m/z 61, 63, 97, 99); DCE-$^{13}C_1$ (m/z 62, 64, 98, 100). No confusion resulted from peaks having the same m/z values for these substances since each compound (exclusive of its isotopomer) eluted at a different time. For quantitation pur-

Table I. Amounts (μg/kg) of 1,2-Dichloroethylene-$^{13}C_1$ Produced by Degradation of Trichloroethylene-$^{13}C_1$ in Unsterilized Soils

time, weeks	soil A[a]	soil B[a]	soil C[a]
6	8	7	11
17	28	31	8
41	78[b]	27	25

[a] See text for soil depth designations. Results are averages for duplicate samples; ranges were ±50%. [b] No duplicate value was obtained.

for the ^{12}C and ^{13}C compounds were identical. This assumption appears to be valid since we observed natural abundance ^{13}C peaks in the unlabeled TCE and DCE mass spectra having 2% of the intensity of the corresponding ^{12}C peaks (theoretical value 2.2%). The pertinent results of this study are discussed as follows:

(1) In the water-only samples, no cross-contamination was observed at any stage of the experiment. Therefore, no exogenous substances appear to have entered the sample vials.

(2) The vials containing the water with the TCE-$^{13}C_1$ spike showed considerable variability in their percent recoveries, indicating substantial and inconsistent volatilization losses of the TCE. We were thus unable to obtain reliable quantitative data measuring the conversion of TCE to DCE by following the rate of loss of TCE. Any experiment that measures only the loss of TCE appears to suffer from these volatilization problems and from adsorption problems (to be discussed next). No degradation products of TCE-$^{13}C_1$ were observed in these water and TCE-$^{13}C_1$ samples, so soil or microorganisms contained in the soil must be present to effect this conversion.

(3) In the samples containing water, soybean meal (whether sterilized or not), and the TCE-$^{13}C_1$ spike, no conversion of TCE-$^{13}C_1$ to DCE-$^{13}C_1$ was observed. Thus, these control samples eliminate the soybean meal as a potential source of TCE degradation. However, adsorption of the TCE on the soybean meal was significant. From 50 to 60% of the TCE spike was adsorbed after 6 weeks. As seen from the sterilized soil samples (where, except in one case of incomplete sterilization, *no* conversion of TCE to DCE occurred), another 10–15% of the TCE was adsorbed on the soil. Thus, adsorption losses pose another major problem in a study like this. Experiments that monitor the loss of, for example, TCE and attribute it solely to degradation are potentially suspect, particularly if care is not taken to account for volatilization and adsorption losses.

(4) The 6-week method blanks (containing water, soil, and soybean meal with no TCE-$^{13}C_1$ spike) showed no generation of any substance (TCE or DCE, labeled or unlabeled) not already present in the soil itself.

(5) Conversion of TCE-$^{13}C_1$ to DCE-$^{13}C_1$ was noted in *all* unsterilized soils. Table I summarizes the amounts of labeled DCE produced. As seen from Table I, a general and gradual increase in the amount of DCE-$^{13}C_1$ produced occurs with time. (Of course, due to adsorption and volatilization losses, the amounts of DCE-$^{13}C_1$ actually produced are no doubt larger than those reported here. Actual amounts of TCE — DCE conversion in "real" soils may be even larger than those reported here, since the soybean meal added to ensure anaerobiosis may well have been a more attractive energy source for the soil microbes than the TCE. Indeed, breakdown products of the soybean meal were also noted in the unsterilized soil samples.)

(6) With one exception, *no* sterilized soils demonstrated any TCE — DCE conversion. One of the two 41-week C soil samples showed the presence of 2 ppb of DCE-$^{13}C_1$. This may, however, be the result of an incomplete sterilization since this was only observed for one of the longest time samples.

Since conversion of TCE-$^{13}C_1$ to DCE-$^{13}C_1$ occurred almost exclusively in unsterilized soils, microbial participation seems certain. Some caution should be exercised in drawing this conclusion, however, since Kaufman (*14*) has reported that autoclaving changes not only the biological properties of the soil but also its physical and chemical properties. Nevertheless, on the basis of our results for TCE and those of Parsons et al. (*10*) for PCE, it appears that the degradation of TCE to DCE in the soil is indeed of biological origin.

(7) Only 1,2-DCE-$^{13}C_1$ was produced whenever TCE-$^{13}C_1$ was degraded. No 1,1-DCE-$^{13}C_1$ (which elutes more rapidly than 1,2-DCE) was found in any sample. Under our experimental conditions, *cis*- and *trans*-1,2-DCE coeluted (and cannot be differentiated on the basis of their mass spectra). Thus, we could not identify which geometrical isomer was formed, or if a mixture of the two was produced. (In their study of PCE biodegradation, Parsons et al. (*10*) were able to separate the cis and trans isomers chromatographically. They found that *cis*-1,2-DCE is significantly favored over the trans isomer.)

Summary

By using TCE isotopically labeled with a single ^{13}C atom, we have shown that TCE is definitely dechlorinated in the soil to 1,2-DCE. Isomer specificity was also observed; no 1,1-DCE was detected. The TCE — DCE degradation appears to be biological in nature, since soil samples which had been sterilized exhibited no such conversion.

Since it has been shown that microbes that have adapted to degrade one member of a homologous series have also simultaneously adapted to degrade other members of the same series (*15*), the possibility exists that DCE can be further biotransformed into vinyl chloride in soils. Monitoring data at the Des Moines site (*11*) and elsewhere (*16*), this work and the work of Parsons et al. (*10*) all strongly support this DCE — vinyl chloride contention. Considering the well-known carcinogenicity of vinyl chloride, further research along these lines is definitely warranted.

Acknowledgments

We gratefully acknowledge John Caoile (of Ecology and Environment, Inc.) for obtaining the soil samples and Carl Bailey and Angelo Carasea (of the Region VII Environmental Protection Agency Laboratory) for providing technical assistance.

Registry No. TCE, 79-01-6; DCE, 540-59-0.

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Gas-Phase Hydrogenolysis of Polychlorobiphenyls

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■ Chloroarenes in an atmosphere of hydrogen are thermally dechlorinated to yield HCl and benzene as major products between 700 and 925 °C, with residence times of ca. 10 s. Polychlorobiphenyls (PCBs) are both dechlorinated and split into chlorinated benzenes, with splitting about twice as fast as dechlorination. Thermal hydrogenolysis, which occurs via radical mechanisms involving H atoms, may therefore be considered as a useful method for workup of (toxic) chlorinated wastes.

Following studies on thermolysis (*1–3*) and on several free-radical gas-phase aromatic substitutions—chlorination (*4*), cyanation (*5*), nitration (*6*), and oxidation (*7*)—we are now engaged in thermal conversions of benzene and derivatives with hydrogen. Within this category, "hydrocracking" of chlorinated arenes deserves special attention. In general, reaction 1 is of potential interest as a method

$$\mathrm{Ar(R)Cl + H_2 \xrightarrow{\Delta} Ar(R)H + HCl} \qquad (1)$$

for dechlorination of (highly) chlorinated industrial waste materials etc. Thermolysis of chlorinated benzenes in an excess of H_2 (quartz flow reactor, atmospheric pressure, residence time 5–15 s) proceeds smoothly at 750 °C and shows very high degrees of conversion (HCl formation) at ca. 900 °C (*8*). Sooting is unimportant even at 900 °C provided that the H_2:arene molar intake ratio is above 10. Aliphatic and olefinic chlorides, in general, react much faster than chlorobenzenes (*8*).

Polychlorobiphenyls (PCBs) have found widespread application, especially as transformer oil, its use and disposal entailing considerable environmental problems. We therefore thought it worth while to examine the behavior of PCB in hydrocracking (eq 1). Our observations, including those on appropriate model compounds reported below, confirm our expectation that PCB can be completely converted into HCl and non-chlorinated organic products, mainly benzene. Hydrocracking thus constitutes an environmentally clean alternative to incineration.

Representative examples with Aroclor 1248 (Cl = 48% wt) are outlined in Table I. That conversion of PCB is essentially complete and is illustrated by Figure 1. Dechlorination of chlorobenzene (PhCl) is ≥97%; monochlorobiphenyls are seen in minor amounts only, biphenyl comprising ca. 0.7% on the PhCl feed. This biphenyl stems from PhCl—or better, from benzene made therefrom—rather than from PCB (vide infra). Notable

Table I. Thermolysis of PCB in Chlorobenzene with Hydrogen[a]

	run no.			
	1	2	3	4
T, °C	715	760	805	875
τ, s	8.9	8.3	8.3	7.6
conversion[b] of PCBs, %	(ca. 10)	28	70	>99.9[g]
$PhCl_2$,[c,d] %	4.8	8.5	1.5	0.2
$PhCl_3$,[e] %	1.2	1.5	0.10	0
Ph_2,[c] %	0.010	0.034	0.040	0.60
$ClPh_2$,[d,f] %	0.053	0.18	0.13	<0.03
PhH:PhCl molar ratio	0.059	0.19	0.92	35

[a] Spiralized quartz tubular flow reactor (3.5 m, 46 cm³); inflow (mmol/h): H_2, 221 ± 4; PhCl, 10.2; Arochlor 1248 (0.69); duration of runs 40–65 min.; product collected in a trap cooled with liquid N_2. [b] By GLC with PhBr as internal standard; total of surface area from dichlorobiphenyl on (retention time > 27 min, Figure 1), assuming response to be independent of chlorine content. These numbers parallel those for degrees of dechlorination of PhCl, $PhCl_2$ or $PhCl_3$ under the same conditions. [c] Mole percent on PCB in × 0.5. [d] Mole percent on benzenes out. [e] Isomer distribution (ortho:meta:para, %): run 1, 40:25:35; run 2, 37:27:36; run 3, 32:33:35. [f] Isomer distribution (ortho:meta:para, %): run 1, 31:34:35; run 2, 32:35:33; run 3, 34:38:28. [g] Confirmed by GC with electron capture detection.

The di- and trichlorobenzenes clearly stem from splitting of PCB and account for ca. 6% of the Arochlor feed. Chlorobenzene is produced via the same route but is obscured by its use as diluent. Its amount can be estimated from what is known about the composition of the PCB mixture. Specifically, the identified portion of Arochlor 1248, ca. half, is composed of the following ratios of phenyl units: Ph:PhCl:$PhCl_2$:$PhCl_3$ = 0.02:1:1.3:0.16. If changes due to the small degree of dechlorination are neglected, PhCl from PCB would thus be 1:(1.3 + 0.16) × 6 ≈ 4%, so as to give a total degree of splitting of about 10%. PhCl alone yields ca. 6% of HCl under these conditions so this mode of hydrogenolysis is about twice as fast as dechlorination.

As we have reported elsewhere (*8*, *9*), methane is also formed, ranging from 0.2% (run 1) to 1% (run 4) on PhCl feed; small amounts of C_2H_4 and C_2H_6 and traces of C_2H_2 are also produced.

Simultaneous splitting and dechlorination of PCB will cause the yields of $PhCl_3$ and $PhCl_2$ to pass through a maximum with increasing temperature. The same holds for monochlorobiphenyl ($ClPh_2$) produced from PhCl. The

THE DOW CHEMICAL COMPANY

THE DOW CENTER
MIDLAND, MICHIGAN 48640

October 5, 1984

AIRBORNE

Document Control Officer
Management Support Division
Office of Toxic Substances (WH-557)
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Sir/Madam:

Attached for your information please find a copy of a summary of a report on a Lifespan Oral Carcinogenicity Study of Vinyl Chloride in Rats which we recently received and which I discussed with David Williams on Tuesday, October 2, 1984. This study was conducted in Europe by Civo Institutes TNO and was sponsored by Verband Kunstofferzeugende Industrie E.V. (VKI).

It is our understanding from VKI that the EPA will soon be receiving a copy of the final report. We do not have a copy.

Upon review of this summary, we have been unable to determine whether this study presents any substantial risk information under the EPA's Statement of Interpretation and Enforcement Policy. 43 Fed. Reg. 11110 (March 16, 1978). It appears from the minimal data presented in this summary that the study is corroborative of effects already documented in the scientific literature.

Sincerely,

John A. Gray

John A. Gray
Attorney
2030 Willard H. Dow Center
517/636-0933

To: Bob Oubre
From Fred Hoerger

Page 1 of 4

Attachment

bcc: F. D. Hoerger, 2020 WHDC
H. Schumacher, Horgen

SUMMARY

1. The oral carcinogenicity of vinyl chloride monomer (VCM) was examined in a lifespan study (149 weeks) with five groups of Wistar rats, each consisting of 100 males and 100 females, except for the top-dose group which comprised 30 males and 30 females. VCM was administered by incorporating polyvinyl chloride (PVC) powder with a high VCM content into the diet. The diet was provided daily for a period of 4 consecutive hours, whereas food was withdrawn during the other 20 hours. The use of this way of oral VCM administration resulted in the following exposure levels: 0 (control), 0.014, 0.13 and 1.3 mg VCM/kg body weight/day. An extra control group of 100 rats/sex was housed in a separate room.
Additional satellite groups of 18 male and 10 female rats, each receiving the same treatment as the main groups were used for determinations of glutathione levels in the liver after 9 and 18 months.
Observations were made of general appearance mortality, growth, food intake, thrombocyte count, prothrombin time, glutathione levels in the liver, gross pathology and microscopic pathology of the liver and of all grossly visible tumours or presumable tumours in the abdominal cavity, the glands of Zymbal and the mammary glands.

