Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions From the Class I Unit of the B.K.K. Landfill, West Covina, California

April, 1996

Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

ADDENDUM HEALTH RISK ASSESSMENT OF AMBIENT FUGITIVE VINYL CHLORIDE EMISSIONS FROM THE CLASS I UNIT OF THE B.K.K. LANDFILL, WEST COVINA, CALIFORNIA

APRIL 1996
FINAL

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Responsibility for the contents of this report resides solely with OEHHA-HWTS.

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Office of Environmental Health Hazard Assessment

HAZARDOUS WASTE TOXICOLOGY SECTION

Addendum Health Risk Assessment, B.K.K. Landfill, West Covina, California

FACT SHEET

APRIL, 1996

This fact sheet summarizes the Office of Environmental Health Hazard Assessment's (OEHHA) "Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions From the Class I Unit of the B.K.K. Landfill, West Covina, California."

The addendum report is an update of the 1990 interim health risk assessment conducted by the Hazardous Waste Toxicology Section (HWTS).

In 1981, vinyl chloride was found in ambient air in residential neighborhoods adjacent to the B.K.K. Landfill. This is of concern because vinyl chloride is a known human carcinogen and is not normally detected in the South Coast air basin.

Emissions of vinyl chloride are clearly associated with the Class I Unit of the landfill. Wastes containing vinyl chloride were deposited in the Class I Unit until June 1981. Emissions from the now closed Class I Unit are due to vinyl chloride-containing wastes deposited in the Unit and to the breaking down of other wastes containing chlorinated hydrocarbons. The emissions have been significantly reduced over time.

OEHHA's health risk assessment is possible because there were monitoring data collected by a regulatory agency, in residential neighborhoods almost daily, for nearly a decade.

The B.K.K. Landfill