2. General health, behaviour, body weight and food intake were not adversely affected by the test substance.

3. In the second half of the experimental period, mortality in the extra control group was higher than in all other groups. This was most probably due to a high incidence of chronic respiratory disease in the extra control group. In the final stage of the study, the mortality in the top-dose group was slightly higher than in the lower dose groups and the controls.

4. Thrombocyte count, prothrombin time and liver glutathione levels did not show treatment-related differences among the groups.

5. A clearly higher incidence of grossly visible, tumourous, liver nodules was found in both males and females of the top-dose group than in any of the other groups. Moreover, in females of the top-dose group the incidence of hepatic cysts was considerably higher than in controls.

6. Microscopic examination of the liver revealed increased incidences of liver-cell polymorphism, hepatic cysts, foci of cellular alteration, neoplastic nodules and hepatocellular carcinomas in the top-dose group as compared to the control group. Moreover, a hepatic angiosarcoma was found in one male and two females of the top-dose group, whereas no such tumours were encountered in any of the other groups.
The number of animals bearing foci of cellular alteration in the liver was also statistically significantly increased in females of the mid-dose group as compared to controls. In addition, in females but not in males, the incidence of basophilic foci of cellular alteration in the liver was statistically significantly higher in both the low- and the mid-dose group than in the control group.

7. There was no evidence of VCM-feeding affecting the incidence of abdominal mesotheliomas or the type and incidence of mammary gland tumours. No Zymbal gland tumour was found.

8. It was concluded that under the conditions of the present experiment:

- VCM at a level of 1.3 mg/kg body weight/day induces neoplastic and non-neoplastic changes in the liver of rats,

- VCM at a level of 0.13 mg/kg body weight/day may lead to more female rats bearing foci of cellular alteration in the liver,

- VCM at levels of 0.014 or 0.13 mg/kg body weight/day may result in an increased incidence of basophilic foci of cellular alteration in the liver of female rats,

- 0.13 mg VCM/kg body weight/day is a "no-observed-adverse-effect-level" with respect to the induction of tumours in rats.

9. Risk estimation based on the results of the present rat study and taking into account the prudence of the linear model applied and a lesser sensitivity of humans to the carcinogenic action of VCM in comparison with rats, indicates that the cancer risk of a likely maximum oral daily intake of 0.1 µg VCM per person per day can be practically neglected.

RISK MANAGEMENT OF EXISTING CHEMICALS

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CHAPTER 10

VINYL CHLORIDE AND TSCA

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Air Products and Chemicals, Inc.[1/]

INTRODUCTION

The well-known regulatory history of vinyl chloride and its role as a bellwether of current regulatory philosophy makes it a useful paradigm for examining the relationship of existing laws and the Toxic Substances Control Act (TSCA) for control of chronic hazards.

To this end, we will first review some of the highlights of its regulatory history, and then engage in some speculation as to the response these events might elicit today under TSCA.

INDUSTRIAL AND COMMERCIAL USE OF VINYL CHLORIDE

Vinyl chloride became of industrial importance about fifty years ago, approximately a hundred years after its discovery, when Semons discovered that its polymer could be converted into useful articles by plastization with phthalate esters. Commercial development began first in Europe and then in this country in the late

1/ Air Products and Chemicals, Inc., 1983.

thirties, largely using existing rubber processing equipment, for it was rubber which it initially replaced in the market. For the same reason, the use of polyvinyl chloride (PVC) was sequestered by the government during the war years, and it was not until the early fifties that widespread consumer applications developed. PVC is now a mature product, and its growth rate falls in step with the Gross National Product. Presently, about six billion pounds are used annually in this country, and about four times that in the world.

Some of the broader toxicological attributes of vinyl chloride (VC) were recognized in the thirties. It was known to be an anesthetic, but problems with cardiac arrythmia prevented its use in that application.[2/] As pathological techniques improved, industry scientists recommended in the early sixties that exposure be limited to 50 ppm, because of temporary liver enlargement in animals at that level,[3/] but the American Conference of Governmental and Industrial Hygienists considered this overly conservative and accepted instead the 500 ppm recommendation of Harvard scientists.[4/] This was the value adopted by the Occupational Safety and Health Administration (OSHA) in its formative days.

Also in the early sixties, the European industry recognized among its workers a disease termed acroosteolysis, AOL, which is a degenerative disease of the bone tufts, particularly in the fingers, that is accompanied by Reynaud's phenomenon.[5/] An extensive epi-

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2/ W. F. von Oettigin, "The Halogenated Hydrocarbons, Their Toxicity and Potential Dangers," Public Health Service Publication No. 414 (Washington, D.C.: U.S. Department of Health, Education and Welfare, 1955).

3/ T. R. Torkelson, F. Oyers, and V. K. Rowe, "The Toxicity of VC as Determined by Repeated Exposures of Laboratory Animals," American Industrial Hygiene Association Journal, XXII (1961), p, 354.

4/ American Conference of Governmental and Industrial Hygienists, "Documentation of the Threshold Limit Value, 1963" (Cincinnati, OH, 1963).

5/ S. Suciu, J. Drejman, and M. Valaskai, "Study of Diseases Caused by Vinyl Chloride," Medical Intern., XV (1963), p. 967.

demiological survey here and in Europe found about a hundred possible cases which were associated closely with manual cleaning of reactor walls between polymerization batches, but neither the precise etiological agent nor the disease mechanism was identified.[6/]

An attempt was made to reproduce this disease in rats by the medical department of one of the European producers. An exact duplication of the human disease was not seen, but many of the rats developed tumors at numerous sites. The reporting of this finding by Viola[7/] in 1970 evoked little interest in the regulatory community, possibly because of the very high doses used, several thousand ppm, which were frankly toxic to the animals, and the fact that the tumors were largely metastatic from the Zymbal gland, an organ not present in humans.

Nevertheless, both the European and domestic producers formed consortia to perform bioassays at lower concentrations and also began epidemiological surveys of their employees.

Preliminary results of the European bioassay became available first in early 1973, and showed tumor development at much lower concentrations in organs which do have human counterparts. This result was transmitted to regulatory officials that summer, and industry screening of employee records was intensified.[8/] This resulted in the recognition that winter by an industry medical director of a cluster of three rare liver tumors termed angiosarcoma, ASL, in the employees of one facility.[9/] The reporting of this fact to

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6/ W. A. Cook, et al., "Industrial Hygiene Evaluation of Thermal Degradation Products from PVC Fetus in Meat-wrapping Operations," Arch. Environ. Health, XXII (1971), p. 74. Also, B. D. Diman, et al., "Occupational Acroosteolysis I, An Epidemiological Study," ibid., p. 61.

7/ P. L. Viola, "Pathology of Vinyl Chloride," Medicina del Lavoro, LXI (1970), p. 174.

8/ A. W. Barnes, "ICI Ends Its Silence on Vinyl Chloride," Chemical Engineering News, (July 8, 1974), p. 21.

9/ J. L. Creech and M. N. Johnson, "Angiosarcoma in Workers Exposed to Vinyl Chloride as Predicted for Studies in Rats," Journal of Occupational Medicine, XVI (1974), p. 150.

government officials led to the current regulatory status of vinyl chloride.

It also led to a virtual explosion of research on the chronic toxicity of VC. The body of scientific literature on the oncogenicity of vinyl chloride is as large as that for any other substance. It is recognized that VC is a classical procarcinogen. Metabolism by the mixed function oxidase in the liver converts it to the ultimate carcinogen, an epoxide. Detoxification of this intermediate by the sulfhydryl group of glutathione or other proteins removes the toxic potential.[10/] Both of those mechanisms are saturable.[11/] An overload of the metabolic step assures that the vinyl chloride will pass through the liver and some will be metabolized in other organs. An overload of the detoxification step allows escape of the toxicant into the sinusoidal passages of the liver where interaction with the chromosomal protein causes ASL to develop. An overload of both mechanisms can lead to tumor development outside of the liver, as is seen in mice and rats at very high doses. Despite the large data base, however, information on the precise mechanism of these various steps still is lacking. We do not even understand why some persons respond with AOL and some with ASL, but none with both diseases.

REGULATORY STANDARDS

OSHA proceeded promptly in early 1974 to set an emergency temporary limit of 50 ppm for worker exposure, and later that year reduced the limit to one ppm, the current figure. Industry was given a grace period during which respirators could be used to meet this requirement, but now that level must be met by engineering practices.[12/]

10/ W. K. Lelbach and H. J. Marsteller, "Advance in Internal Medicine and Pediatrics" Springer-Verlag, XLVII (New York, 1981).

11/ R. Hefner, P. Watanabe, and P. Gehring, "Percutaneous Absorption of Vinyl Chloride Gas in Rhesus Monkey," Toxicology and Applied Pharmacology, XXXIV (1975), p. 529.

12/ OSHA Standard for Vinyl Chloride, 29 CFR 1910.1017.

The Environmental Protection Agency (EPA) promulgated a combined engineering and works practice standard in 1976 which has resulted in ambient concentrations in the fractional ppb range near producing or using facilities.[13/]

In the meanwhile, the Food and Drug Administration (FDA) and Consumer Product Safety Commission (CPSC) established prohibitions on the use of VC in aerosol or other consumer applications, a practice which had been discontinued in 1973. The Bureau of Alcohol, Tobacco and Firearms of the Treasury Department (BATF) had already banned the use of PVC liquor bottles in 1973 because of concern for taste effects from migration of residual VC into the contents. In 1975 the FDA proposed revocation of the generally regarded as safe (GRAS) status of rigid PVC packaging under the Delaney clause, also because of migration concerns, but that proposal never has been promulgated, and the FDA has stated that it is considering withdrawal of the proposal and recommending to BATF the reauthorization of plastic liquor bottles in light of the current very low residual monomer levels in fabricated PVC articles.

Other regulations have followed as new statutes and rules have come into play. The Department of Transportation (DOT) and the Coast Guard regulate the transportation of VC, of course, and VC is listed as a priority pollutant and hazardous waste under various water and solid waste rules, and has a reportable quantity of one pound under Superfund.

Did the existing laws operate satisfactorily at the time of discovery of the chronic hazards of VC? It appears that they did. A leading medical authority who was deeply involved in the worker health evaluation in 1974 has termed VC a "success story." Reevaluation of the risk to employees under the current 1 ppm standard by a conservative nonthreshold extrapolation method[14/] yields a lifetime estimate of less than 10^{-8}, a risk level which is not thought to be of concern. The comparable risk estimate for the general populace is

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13/ EPA Standard for Vinyl Chloride, 40 CFR 61.60.

14/ P. J. Gehring, P. G. Watanabe, and C. N. Park, "Risk of Angiosarcoma in Workers Exposed to Vinyl Chloride as Predicted for Studies in Rats," Toxicology and Applied Pharmacology," XLIX (1979), p. 15.

several orders of magnitude lower. EPA has stated on several occasions that it believes that vinyl chloride is regulated adequately.

RISK ASSESSMENT

Risk assessment has been a popular avocation among those interested in VC, and more than a dozen have been performed.[15/] These can be divided generally into two classes: those which rely solely on animal data; and those which attempt to incorporate the human experience.

Those in the first class yield similar results, and show the normal spread of estimates from the various mathematical models in common use. These range from 1,500 to 10^{-5} ppb for a lifetime risk of 10^{-6}, or eight orders of magnitude. It is necessary to eliminate the high-dose data points, that is, those over 2,500 ppm from the Maltoni data[16/] in order to get reasonable fits to most models, because these doses show broad systemic toxicity. The lower doses, 500 ppm and below, as a group fall into a general pattern on a log-probit plot, but individual two or three dose experiments show tremendous differences in slope when plotted separately. The popular multihit model predicts a lifetime risk of 10^{-6} at fractional ppb levels.

The human factor was accounted for in two ways. The EPA used some preliminary employee epidemiological data to confirm its animal-based extrapolation.[17/] Unfortunately, the human data were

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15/ J. T. Barr, "Risk Assessment for Vinyl Chloride in Perspective," (Paper 82-9.2 presented at the 75th Annual Meeting of the Air Pollution Control Association, New Orleans, LA, 1982), Lines 20-25.

16/ C. Maltoni, et al., "Vinyl Chloride Carcinogenicity Bioassays (BT Project)," (Paper presented at "Le Club de Cancerogenese Chemique," Institute Curie, Paris, November 10, 1979).

17/ A. M. Kusmack and R. E. McGoughy, "Quantitative Risk Assessment for Community Exposure to Vinyl Chloride," (Washington, D.C.: U.S. Environmental Protection Agency, December 5, 1975).

selected from those locations known to have ASL cases, while other facility data were omitted. They also were in error on the past exposures by more than an order of magnitude. This resulted in an estimate of 20 cases per year from the estimated 1974 ambient concentrations for the population within five miles of production and processing facilities.

The EPA seldom bothers to check its estimates against available data, so it sometimes comes up with results such as that made for arsenic a few years ago that would have predicted 18 million cases of skin cancer a year in this country if it had been applied to Agency data on the average arsenic concentrations in drinking water. Similarly, a survey of all known ASL cases in this country for the ten years before 1974 showed no cases associated with residency near such plants,[18/] rather than the 200 predicted cases. It is reasonable to assume that if any cases had developed since that time, the publicity associated with it would have brought them to light. Thus we have 110 million-person years of negative history for nearby residents. This places an upper limit on risk of less than 10^{-7} per ppm-yr.

Two studies applied pharmacokinetics in an attempt to obtain relevant human data. Gehring and coworkers estimated a lifetime risk of 10^{-8} at one ppm from the probit model, based on a biotransformation of rat data. The unconstrained linear model predicted no risk at less than 99 ppm.[19/]

Anderson, Hoel and Kaplan carried this procedure one step further, and applied it to bound metabolic products, rather than to the total amount metabolized. Their results gave a lifetime risk of 10^{-7} at less than one ppm, with the probit model, or at less than two ppm with the linearized multistep model.[20/]

18/ H. Popper, et al., "Development of Hepatic Angiosarcoma in Man Induced by Vinyl Chloride, Thorotrast, and Arsenic," American Journal of Pathology, XCII (1978), p. 349.

19/ P. J. Gehring, P. G. Watanabe, and C. N. Park, Toxicology and Applied Pharmacology, XLIX (1979), p. 15.

20/ M. W. Anderson, D. G. Hoel, and N. L. Kaplan, "A General Scheme for the Incorporation of Pharmacokinetics in Low-dose Risk Estimation for Chemical Carcinogens. ibid., LV (1980), p. 154.

Thus we see that risk is in the eye of the estimator, but it is clear that estimates incorporating human data reflect the human experience for VC far better than do the direct application of animal data.

There was understandable uncertainty on the part of both the regulators and industry in 1974. This was the first commodity chemical to be regulated under the relatively new statutory situation as the result of new information. Nevertheless, both the regulatory agencies and industry acted promptly to reduce exposures and emissions to an acceptable level.

The current count of occupational ASL cases is about 100 worldwide, with 30 of these in this country.[21/] All these cases had their first exposure in 1964 or earlier, and there appears to be room for optimism that the steps taken in the mid-sixties because of the AOL information will have prevented any significant number of cases developing from exposures commencing after that date. Certainly it is reasonable to expect that there have been no new cases initiated after the early seventies.

Had TSCA been in place in the mid-sixties, would it have made any difference in the course of events? It appears unlikely that it would. Certainly the AOL discovery would have resulted in a series of 8(e) notices to TSCA. The probable outcome of that would have been either a recommendation from the Interagency Testing Committee (ITC) for more tests, or a Section 4 testing requirement. It is possible that, because of its commercial importance, VC could have been placed on the ITC list before the AOL data became available. Additional data could have been called for under Sections 8(a) and (d). The result of all this most likely would have been a negotiated testing rule, under which industry would have initiated a series of studies which would have culminated in a bioassay, and the carcinogenicity of VC would have been discovered in due time. Yet, this is precisely what did happen in the absence of TSCA, except that the preliminaries were omitted, and the bioassay was performed concurrently with the screening tests. Thus it is possible that the

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21/ J. Stafford, personal communication, Liver Angiosarcoma Cases, April 15, 1983.

final critical data were obtained earlier than would have occurred under present conditions.

Bear in mind that most of today's powerful testing methods were not available twenty years ago. That fact would not have been changed by legislative fiat, and any decision made at that time had to be made in light of the available knowledge.

If the data of Viola suddenly became available today instead, would there be any significant difference in the outcome, or the timing of that outcome? Probably so, but only because of the vastly more powerful scientific tools which we have available to us now. Neither the speed of agency motion nor the rate at which industrial facilities can be built or modified has increased. If anything, the latter has slowed, given the multiplicity of permits and approvals now required. Overall, it is possible that if today we knew nothing more about VC than was known in 1970, we would arrive at a regulated state a few months earlier than was achieved in 1974, but scientific progress, and not legislative or regulatory advancement, should get the credit.

What if VC were to become a new product today? Would it run the same course in which it would be 40 years before there was full recognition of its chronic potential? Certainly not. Again, however, the reason is due more to scientific progress rather than statutory development.

One change might be apparent. If VC were the subject of a Premanufacture Notification (PMN) today, rather than being the model to which all other aliphatic olefins are compared for structure-activity analysis, it would be judged by the others in its family. This comparison would be less dogmatic than the reverse is now. Ethylene and vinylidene chloride are not animal carcinogens; the relevance to humans of the carcinogenicity of high doses of trichloroethylene (TCE) is equivocal and controversial; and vinyl acetate has only a preliminary "non-negative" report. Thus, this class of substances would have lost its leader for structure activity comparison, and a decision as to the need for further testing from that analysis would not be clear-cut, based on analogous compounds.

Neither would a full minimum premanufacture data (MPD) set be of any great assistance. VC responds poorly to the classical in-vitro tests, and only recently has it become possible to obtain reproducible positive results in many of these. If the position were taken

"little lists" for the executioners apparently is too great to be resisted,[26/] as Lester Lave pointed out recently.

We believe that EPA can best obey its statutory mandate by developing a more efficient system for establishing priorities, and by implementing more effectively its Section 9 procedures.

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Risk Assessment for Vinyl Chloride in Perspective

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For Presentation at the 75th Annual Meeting of the
Air Pollution Control Association
New Orleans, Louisiana June 20-25, 1982

Introduction

The combination of circumstances which found the carcinogenic hazard of vinyl chloride (VC) being discovered at about the same time as the science of risk analysis was undergoing rapid development, and the great commercial interest and long history of use of the substance has resulted in a body of literature and pharmacological data greater than one can expect to have for most substances. It is therefore instructive to review the many risk assessments which have been prepared for VC against the available biological information to determine if we can evaluate the extrapolation methods used, and to discuss the current regulations for VC in light of this comparison.

Hazards of Vinyl Chloride

It is necessary to decide first which of the hazards presented by VC should be the basis for the risk estimation. The substance presents the acute hazards of frostbite from exposure to the liquid, of anesthesia at concentrations over 8,000 ppm and suffocation at higher concentrations (von Oettinger, 1955). It also forms explosive mixtures in air above 3.75 volume percent, and so the efforts to control the physical safety of operations generally preclude exposure to acutely toxic concentrations.

These control efforts were reinforced in the mid-1960's where it was discovered (Suciu, 1963) that workers who had been exposed to very high levels of VC developed "vinyl chloride disease," the primary manifestation of which was acroosteolysis (AOL), a degenerative disease of the bone tufts in the hands, and more rarely of the feet and lumbar region. Although crippling to some degree, this disease is not fatal, and is at least partially reversible if exposure is eliminated (Granger, Walker and Ward, 1980).

Almost ten years later it was found that some of the workers having similar exposure also were developing angiosarcoma of the liver (ASL), a rapidly fatal disease. Oddly enough, there is only one possible case of a worker developing both AOL and ASL (Stafford, 1981) among the 80-plus cases of AOL and 90-plus cases of ASL now known worldwide, although both are diseases of the vascular system. Several large epidemiology studies were conducted on workers exposed to VC (Baxter and Fox, 1976; Chiazze, 1980; Duck, Carter and Coombu, 1975; Equitable Environmental Health, 1978; Fox and Collier, 1977; Frentzel-Beyme, Schaltz, and Theiss, 1978; Theriault and Allord, 1981), and ASL was the only fatal disease found consistently to be in excess in these

persons. Animal studies have shown an excess of tumors at other sites, but the lowest exposures at which these occur are considerably higher than that for ASL. For example, Maltoni (1979) reported the following data:

Site	Concentration For Significant Elevation
Forestomach papillomas:	30,000 ppm
Neuroblastomas:	10,000 ppm
Zymbal gland carcinomas:	10,000 ppm
Nephroblastomas:	250 ppm
Liver angiosarcoma male:	200 ppm, 50 mg/kg
female:	50 ppm, 16.7 mg/kg
Mammary adenocarcinoma:	5 ppm

The low concentration for onset of mammary tumors was of concern when a preliminary study of fabrication employees reported an excess of breast tumors (Chiazze, et al., 1979) but a follow-up case-controlled study (Chiazze, 1980) found no association between the cases and VC exposure. The largest study of VC-PVC workers in the United States reported slight excesses of brain and lung tumors (Equitable Environmental Health, 1978), but this was not seen in the other studies referenced above. The excess of brain tumors was small, and not dose- or exposure-related. The overall excess of lung tumors resulted from an excess in one plant only, and reexamination of those cases also showed no association with VC exposure (Waxweiler, 1978).

Vinyl chloride has been found to be active in several in vitro mutagenetic tests with bacteria and yeasts (Hopkins, 1979) and it appears to cause chromosome abnormalities in exposed workers, but these changes are reversible when exposure is reduced (Hansteene, 1978) and several studies of neighborhoods around PVC plants have failed to show a supportable association with birth defects (Edmonds, 1975, 1976). It is not a teratogen in rodents (Johns, 1977).

Therefore it appears reasonable to assume that if there is any significant chronic risk other than ASL, it is considerably smaller than that for ASL, and that an adequate risk assessment can be based on only the liver tumors.

Review of Risk Assessments

1. Schneiderman, 1975

One of the first attempts to utilize animal data to estimate risks at very low exposures was that of Schneiderman, Mantel and Brown (1975). They used preliminary Maltoni results to compare the estimates obtained from three possible mathematical models. The 99% assurance level of a "safe" dose at a lifetime risk of 10^{-6} was estimated from several extrapolation models as follows:

Log Probit (slope = 1)	73 ppb
Logit (slope = 3.45)	119 ppb
Logit (slope 2.3, one-hit)	2.1 ppb

The authors discussed the recognized difficulties of extending these rat data to humans and of providing animal experiments that could answer satisfactorily the question of human risk at very low doses.

2. Kuzmack and McGaughy, 1975

The EPA was the first group to attempt a human risk assessment for vinyl chloride (Kuzmack and McGaughy, 1975). This pioneering effort attempted to use both animal and human data, and to show comparative results from both the linear and log-probit models. It concluded that there was an individual risk of 71×10^{-6} per ppm of lifetime exposure to VC by the linear extrapolation method, and that the log-probit results were one-tenth to one-hundredth of that.

This effort is subject to several serious criticisms. The exposure data used for human experience was that from a group with less than average exposure, while the ASL rate was chosen from only those plants which did report cases, and ignored the remainder of the population. Thus, their incidence rate of 7.5% compares to an actual figure of about 0.1%.

They used as their primary method a linear extrapolation of rat data, which often has been seen to overestimate the actual rates by at least two orders of magnitude, and they assumed the total cancer rate to be twice that found for ASL.

This same estimate was used by the EPA (1979) to estimate the concentration of VC in drinking water which would produce various levels of risk. These estimates are, of course, subject to the same criticisms.

Nisbet (1978) challenged the estimate of Kuzmack and McGaughy (1975) when it was used by Wilson in testimony before the OSHA hearing on its generic cancer policy. Nisbet stated that his calculations showed the risk to be 10-30 times greater, by the same calculation method. Wilson (1978) suggested several flaws

in the Nisbet procedure, including the fact that he chose for hi extrapolation one point at 25 ppm from Maltoni experiment OT-15, and that this point is not in good agreement with the whole body of data. Further, he chose to use total cancer incidence in the rats, including those at zymbal glands, which have no counterpar in humans. Both Wilson and Kuzmack and McGaughy had used a factor of two times ASL to account for possible cancer at other sites. Wilson did acknowledge a mathematical error which made his results half the proper number.

Albert (1978) applied this same general procedure to other poten tially carcinogenic air pollutants in the United States and calculated the expected annual cancer deaths as follows:

Arsenic	15.6
Benzene	77.8
Cadmium	26.2
Coke ovens	149.5
VC (after regulation)	1.0

3. Gehring, 1979

Gehring, et al., (1979) applied an experimentally derived bio-transformation correction (Gehring, et al., 1978) to rat data an estimated the incidence in humans at two different exposures by means of four different extrapolation models. Their estimates a 500 and 200 ppm TWA bracket the observed experience for humans when derived from the probit and the unconstrained linear models. The linear-through-zero and one-hit models consistently over-estimated the incidence. Although not considered by the authors the linear and probit models match rather closely the total U.S. experience of occupational ASL at an assumed 1,000 ppm exposure. The linear model predicts no incidence below 99 ppb in humans. The probit model predicts a human risk of 1.5×10^{-6} at 1 ppm. Thus, a mechanism for adjusting for the difference in metabolism between animals and humans appears to be useful.

A limitation of the Gehring procedure is that it uses partial Maltoni data, and tests the results against the CMA epidemiology study. That study was not the "end of the experiment"; it stoppe at the end of 1973, and several deaths have occurred since then. Neither did it cover the entire population, but only the employee of those plants which met certain criteria for data retention and length of operation. The Stafford (1981) data does cover the entire population and extends the history for seven years. The size of the population is not known, but a reasonable estimate, based on normal worker turnover rates and the number of plants not included in the CMA study, is certainly not less than 25,000. This would give a gross incidence of about 0.1%. Of these, the number actually exposed to substantial exposures would be about 25-30 per plant at any one time. Multiplication by 25 plants, and a factor of three for the turnover during this period, would give about 2,000 highly exposed persons, for an effective inci-

dence of just over 1%. Personal experience would indicate that, for the period prior to 1964, when all of the first exposures of the fatal 26 cases had occurred, the average exposures of this highly exposed group certainly was in excess of 1,000 ppm for the working day. Maltoni (1979) found a 1% incidence at about 1-10 ppm in rats. Calculation of the dose equivalent to a 1% incidence in rats gives 0 ppm by the linear method and 7.5 ppm from the log-probit equation for the combined Maltoni inhalation experiments. This crude and subjective estimate would then say that man is about 100 times as resistant as the rat to VC inhalation, a figure generally in agreement with other estimates (NCAB, 1979).

4. Food Safety Council 1978, 1980

The Food Safety Council has recommended (FSC, 1978) the use of the gamma multi-hit model because of its flexibility in handling dose response data of varying curvilinearity at low doses. It has calculated (FSC, 1980) the maximum likely and lower 97.5% limit doses for substances at various risk levels and with different models. For VC, at 10^{-6} risk, these results are as follows (based on early Maltoni data):

One-hit	2.0×10^{-2} ppm
Armitage-Doll	2.0×10^{-2} ppm
Weibull	2.1×10^{-9} ppm
Multi-hit	3.9×10^{-10} ppm

For this substance, the goodness of fit of the Weibull model (0.56) was superior to that of the multi-hit (0.32). Neither of the other two models gave acceptable fits. This was in part because of the concave shape of the curve, which included all of the high doses in the dose response data.

5. Dow, 1979

A Dow Heath Team performed a relative risk estimation for several compounds (Langer, et al., 1979) which considered probable exposure, the consequence of exposure, the physical state of the substance during processing, and the current exposure standards. This resulted in a value of 480 for VC in a "closed system but with employees in the vicinity." The same procedure assigned hazard rating values to some other substances as follows: benzene, 10; phosgene, 410; hydrogen sulfide, 5; arsine, 9,700; and bis-chloromethyl ether, 69,700. In a batch operation with occasional manual handling, the hazard rating for VC increased to 9,700 by this method.

6. Mehir, 1980

Mehir, et al., (1980) conducted a series of tests for the Consumer Product Safety Commission, a part of which consisted of exposing rats and mice to a series of short, high exposures, rather than

the usual extended low dosage. They included one-hour exposures to rats and mice at 50, 500, 5,000, and 50,000 ppm, 10 and 40 hour exposures at 500 ppm, and 49 and 100 one-hour exposures at 50 ppm. After lifetime observation they found no effects on rats, or their offspring, nor on mice exposed to less than 500 ppm. Those exposed to over 500 ppm developed pulmonary adenomas, but they also had suffered from pneumonitis.

They considered the published data on animal exposures and concluded that there was a lifetime dose below which no oncogenic response is seen. This was estimated to be 5,000 ppm-hrs for mice and greater than 50,000 ppm for rats, regardless of whether the dose was administered over a short or long period. This concept of equality of effectiveness for all modes of exposure does not have general acceptance and would not appear to be correct, based on our present understanding of carcinogenesis. Dose-rate effects are, of course, well known. However, the degree to which this can be extended to all types of effects is not known.

These authors also used the Crump-Guess model (Crump, Guess and Deal, 1977) to evaluate their data on mouse pulmonary cancer, and estimated that exposure to 5,000 ppm VC doubles the probability of cancer, while 50,000 ppm increased the risk nine-fold. In view of the fact that pneumonitis was present in all animals exposed above 500 ppm, it is questionable if this was a direct oncogenic response, or the result of an nongenetic event because of severe lung damage. Maltoni (1979) also reports an increase in lung tumors in mice, but not in rats or hamsters. Thus, the significance of this finding to risk in humans is questionable.

7. Anderson, 1980

Anderson, et al., (1980) extended the work of Gehring, et al., (1978 and 1979) to incorporate the amount of metabolic products from VC which actually was bound to the DNA of exposed rats, (Gehring and Blau, 1977) rather than the total amount metabolized. They assigned various values to the parameters in a Michaelis-Menten equation depicting the kinetics of the metabolic process, and compared the results from extrapolation to low doses by log-probit and multi-hit models. They found that the two extrapolation models responded quite differently to these variations at very low doses, and that it was not possible to select one model as the more appropriate from the high-dose data. Use of the values of Gehring for the primary parameters gave estimates of the dose equivalent to lifetime risks of 10^{-7} of less than 1 ppm for the probit model and less than 2 ppm for the multistage model, a correspondence which the authors pointed out was better than the precision of interspecies comparisons.

8. EPA, 1980

The final version of the water quality criteria document for VC (EPA, 1980) used a different approach for risk estimation. The slope of the incidence of all tumors at the lowest doses of Maltoni experiment BT-1 was adjusted for the fraction of exposure, the equivalent feeding level to give the same blood concentration of VC as by inhalation (see Withey and Collins, 1976), and the ratio of the surface area of humans vs. rats, to produce an estimate that a lifetime risk of 10^{-5} would be caused by drinking 2 l/day of water containing 20 g/l. There is some confusion in the mathematics given in the report, and the assumptions on which the adjustments are made are far from having general acceptance, although generally following NAS recommendations. It appears that this procedure overstates the risk by several orders of magnitude.

9. NAS, 1980

The National Academy of Science (1977) calculated the upper 95% confidence limit for risk from drinking water containing vinyl chloride from the probabilistic multistage model and early Maltoni rat data. They report (NAS, 1980) a lifetime risk of 10^{-6} as being equivalent to 3.0810^{-} mg/kg/day. For a 70 kg person consuming 2 l/day, this would calculate to an acceptable level of 1 g/l. The difference between the EPA and NAS numbers comes from the different curve-fitting methods for the animal data.

10. Gaylor and Kodell (1980) applied linear "interpolation" to the same early Maltoni data used by the Food Safety Council (1978) to arrive at a predicted maximum risk of 10^{-6}. The upper 97.5% confidence limit of the animal data was taken as one point on the interpolative line, and zero incidence at zero exposure as the other. This produced a lower 97.5% confidence limit dosage of $7.1 \times 10^{-}$ ppm for a lifetime risk of 10^{-6} in rats. Their application of the Armitage-Doll multistage model gave 5.2 $\times 10^{-}$ ppm as the dosage at 10^{-6} lifetime risk compared to $2 \times 10^{-}$ by the Food Safety Council. The difference is due to alternative assumptions on the value of the exponential dose term.

11. Crump and Guess (1980) reviewed some of the earlier risk estimates for vinyl chloride in drinking water, and recalculated the risks, using the one-hit and multistage models. They arrived at an upper 95% confidence limit of lifetime risk for drinking water containing 1 g/l of VC of 4×10^{-6}, based on early Maltoni inhalation data. Using the assumption that a 0.2% incidence of ASL in workers had resulted from a lifetime exposure of 70 g/kg, they obtained a maximum likelihood risk of 10^{-6} from 0.34 g/l by both the multistage and linear models, with 95% lower confidence limits of the same risk at 0.24 g/l. These two models reduce to a linear form when used at very low doses and with the assumption of no threshold value.

These authors cite EPA data on the occurrence of VC in public water supplies which by their methods yield a lifetime risk of $3.7 \times 10^{-}$, or 12 deaths per year from this cause in the United States. None of these has been observed, despite the accumulation of 15 years' data on ASL deaths (Popper, 1978).

12. Scott (1981) ascribed the decreased incidence of tumors in rats at the higher doses to a cell killing process, and adopted the Weibull model to account for this. Application of the model to some early Maltoni data produced a curve which fit the data from 50-10,000 ppm. He did not attempt to extrapolate to doses beyond the experimental range.

13. Carlborg, 1981, also applied the Weibull model to 31 bioassay reports on a variety of animal carcinogens. He concluded that the one-hit model was not appropriate and that carcinogens could be divided into categories according to the shape of the curve, e.g., concave or convex. He found that the early Maltoni data on VC fell into the former category. Application of his parameter estimates to those data, assuming no spontaneous incidence of ASL, gives $2.5 \times 10^{-}$ ppm for a lifetime risk of 10^{-6} for rats. Later calculations including all of the published Maltoni data did not change the results significantly (personal communication).

He found the Weibull shape parameter to be approximately 0.5, which is assumed to be the number of stages for tumor initiation. This is consistent with the finding by Gehring (1977) of a saturable metabolic path which produces the proximate carcinogen. It also suggests that the number of "stages" is the number of finite-rate steps before the rate-limiting step. There may be other stages following, but they are not rate controlling. Actually, there appears to be at least two saturable mechanisms involved in the pharmacokinetics of VC, the metabolism to the ultimate carcinogen and the detoxification by sulfhydryl groups.

14. One further evaluation of human risk can be made from the experience of persons residing near VC-PVC plants. The EPA estimated (Kuzmack and McGaughy, 1975) that five million persons lived within five miles of these plants, and were exposed to an annual average concentration of 17 ppb. The present distribution of plants was generally well-established by 1959, thus we have 22 years of history, or about 110 million person-years. About five or six of these plants, with 1-2 million neighbors, go back another 20 years, but these data are not firm enough for inclusion.

The fact that no case of ASL has been confirmed as arising from these ambient exposures places the upper bound of risk at less than $2.7 \times 10^{-}$ per ppm-yr. It is believed that the exposure data were overestimated by EPA, and thus this result may be too low, but it is in the same general range as that arrived at by Gehring (1979) and Anderson (1980) after making corrections for pharmacokinetics.

Extending this crude calculation, these five million persons are now supposed by EPA to be exposed to 0.2 ppb (probably a high figure), which would predict no more than 0.0003 deaths per year, or one per 3,700 years in that whole population due to VC exposure. But it also must be recognized that with approximately 20 cases per year of ASL in the general population, there can be expected from a purely statistical basis that there should be one case every two years or so among this group of 5 million plant neighbors.

The results of these estimates discussed above are compared in Table I, after conversion to a uniform 10^{-6} lifetime risk. Estimates 5, (Dow 1979) and 12 (Scott, 1981) were not in a form to permit this comparison. See OSHA, (1980), for references to a few other estimates that were not considered here.

It can be seen that the results fall into two major categories, those which project that the risk of 10^{-6} occurs at exposures of greater than 1 ppm, and those which find that risk in the ppb range. The estimates which yield the higher allowable exposures are based on human data (Nos. 3, 7 and 14) or use a log-probit extrapolation model (No. 2, second estimate), or predict a threshold (No. 6). The remainder generally are based on the linear, non-threshold model, and make no biological correction. The result is a difference of 3 or 4 orders of magnitude. The estimates which yield the higher allowable exposures are in better agreement with human experience than are those of the other group.

Additional Data

All of the extrapolations reported here have used for the original Maltoni data from his experiment BT-1. He has now reported (Maltoni, 1979) three other comparable inhalation experiments on the same strain of rats, and one on another strain, in addition to two ingestion studies. The results of these experiments are shown in Figure 1, on a log-probit scale. It can be seen that they all follow a similar pattern, but that there are large variations in slope between the various data groups. Table III contains the log-probit equations calculated from some of the individual experiments, and various groups of experiments. Excellent fits are obtained for a single experiment, as would be expected from the small number of data points, but adequate fits are obtained for the group as a whole. Inclusion of the historic control data on ASL (0.09% spontaneous incidence) did not affect the fit substantially, except for the very low dose data. Inclusion of the 0,0 (origin) as a data point did give significantly poorer fits. The combined experiments indicate that a lifetime risk of 10^{-6} for rats is obtained from a dose in the 1-2 ppb range.

Similar variation is seen with the other mathematical expressions, such as linear or exponential equations.

Regulatory Status

The current regulatory status of vinyl chloride is summarized in Table II. The first regulatory action on VC was taken in 1973 when the Bureau of Tax, Alcohol and Firearms prohibited the use of rigid PVC as liquor containers. This was based on it being present as an adulterant and not on any consideration of risk. The Consumer Product Safety Commission (CPSC), the Food and Drug Administration, (FDA), and the EPA all acted to ban the use of VC as an aerosol propellant thus establishing a zero risk position. The FDA proposed (FDA, 1975) to withdraw the prior sanction status of rigid PVC as a food package component because of the concern for residual VC that might migrate. The FDA has taken no further action on this proposal, and now is considering a "constituent" policy which would permit a lifetime exposure at some acceptable risk level. This risk has been proposed recently to be 10^{-6} lifetime for the gluttonous consumer. As was discussed above, the EPA required a best available technology approach which reduces the average exposure to those within 5 miles of a plant to about 0.2 ppb, by EPA estimates. OSHA established a rule in 1974 which set 1 ppm for 8 hours as the maximum permissible exposure, and also set 0.5 ppm as an action level below which most features of the regulation did not apply. These were chosen as feasible levels, and not necessarily "safe" doses (OSHA, 1974; EPA, 1976).

The EPA has established an exposure to the general population only 0.1% of that allowed in the workplace. The CPSC has required zero exposure, and the FDA has considered that approach. Depending on which method of estimation the FDA may choose, its allowable exposure could be either greater or less than those currently set by EPA and OSHA. It has been estimated that the maximum amount of VC ingested by the average European, who uses much more plastic packaging than we, is less than 2_6 g/day, (CEFIC, 1976) which would be in the order of a 10^{-5} or 10^{-6} lifetime risk by even the most conservative models.

There have been various estimates made of the cost-effectiveness of the Federal regulation for vinyl chloride. Graham and Vaupel (1981) estimated that the OSHA rule cost $7.5 million per life saved, and $490 thousand per life-year saved over the option of leaving the exposure limit at 50 ppm. Luken and Miller (1981) state that the imputed value of a life from the OSHA standard is $4 million. Morrell (1982) uses an annual cost of $20 million and an annual benefit of 0.1 life saved to derive a cost/benefit of $200 million per life for the OSHA rule. The EPA has reported (EPA, 1979) that the cost of compliance with its VC standard was $296 million through July 7, 1981, and will be an additional $470 million during the next five years, all in 1977 dollars. If the EPA estimate of up to 20 deaths per year were correct, this would be a cost of $4.7 million per life. However, as discussed here, there is no evidence that any lives have been saved by this rule.

There are many difficulties in obtaining accurate estimates of this type, and serious problems in determining the proper value to be assigned to a life. nevertheless, the doubtful nature of the claims

for any significant benefit from these rules suggests that at best, these regulations are excessively costly to society. Therefore, we must attempt to improve both our data base and our methods for interpreting and applying the data.

Discussion

What can be learned from this exercise other than the already recognized fact that various extrapolation models can yield very different results? In this case, at least, there are several points which are worth considering.

1. Vinyl chloride is no exception to the rule that human data always must be incorporated whenever possible. The epidemic of occupationally induced ASL which was feared in 1974 has not materialized, probably due to the steps that were taken in the early 1960's to reduce exposure because of the discovery of AOL. No instances of ASL from exposure to VC in the general population have been substantiated. The overprediction of occupational cases was due to the underestimation of worker exposure and overreliance on raw animal data without proper pharmacokinetic adjustment. We are not now able to extrapolate reliably between similar species and certainly not from rodents to humans, without much additional data.

2. The regulations for vinyl chloride were not based primarily on scientific data, but on socioeconomic and political decisions. This is no surprise (Crandall and Lave, 1981), but is a fact which should be acknowledged openly, along with the understanding that this position will continue to penalize good science.

3. Mathematical extrapolation models are not adequate in themselves for predictions of risks much beyond the experimental range, no matter how good the fit is to the data in the observed range. The variability of relatively small experimental groups adds to the error range. Thus, bioassays intended for quantitative risk assessment applications should be at as low doses as possible, and as large as possible, and should be interpreted very cautiously.

4. The current state of the art is such that quantitative risk assessments may be useful for determining relative risks from similarly acting carcinogens, but are not suitable for across-the-board application to all mechanisms of carcinogenesis.

   This is not to say that we should abandon efforts at developing more effective risk assessment methods. We must, however, recognize the problems inherent in blind application of mathematical models without proper assessment of the available biochemical data, or an understanding of how applicable the experimental data are to humans.

We have available to us at least as much data regarding vinyl chloride as we have for any other substance, and we still have difficulty in deriving a suitable expression for risk from a purely mathematical or statistical basis. Only when human relevance is considered can we arrive at a prediction that approximates actual experience.

The regulators are faced with a tremendously difficult task when they are presented with a few pieces of animal data which suggest the need for concern and potential regulation. We must develop a suitable program to obtain and use as much relevant data as possible to assure that rational regulations are possible. The vinyl chloride experience can help us understand the kind of data which are needed.

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FIGURE 1
GRAPHICAL REPRESENTATION OF TABLE III
LOG-PROBIT PLOT

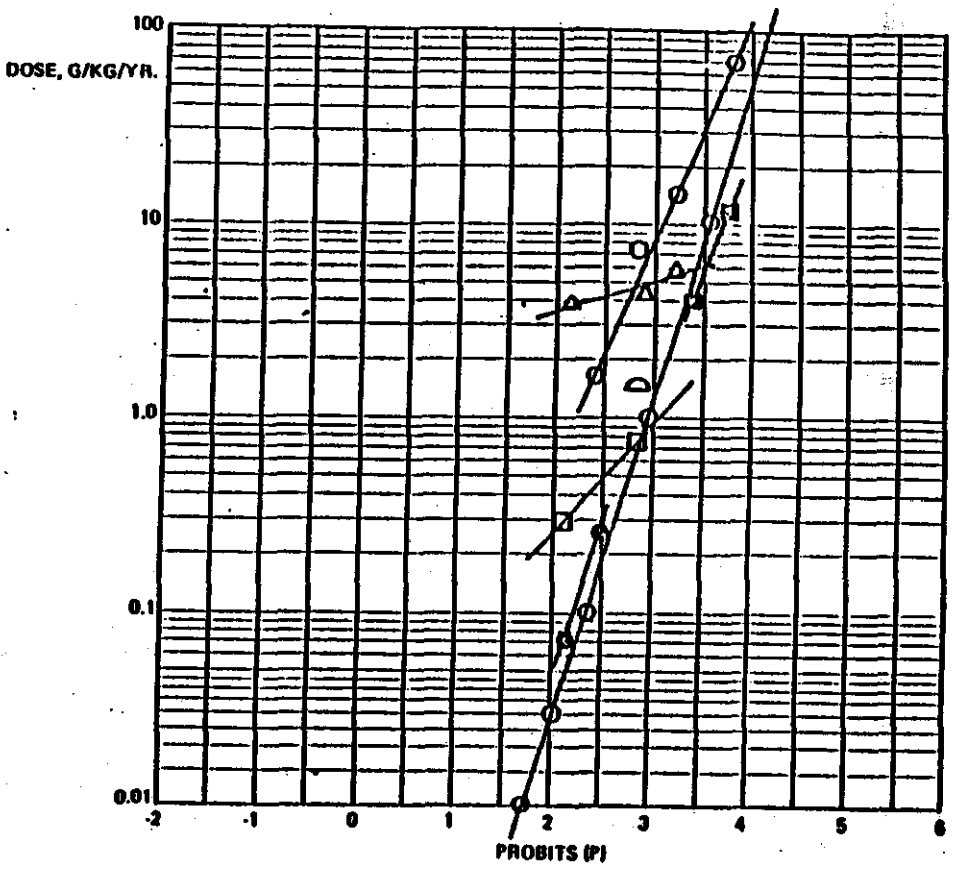

TABLE I
SUMMARY OF
QUANTITATIVE RISK ASSESSMENTS FOR VC

ESTIMATE NO.	AUTHOR	BASE SPECIES	EXPOSURE FOR 10^{-6} LIFETIME RISK	COMMENTS
1	SCHNEIDERMAN, 1975	RAT	73 ppb	LOG-PROBIT
			119 ppb	LOGIT SLOPE = 3.45
			2 ppb	LOGIT SLOPE 2.3, 1-HIT
2	KUSMACK & McGAUGHY, 1975	RAT, HUMAN	14 ppb	LINEAR THROUGH ZERO
			140-1400 ppb	LOG-PROBIT
3	GEHRING, 1975	RAT, HUMAN	>1 ppm	BIOTRANSFORMAL DATA AND LINEAR OR LOG-PROBIT
4	FOOD SAFETY COUNCIL, 1980	RAT	2×10^{-6} ppb	WEIBULL
6	HEHIR, 1980	RAT, MOUSE	THRESHOLDS SEEN IN BOTH SPECIES	
7	ANDERSON, 1980	RAT, HUMAN	>1 ppm	DNA BINDING
8	EPA, 1980	RAT	4 μG/DAY	FOOD OR WATER
9	NAS, 1980	RAT	3×10^{-5} MG/KG/DAY	WATER
10	GAYLOR & KODELL, 1980	RAT	0.7 ppb	UPPER 97.5% CONFIDENCE LIMIT OF LINEAR MODEL
			0.5 ppb	ARMITAGE-DOLL MODEL
11	CRUMP & GUESS, 1980	HUMAN	0.7 μG/DAY	APPLYING WORKER DATA TO WATER, UPPER 95%
		RAT	0.5 μG/DAY	CONFIDENCE LIMITS
13	CARLBORG, 1981	RAT	2.5×10^{-5} ppb	WIEBULL
14	THIS PAPER	HUMAN	>1 ppm	NEGATIVE EPIDEMIOLOGY

TABLE II
REGULATORY STATUS OF VINYL CHLORIDE

AGENCY	CONTROLLED LEVEL	PHILOSOPHY
BATF	BANNED AS LIQUOR BOTTLE	ADULTERANT
CPSC	BANNED IN CONSUMER PRODUCTS	ZERO RISK
FDA	USE IN RIGID FOOD PACKAGING QUESTIONED	CONSIDERING ACCEPTABLE RISK
EPA	APPROXIMATELY 0.2 ppb	BEST AVAILABLE TECHNOLOGY
OSHA	1 ppm 8-HR TWO MAXIMUM 0.5 ppm ACTION LEVEL	LOWEST FEASIBLE LEVEL

TABLE III
EQUATIONS FOR CURVES FITTED TO VARIOUS SINGLE AND COMBINED MALTONI EXPERIMENTS

EXPERIMENT	LINEAR y=ax + b			LOG PROBIT P=a IN DOSE + b			CONCENTRATION AT 10^{-6} RISK, (LOG-PROBIT), ppm
	a	b	r	a	b	r	
BT-1	0.26	3.36	0.97	0.35	2.76	0.99	0.03
PLUS CONTROLS	0.27	2.47	0.97				
BT-2	3.05	-8.59	1.0	1.60	0.88	1.0	23
PLUS CONTROLS	1.52	-1.13	0.88				
BT-15	6.5	-1.06	1.0	0.69	3.46	1.0	0.34
PLUS CONTROLS	5.28	-0.30	0.95				
ALL INHALATION STUDIES (4)	0.26	3.07	0.91	0.27	2.98	0.82	0.002
PLUS CONTROLS	0.27	2.80	0.91				
ALL INGESTION STUDIES (2)	1.75	0.15	0.95	0.30	3.22	0.88	0.002
PLUS CONTROLS	1.74	0.20	0.95				
ALL STUDIES (6)	0.29	3.60	0.73	0.27	3.05	0.82	0.001
PLUS CONTROLS	0.29	3.41	0.72				
ALL STUDIES; LOW DOSES ONLY				0.51	2.53	0.75	0.42
PLUS CONTROLS	1.03	0.97	0.79	0.17	2.71	0.49	0.0002

APPENDIX V

HEALTH EFFECTS REQUEST TO DHS AND LETTER OF RESPONSE

memorandum

To : Kenneth Kizer, Director
Department of Health Services
714 P Street
Sacramento, CA 95814

Date : June 17, 1985

Subject: Evaluation of Vinyl Chloride

From : James D. Boyd
Executive Officer
Air Resources Board

I am writing to request formally that the Department evaluate the health effects of vinyl chloride as a candidate toxic air contaminant in accordance with Assembly Bill 1807 (Tanner). According to Health and Safety Code Sections 39660-62, your Department has ninety days to submit a written evaluation and recommendations on the health effects of vinyl chloride to the Air Resources Board and may request a thirty day extension.

Attached for your staff's consideration in evaluating vinyl chloride are: Attachment I - a suggested list of topics that we believe should be included in your vinyl chloride evaluation and recommendations; Attachment II - a list of references on vinyl chloride health effects which were presented in an ARB letter of public inquiry; Attachment III - additional references and comments received from the public in response to the inquiry letter; and Attachment IV - ambient vinyl chloride concentration data and emission data which should be used to estimate the range of risk to California residents as required in Health and Safety Code Section 39660(c).

My staff is available for consultation in conducting this health effects evaluation. We look forward to continuing to work closely with you and your staff in carrying out this legislative mandate. If you have any further questions regarding this matter, please contact me at 445-4383.

Attachments

cc: Jananne Sharpless
Alex Kelter, w/attachments
Raymond Neutra, w/attachments
Peter D. Venturini
Assemblywoman Sally Tanner
Claire Berryhill
Emil Mrak, Chairman and Members
of the Scientific Review Panel
Senator Ralph Dills
Senator Art Torres
John Holmes ARB

ATTACHMENT IV A

SUMMARY OF AMBIENT VINYL CHLORIDE CONCENTRATIONS

Vinyl chloride has been produced in one industrial facility and used by four facilities in California, all of them in the South Coast Air Basin (SCAB). In May 1978, the Air Resources Board (ARB) adopted an ambient air quality standard for vinyl chloride of 10 ppb, 24-hour average. Subsequent ambient monitoring in the SCAB found the 10 ppb standard to be exceeded frequently in the vicinity of these facilities from 1979-1981. However, since 1982 the recent monitoring data for VC near these vinyl chloride facilities has shown all values to be below 10 ppb, without a determination of the actual value. These reductions in ambient concentrations are likely due to the closure of the production facility in 1982 and implementation of regulations by the South Coast Air Quality Management District (SCAQMD) designed to reduce vinyl chloride emissions.

Vinyl chloride has been detected in the community near the BKK Class I landfill in West Covina. In 1983, the Department of Health Services (DHS), ARB, and the South Coast Air Quality Management District issued a report detailing ambient concentrations (report attached). As the report indicates, the average vinyl chloride concentrations varied with location. The worst case residential location, Station A, had mean 24-hour VC concentrations of 7.1-7.3 ppb, with a maximum reading of about 39 ppb. Data for this report were collected over three months (July 19-October 15, 1982), with 24-hour samples taken five days per week.

A newly discovered potential source of vinyl chloride emissions into the air is that of sewage treatment facilities. An EPA contractor recently made some estimates of vinyl chloride emissions, as well as other volatile aromatic compounds, from the "Top 20" sewage treatment plants, nationwide. (Please see Appendix D of Versar Memorandum, Attachment IVC.) In this document, the Hyperion facility, which is located in the SCAB, was calculated to release 171 metric tons/year of vinyl chloride. ARB staff modeled this emission estimate (assumptions on Attachment IVB) and predicted 8 ppb above any background as an annual average vinyl chloride concentration. The 24-hour maximum VC concentration prediction is 23 ppb above background. ARB and SCAQMD plan to confirm these estimates with source and ambient vinyl chloride testing at the Hyperion facility in the summer of 1985.

Summary of the Health Effects of Vinyl Chloride

I. HEALTH EFFECTS

The health effects of vinyl chloride have been reviewed by several sources. Two good reviews are by the International Agency for Research on Cancer (IARC, 1979) and the U.S. Department of Health, Education and Welfare (U.S. HEW, 1978).

A. Carcinogenicity

1. Humans - Epidemiological studies have shown that vinyl chloride causes angiosarcoma of the liver in humans. Strong evidence also exists that vinyl chloride may cause cancer of the central nervous system, especially glioblastoma multiforme. Evidence also exists that vinyl chloride induces cancers of the lung and lymphatic system but this evidence is weaker. (IARC, 1979; U.S. HEW, 1978)

2. Animals - Vinyl chloride has been shown to be carcinogenic in several animal species after oral and inhalation administration. Liver angiosarcomas were observed in mice, rats and hamsters exposed to vinyl chloride. Other tumors seen were mammary adenocarcinomas, lung adenomas, Zymbal gland tumors and angiosarcomas at sites other than the liver. Doses in the inhalation experiments ranged from 50 to 10,000 ppm. A significant increase in some tumors (angiosarcomas) was seen at the low dose (50 ppm) level. (IARC, 1979; U.S. HEW, 1978)

B. Mutagenesis

Vinyl chloride is mutagenic in several test systems. Vinyl chloride has been found to be mutagenic in several strains of bacteria, insects and mammalian cells. Chromosomal aberrations have been induced in workers exposed to vinyl chloride. (IARC, 1982)

C. Teratogenicity

Evidence that vinyl chloride causes teratogenic effects in humans or animals is equivocal. Vinyl chloride has been implicated in causing increased fetal deaths in the wives of vinyl chloride exposed worker's and birth defects in children of workers. Evidence is inconclusive. (IARC, 1979)

D. Pharmacokinetics

The metabolism of vinyl chloride has been reviewed by several authors (IARC, 1979). Absorbed vinyl chloride is eliminated predominantly via metabolism and exretion of metabolites into the urine. A small amount is excreted via the expired air as unchanged vinyl chloride. As the concentration of vinyl chloride to which an animal is exposed is raised, a larger percentage of the absorbed dose is eliminated as unchanged vinyl chloride in the expired air. The initial product of metabolism is believed to be chloroethylene oxide. Vinyl chloride, in the presence of a microsomal enzyme fraction, binds to RNA *in vitro* and to RNA and DNA *in vivo*. Chloroethylene oxide is believed to be involved in the covalent

binding to RNA and DNA. Since an abundance of animal pharmacokinetic data exists, it may be possible to incorporate it into the dose-response assessment. (IARC, 1979)

E. Acute and Chronic Effects (non-carcinogenic)

Acute exposure to vinyl chloride causes narcosis, cardiac irregularites and liver and kidney toxicity. These effects are seen at relatively high doses. Liver toxicity is evident as centrilobular degeneration, hepatic fibrosis and necrosis. Degeneration of bone, nerves and connective tissue is seen after chronic exposure. Acroosteolysis, a degeneration of the bones in the fingers, occurs in workers. Disturbances in liver, kidney and pulmonary function also occur after chronic exposure.

II. THRESHOLD

The U.S. EPA proposed a National Emission Standard for vinyl chloride in 1975, which was promulgated in 1976. The proposal for the emission standard states that there is no known threshold for vinyl chloride's toxic effects. (Federal Register, 1975)

III. DOSE-RESPONSE ASSESSMENT

The U.S. EPA's Carcinogen Assessment Group has performed a risk assessment of vinyl chloride's carcinogenic effects (U.S. EPA, 1975). The potency slope for vinyl chloride, derived from an animal inhalation study, is $1.75 \times 10^{-2} (mg/kg/day)^{-1}$.

REFERENCES

Federal Register, 1975, Vinyl Chloride National Emission Standard for Hazardous Air Pollutants, FR 40:59532.

IARC, 1979, International Agency for Research on Cancer, IARC monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Volume 19, pages 377-438.

IARC, 1982, International Agency for Research on Cancer, IARC monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Supplement 4, pages 260 - 262.

U.S. EPA, 1975, Carcinogen Assessment Group, "Quantitative Risk Assessment for Community Exposure to Vinyl Chloride", Arnold M. Kuzmack and Robert E. McGaughy, December 5, 1975.

U.S. HEW, 1978, U.S. Department of Health, Education and Welfare, Vinyl Chloride: An Information Resource, DHEW Publ. No. (NIH) 78-1599

State of California — Department of Health Services

Memorandum

To : Peter Venturini
Chief, Stationary Sources Division
Air Resources Board
1102 Q Street
Sacramento, CA 95814

Date : JAN 25 1989

Subject: Health Effects of Vinyl Chloride

RECEIVED
JAN 30 1989
Stationary Source Division
Air Resources Board

From : Public Health
714 P Street
Sacramento, CA 95814
(916) 445-2927

Attached is the document prepared in response to your request for the assistance of the Department of Health Services in evaluating the health effects of vinyl chloride as a potential toxic air contaminant.

Harvey F. Collins, Ph.D.
Deputy Director

Attachment

cc: Jack C. Parnell, Director of Food & Agriculture
California Department of Food & Agriculture
1220 N Street
Sacramento, CA 95814

Jananne Sharpless, Chairwoman
Air Resources Board
P. O. Box 2815
Sacramento, CA 95812

Assemblywoman Sally Tanner
The State Capitol
P. O. Box 942849
Sacramento, CA 94249-0001

Copies of document can be requested from:

California Department of Health Services
Hazard Evaluation Section
2151 Berkeley Way, Room 515
Berkeley, CA 94704

APPENDIX VI

LANDFILL GAS TESTING PROGRAM

APPENDIX VI

LANDFILL GAS TESTING PROGRAM DATA

State law (Health and Safety Code Section 41805.5) requires owners or operators of all active and some inactive landfills to perform air quality solid waste assessment testing to characterize the gas within landfills and the ambient air around the landfills, and to determine if the landfill gas is migrating underground beyond the site boundaries. Ten specified air contaminants were selected to be tested for based on health effects associated with long term exposure; particularly carcinogenicity, and the availability of sampling and analysis methods. The ten specified contaminants were as follows: vinyl chloride, benzene, ethylene dibromide, ethylene dichloride, methylene chloride, perchloroethylene, carbon tetrachloride, 1,1,1-trichloroethane (methyl chloroform), trichloroethylene, and chloroform. In addition, landfill gas samples were also analyzed for oxygen, nitrogen, methane, and carbon dioxide.

To accomplish the testing required by state law, the Air Resources Board (ARB) and the California Air Pollution Control Officers Association (CAPCOA) prepared guidelines outlining a testing program to identify sites that pose a potential risk to public health. These guidelines were approved by the ARB for non-hazardous waste sites in December 1986 and for hazardous waste sites in January 1987.

State law requires landfill operators to report the testing results to their local air pollution control district. The districts, in turn, submit summaries of the testing results to the ARB and determine if the sites pose a threat to human health or the environment. The ARB was required to summarize the data submitted by the districts in two reports to the Legislature, due by July 1, 1988 and July 1, 1989. The first report described the early implementation of the landfill testing program. The second report, presented to the Board on June 9, 1989, summarized statewide results of the solid waste disposal site testing reported to the ARB. The report presented preliminary findings based on the results, and described ongoing testing and evaluation activities.

The preliminary findings were that: 1) one or more of the specified contaminants, selected as indicators of hazardous waste, were present in approximately 240 out of the 356 landfills tested, regardless of whether the site accepted hazardous waste or non-hazardous waste; 2) hazardous and non-hazardous waste sites appeared to be similar in their ability to produce toxic gases; 3) in some cases, toxic gases escaped from landfills and dispersed into the ambient air; and 4) methane at concentrations exceeding the regulatory standard of five percent was found to be migrating off-site underground at approximately 20 percent of the sites. The Board asked that the staff return in 1990 with further analysis of the data.

Since the 1989 report was presented, further analysis has been conducted and additional data has been collected. In September 1990, the further analysis will be presented to the Air Resources Board as an informational presentation. The following tables are excerpted from the ARB staff report and summarize the findings regarding the presence of vinyl chloride in the landfill gas and in the ambient air surrounding landfills tested. The landfill testing detected vinyl chloride inside of approximately half of the landfills tested and in the ambient air at approximately ten percent of the sites tested.

The limited testing conducted was designed to be used for screening purposes as described in the testing guidelines. For that reason, vinyl chloride may be present in the ambient air at additional landfills, but was not detected in the limited one to three days of ambient testing specified in the testing guidelines for the program. Further interpretation of the data from specific sites must also consider factors such as how the testing was carried out, along with location, size and proximity to sensitive receptors. Further information may also be available in the complete testing reports submitted by site operators to the air pollution control districts.

TABLE 1

CONCENTRATION STATISTICS OF VINYL CHLORIDE IN LANDFILLS*

Number of Landfills Where Detected:	160
Total Landfills Tested:	340
50th percentile (median):	106 ppbv (detection limit)**
75th percentile:	1000 ppbv
95th percentile:	9800 ppbv
Maximum:	72,000 ppbv

* See Attachment A for landfill gas testing guidelines.

** The Testing Guidelines for Active Solid Waste Disposal Sites suggest an analytical procedure for internal landfill gas testing with the same method of determining the limit of detection (LOD) described in Appendix VII of this document. The Guidelines specify that the detection limit is not to exceed 500 ppbv. Because many results below 500 ppbv were measured and reported, a statistical detection limit of 106 ppbv was calculated by averaging the results below 500 ppbv.

Landfill Gas Testing

If the disposal site has an operating interior gas collection system, samples should be taken from the system; additional wells need not be installed. Each installed well should be to a depth of at least 6 feet below the bottom of the intermediate or final cover. The well should not penetrate any leachate liner. During installation the contractor should take appropriate steps to mitigate the public nuisance of gas escape. All wells should be capped when not being sampled.

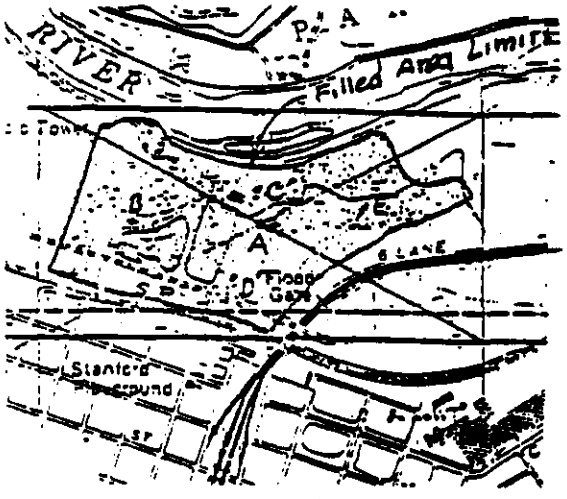

Figure 1: Well Location Example

To locate the wells, draw a box around the disposal site on a scale map with the box sides 100 feet outside the filled area edge. The sides should run north-south, east-west. Connect the opposite corners with diagonals. Locate 5 points: Point A at the diagonal intersection, point B at the center of the largest sector formed by the diagonals and the filled area, point C at the center of the next larger sector, point D at the center of the next larger sector, and point E at the center of the smallest sector. Figure 1 is an example. Five samples should be taken, one sample from each well and analyzed for the Attachment 1 compounds.

To complete the HSC 41805.5 requirements for characterizing landfill gas, the owner should perform an investigation of methane emissions from one 50,000 square-foot grid of the disposal site along with the landfill gas test. The grid selected should be approved by the APCO and the owner should use methods described in these guidelines.

1. Protocol

The technician should make certain the seal around the top of the well does not allow air infiltration. The well should not be sampled until 24 hours after the installation is complete. To sample the well, the technician attaches the pump and withdraws at least 2 well volumes from the well. The technician then attaches the bag and draws a ten liter sample at a one liter per minute rate. The bag should be in a light sealed container and should be analyzed within 72 hours.

If the owner chooses to leave the well intact for future sampling, the pipe should be capped or a valve installed to prevent gas leakage. If the owner removes the well, the hole should be filled and resealed to prevent gas escape.

2. Data

For each sample, the owner should record:

a. Date, time, and sample location.

b. Methane, CO_2, oxygen, and nitrogen concentrations.

c. Concentrations of compounds listed in Attachment 1. Analytical methods are included in Attachment 2.

d. The operating schedule, status, and gas quantity extracted for any landfill gas collection system for the previous 3 days for each day sampled.

ATTACHMENT 1

SPECIFIED AIR CONTAMINANTS

COMPOUND		Detection Limits, ppb Air	Disposal site
Chloroethene (Vinyl Chloride)	CH_2:CHCl	2	500
Benzene	C_6H_6	2	500
1,2-Dibromoethane (Ethylene Dibromide)	$BrCH_2CH_2Br$	0.5	1
1,2-Dichloroethane (Ethylene Dichloride)	$ClCH_2CH_2Cl$	0.2	20
Dichloromethane (Methylene Chloride)	CH_2Cl_2	1	60
Tetrachloroethene (Perchloroethylene)	Cl_2C:CCl_2	0.2	10
Tetrachloromethane (Carbon Tetrachloride)	CCl_4	0.2	5
1,1,1-Trichloroethane (Methyl Chloroform)	CH_3CCl_3	0.5	10
Trichloroethylene	HClC:CCl_2	0.6	10
Trichloromethane (Chloroform)	$CHCl_3$	0.8	2

ATTACHMENT 2

The choice of analytical method is left up to the individual laboratory performing the analysis. The methods provided in Attachment 2 are provided as examples of methods which can be used to sample and analyze for the specified air contaminants identified in Attachment 1. The methods are used by ARB laboratories to quantify the compounds listed *at or below the detection limits specified in Attachment 1.* Table 2-1 summarizes the method detection limits achievable by these methods and the detection limits to be reported for these guidelines:

TABLE 2-1: METHOD DETECTION LIMITS

COMPOUND	Guideline	Method Detection Limits, ppb Haagen-Smit Laboratory	Aerometric Data Division
Chloroethene (Vinyl Chloride)	2	-	1
Benzene	2	0.5	0.5
1,2-Dibromoethane (Ethylene Dibromide)	0.5	0.01	0.005
1,2-Dichloroethane (Ethylene Dichloride)	0.2	0.2	0.1
Dichloromethane (Methylene Chloride)	1	1	0.6
Tetrachloroethene (Perchloroethylene)	0.2	0.004	0.01
Tetrachloromethane (Carbon Tetrachloride)	0.2	0.02	-
1,1,1-Trichloroethane (Methyl Chloroform)	0.5	0.004	0.004
Trichloroethylene	0.6	0.005	0.02
Trichloromethane (Chloroform)	0.8	0.004	0.02

AMBIENT AIR MONITORING

HSC 41805.5 requires that air adjacent to disposal sites be tested and analyzed for specified air contaminants. To comply with HSC 41805.5, disposal site owners should conduct ambient air monitoring at the perimeter of the disposal site. The test should adequately characterize the contaminants in the air. The air column listed in Attachment 1 shows the lower detection limits to be achieved in parts per billion. Each disposal site should perform the ambient air sampling on three separate, not necessarily consecutive, days.

At sites where the owner has chosen to characterize only the gas above the disposal site using the integrated surface sampling technique, all specified air contaminants must be tested and analyzed for in the air samples. A site where landfill gas testing is used *and* where chloroethene (vinyl chloride) is identified in the landfill gas, then the ambient air samples need only be tested for chloroethene (vinyl chloride).

The guidelines contain three suggested procedures for testing the ambient air. These procedures were developed to cover differences in topography and climate which may occur at different sites. Each option has two parts. One addresses sites with different day and night wind patterns and one addresses sites with the same day and night wind patterns. The option chosen will depend on the results of the meteorological survey.

A. OPTION 1

1. General Procedures

HSC 41805.5 requires that air adjacent to disposal sites be tested and analyzed for specified air contaminants. If the disposal site has a gas collection system which does not operate continuously, at least one of the sampling days should be a day before the gas collection system is turned on after a typical inoperative period. This option requires twenty-four hour samples to be taken on 3 separate, not necessarily consecutive, days.

2. Meteorological Survey

A meteorological survey should be conducted prior to ambient air sampling in order to determine the local wind flow patterns which will subsequently be used to help identify the number and location of samplers required for an effective ambient air monitoring program. The operator should submit the survey to the APCO prior to ambient sampling, as part of the monitoring plan. The survey should summarize how wind flow patterns at the site will be characterized based on: previously collected on site meteorological data, data collected nearby (e.g., local airport data), proximity to water or terrain which may influence diurnal variations (e.g., daytime upslope winds, nighttime downslope, or sea breeze conditions), or a plan for on site meteorological data collection prior to ambient monitoring. In completing an on site meteorological survey prior to monitoring, wind sensors should be located nine to twelve feet above the ground and a minimum of sixty feet from obstacles such as trees, shrubbery, and buildings.

3. Ambient Air Sampling

a. General Sampling Criteria

At the completion of the meteorological survey, and on approval of the APCO, ambient air

TABLE 2

CONCENTRATION STATISTICS OF VINYL CHLORIDE IN AMBIENT AIR SAMPLES COLLECTED AT THE PERIMETERS OF LANDFILLS*

Number of Landfills Where Detected:	24
Total Landfills Tested:	251
50th percentile (median):	2 ppbv (detection limit)
75th percentile:	2 ppbv
95th percentile:	2 ppbv
2nd highest value:	13 ppbv
Maximum:	15 ppbv

* See Attachment B for ambient air testing guidelines.

sampling equipment will be installed at the appropriate locations which will be determined by:

1. Site topography,

2. Meteorological survey, and

3. Local land use patterns.

The sampling equipment should be located at or near the perimeter of the waste disposal site, in the clear and away from surrounding obstructions. The inlet probes for the ambient samplers should be located between six and nine feet off the ground (reaching height) and a minimum of sixty feet from obstacles such as trees, shrubbery and buildings. Air flow around the inlet probe should be unrestricted in an arc of at least 270 degrees with the predominant wind direction for greatest expected. pollutant concentration potential included in the 270 degree arc. The sampler locations should be carefully selected to ensure the predicted prevailing wind patterns for the sampling date will come across the main body of the disposal site to the downwind station. Wind speed and direction measurements will continue to be collected throughout the ambient air sampling period to verify that the meteorological criteria are met.

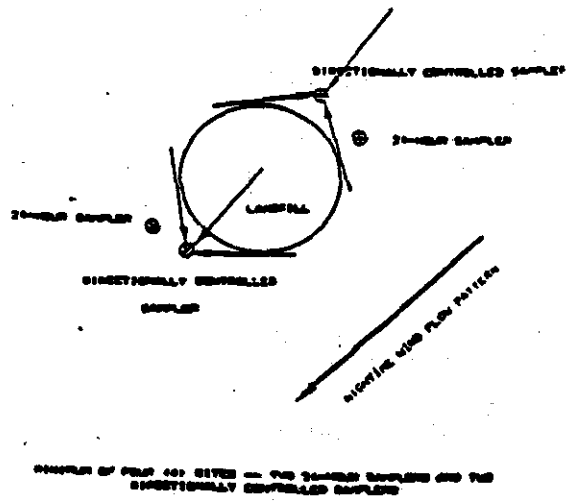

Figure 4: Option 1
Source: South Coast AQMD

Ambient air samples will be collected over a 24-hour period beginning and ending at 10:00 A.M. using the self-contained portable sampling units described in Equipment Description. In general, 24-hour and directionally controlled sampling will be required to ensure that maximum contaminant concentrations are identified for each sampling period. However, directionally controlled sampling may not be required at sites which have a constant wind direction for 24 hours. All samples will be removed from the samplers immediately after the 24-hour sampling period and analyzed for the required compounds. It is recommended that the sample be analyzed within 72 hours of collection.

b. Specific Sampling Criteria

i. At sites that experience different day and night wind flow patterns, a minimum of two 24-hour samplers and two directionally controlled samplers will be required. Twenty-four hour samplers will be placed at the upwind and downwind site perimeters based on the

prevailing wind direction. The directionally controlled sampler(s) located downwind of the disposal site should be placed at sites which will sample under the stable (drainage) wind conditions identified in the meteorological survey. The directionally controlled sampler located upwind of the disposal site should be placed near the upwind 24-hour sampler. The 24-hour samplers will operate continuously for the specified 24 hours and the directionally controlled samplers will only operate when the wind direction is within a wind sector allowing air to pass across the disposal site to the downwind sampler. This will allow the downwind directionally controlled sampler(s) to only collect air that has *passed* over the disposal site and the upwind directionally controlled sampler to only collect air that has *not passed* over the disposal site.

ii. At site that experience a constant wind direction for 24 hours, a minimum of two 24-hour samplers will be required. A 24-hour sampler will be place both upwind and downwind of the site based on the prevailing wind direction so that the upwind sampler only collects air that has *not passed* over the disposal site and the downwind sampler only collects air that has *passed* over the disposal site. Additional 24 hour samplers should be placed at locations which will sample under the stable (drainage) wind conditions identified in the meteorological survey. Since the wind direction does not change, these 24-hour samplers will act as directionally controlled samplers as well as 24-hour samplers. Comparison of the results from these samplers will provide information on ambient air quality standards and the effects the disposal site has on the ambient air quality.

4. Sampling Conditions

Ambient air sampling should be conducted on days when stable and unstable meteorological conditions are characterized by the following meteorological conditions:

a. Stable nights with average wind speeds of five miles per hour or less.

b. Daytime conditions with average wind speeds of ten miles per hour or less.

No sampling will be conducted under the following adverse meteorological conditions:

a. Precipitation

b. Twenty-four hour average wind speeds greater than ten miles per hour.

5. Equipment Description

a. Bag Sampler

1. Pump with a diaphragm made of non-lubricated Viton® rubber. The maximum pump unloaded flow rate is 4.5 liters per minute.

2. One 10-liter Tedlar® bag with a push-pull valve constructed of aluminum and stainless steel with a Viton® o-ring seal.

3. Rotameter made of borosilicate glass with a flow range of three to fifty cubic centimeters per minute. The scale is in millimeters with major graduations (labeled) every 5 mm and minor graduations every 1 mm.

4. Air flow control orifice made with 316 stainless steel capillary tubing.

5. Bypass valve.

6. Fittings, tubing and connectors made with 316 stainless steel or teflon.

7. Clock timer with an accuracy that should be better than 1%.

b. Wind directionally controlled system

1. Wind direction sensor with a vane which has a range of 0 - 540 degrees and a threshold of 1.00 mile per hour or less.

2. Controller and indicator console with an indicator range of 0 - 360 degrees and an accuracy of ± 2% of full scale.

c. Wind speed and direction monitoring with continuous recorder.

1. Anemometer three cup assembly with a range of 0-50 miles per hour and a threshold of 0.75 miles per hour or less.

2. Wind vane with a range of 0 - 540 degrees and a threshold of 1.00 miles per hour or less.

6. Wind Data Reporting

Wind data (speed and direction) will be reported as an hourly average. For example, the data collected between 1:00 P.M. and 2:00 P.M. will be averaged and reported as the 1:00 P.M. hourly average. Wind speeds will be reported in miles per hour. Wind directions will be reported using the sixteen point scale (sixteen directional points corresponding to the mariner's compass rose on which each direction is equivalent to a 22 1/2 degree sector of a 360 degree circle). For example, wind directions would be N, NNE, NE, E ESE, SE, SSE, S, SSW, SW, WSW, W, WNW, NW, and NNW.

B. OPTION 2

1. General Procedures

HSC 41805.5 requires that air adjacent

See Option 1.

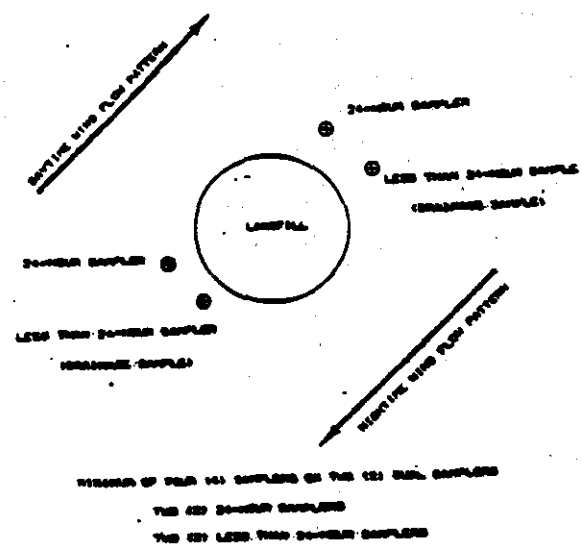

Figure 5: Option 2
Source: South Coast AQMD

to disposal sites be tested and analyzed for specified air contaminants. These guidelines require that 24-hour and less than 24-hour ambient air sampling be conducted on three different, not necessarily consecutive, days.

2. Meteorological Survey

See Option 1.

3. Ambient Air Sampling

See Option 1, Subsection 3a, General Sampling Criteria.

a. At sites that experience different but predictable day and night wind flow patterns, a minimum of two 24-hour samplers and two less than 24-hour samplers will be required. One 24-hour sampler will be placed both upwind and downwind of the site based on the prevailing wind direction. The less than 24-hour sampler(s) located downwind of the disposal site should be placed at sites to sample under the stable (drainage) wind conditions identified in the meteorological survey. The less than 24-hour sampler located upwind of the disposal site should be placed near the upwind 24-hour sampler. The start and stop times for the less than 24-hour samplers will correspond to the stable (drainage) conditions identified by analyzing the the hourly wind roses. The 24-hour samplers will operate continuously for the specified 24 hours and the less than 24-hour samplers will only operate when the wind direction is coming across the disposal site to the downwind sampler. This will allow the downwind less than 24-hour sampler(s) to only collect air that has *passed* over the disposal site and the upwind less than 24-hour sampler to only collect air that has *not passed* over the disposal site.

b. At sites that experience a constant wind direction for 24 hours, a minimum of two 24-hour samplers will be required. A 24-hour sampler will be place both upwind and downwind of the site based on the prevailing wind direction so that the upwind sampler only collects air that has *not passed* over the disposal site and the downwind sampler only collects air that has *passed* over the disposal site. Additional 24 hour samplers should be placed at locations which will sample under the stable (drainage) wind conditions identified in the meteorological survey. Since the wind direction does not change, these 24-hour samplers will act as directionally controlled samplers as well as 24-hour samplers. Comparison of the results from these samplers will provide information on ambient air quality standards and the effects the disposal site has on the ambient air quality.

4. Sampling Conditions

See Option 1.

5. Equipment Description

See Option 1.

6. Wind Data Reporting

See Option 1.

C. OPTION 3

1. General Procedures

HSC 41805.5 requires that air adjacent to disposal sites be tested and analyzed for specified air contaminants. These guidelines require that 24-hour ambient air sampling be conducted on three different, not necessarily consecutive, days.

2. Meteorological Survey

See Option 1.

3. Ambient Air Sampling

See Option 1, Subsection 3a, General Sampling Criteria.

a. At sites that experience different day and night wind flow patterns, a minimum of three 24-hour samplers will be required. One 24-hour sampler will be placed on both upwind and downwind of the site based on the prevailing wind direction. Additional 24 hour samplers will be located downwind of the disposal site at sites which will sample under the stable (drainage) wind conditions identified in the meteorological survey. In addition, one 24-hour sampler will be placed in the vacinity of the disposal site, approximately one mile away, so it will not be affected by the disposal site emissions. This 24-hour sampler should also be approximately one mile away from other possible major emission sources so that the sample it collects will represent the background concentrations for the area. This background sampler whould be located in the clear and away

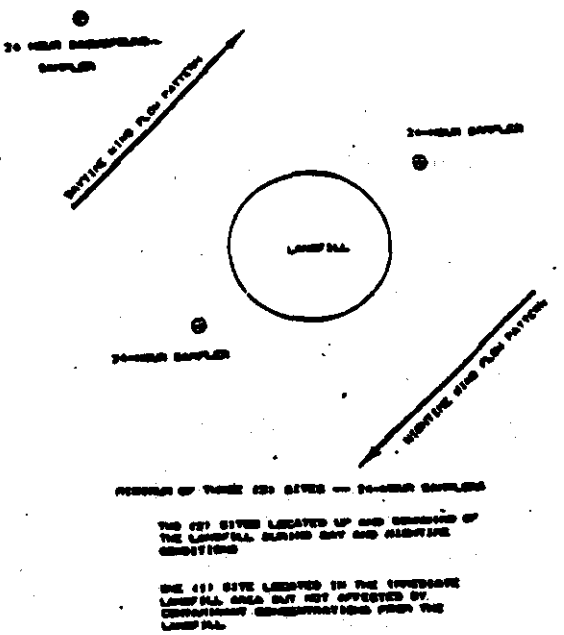

Figure 6: Option 3
Source: South Coast AQMD

from surrounding obstructions. Its inlet probe must be located between six and nine feet off the ground (breathing height) and a minimum of 60 feet from obstacles such as trees, shrubbery, and buildings. Air flow around the inlet probe must be unrestricted. All of the 24-hour samplers will operate continuously for the specified 24 hours. Comparison of the results from the samplers will provide information on the ambient air quality standards.

b. At sites that experience a constant wind direction for 24 hours, a minimum of two 24-hour samplers will be required. A 24-hour sampler will be placed both upwind and downwind

of the site based on the prevailing wind direction so that the upwind sampler only collects air that has *not passed* over the disposal site and the downwind sampler only collects air that has *passed* over the disposal site. Additional 24-hour samplers should be placed at locations which will sample under the stable (drainage) wind conditions identified in the meteorological survey. Since the wind direction does not change, these 24-hour samplers will act as less than 24-hour samplers as well as 24-hour samplers. In addition, one 24-hour sampler will be placed in the vicinity of the disposal site, approximately one mile away, so it will not be effected by the disposal site emissions. This 24-hour sampler should also be approximately one mile away from possible major emission sources so that the sample it collects will represent the background concentrations for the area. This background sampler should be located in the clear and away from surrounding obstructions. Its inlet probe should be located between six and nine feet off the ground (breathing height) and a minimum of sixty feet from obstacles such as trees, shrubbery and buildings. Air flow around the inlet probe should be unrestricted. All of the 24-hour samplers will operate continuously for the specified 24 hours.

4. Sampling Conditions

See Option 1.

5. Equipment Description

See Option 1.

6. Wind Data Reporting

See Option 1.

D. GENERIC ANALYTICAL METHODS

HSC 41805.5 directs the ARB to publish testing guidelines "specifying air contaminants to be tested for and identifying acceptable testing, analytical and reporting mehtods. The following generic analytical methods contain a brief description of the standard operating procedures (SOP) used by the ARB to sample and analyze specific compounds. Specific SOPs are contained in Attachment 2.

1. Method for Vinyl Chloride

Ambient samples are collected over a 24-hour period in a thirty liter Tedlar® bag using a low-volume sampler.

Samples are analyzed using chromatography with Flame Ionization or Photo Ionization Detection and preconcentration techniques. Resultant concentration peak is identified by retention times and quantified by reference to calibration standards.

2. Method for Carbon Tetrachloride, Chloroform, Ethylene Dibromide, Ethylene Dichloride. Methyl Chloroform, Methylene Chloride, Perchloroethylene, and Trichloroethylene

Ambient samples are collected over a 24-hour period in a thirty liter Tedlar® bag using a low volume sampler.

Samples are analyzed using-gas chromatography with Electron Capture Detection and preconcentration techniques. Resultant concentration peaks are identified by retention times and quantified by references to calibration standards.

ATTACHMENT 1

SPECIFIED AIR CONTAMINANTS

COMPOUND		Detection Limits, ppb Air	Disposal site
Chloroethene (Vinyl Chloride)	CH_2:CHCl	2	500
Benzene	C_6H_6	2	500
1,2-Dibromoethane (Ethylene Dibromide)	$BrCH_2CH_2Br$	0.5	1
1,2-Dichloroethane (Ethylene Dichloride)	$ClCH_2CH_2Cl$	0.2	20
Dichloromethane (Methylene Chloride)	CH_2Cl_2	1	60
Tetrachloroethene (Perchloroethylene)	Cl_2C:CCl_2	0.2	10
Tetrachloromethane (Carbon Tetrachloride)	CCl_4	0.2	5
1,1,1-Trichloroethane (Methyl Chloroform)	CH_3CCl_3	0.5	10
Trichloroethylene	HClC:CCl_2	0.6	10
Trichloromethane (Chloroform)	$CHCl_3$	0.8	2

ATTACHMENT 2

The choice of analytical method is left up to the individual laboratory performing the analysis. The methods provided in Attachment 2 are provided as examples of methods which can be used to sample and analyze for the specified air contaminants identified in Attachment 1. The methods are used by ARB laboratories to quantify the compounds listed *at or below the detection limits specified in Attachment 1.* Table 2-1 summarizes the method detection limits achievable by these methods and the detection limits to be reported for these guidelines:

TABLE 2-1: METHOD DETECTION LIMITS

COMPOUND	Guideline	Method Detection Limits, ppb	
		Haagen-Smit Laboratory	Aerometric Data Division
Chloroethene (Vinyl Chloride)	2	-	1
Benzene	2	0.5	0.5
1,2-Dibromoethane (Ethylene Dibromide)	0.5	0.01	0.005
1,2-Dichloroethane (Ethylene Dichloride)	0.2	0.2	0.1
Dichloromethane (Methylene Chloride)	1	1	0.6
Tetrachloroethene (Perchloroethylene)	0.2	0.004	0.01
Tetrachloromethane (Carbon Tetrachloride)	0.2	0.02	-
1,1,1-Trichloroethane (Methyl Chloroform)	0.5	0.004	0.004
Trichloroethylene	0.6	0.005	0.02
Trichloromethane (Chloroform)	0.8	0.004	0.02

APPENDIX VII

ARB MONITORING AND LABORATORY DIVISION'S METHOD FOR CALCULATING THE LIMIT OF DETECTION

Analytical Limits of Detection (LOD) must be calculated. The LOD for each method must be calculated by the following equation (reference):

$$LOD = A + 3S$$

where

A is the least squares intercept calculated from the multipoint data (section 4.1.2).

S is the standard deviation of replicate determinations of the lowest standard. At least 3 replicates are required. The lowest standard must be run at 1 to 5 times the estimated detection limit. If data is not available in the concentration range near the detection limit, S may be estimated by:

$$S = RSD \times A$$

where RSD is the relative standard deviation of the lowest standard analyzed.

The equation as listed above was obtained from the Compendium of Methods for the Determination of Toxic Organics in Ambient Air. Research Triangle Park, North Carolina: U.S. Environmental Protection Agency; 1984 April: Method T)1. Publication No. EPA-600/4-84-041.

Note that the Laboratory Services Section policy is to report all analysis results above the analytical limits of detection. However, data errors may approach $\pm$ 100% at levels < 10 x LOD.

All analysis methods must be written in detail as a Standard Operating Procedure to be used in the laboratory. Any subsequent revisions or improvements are documented. The procedures are reviewed yearly by laboratory management and the Quality Assurance Section to insure that they are being followed properly.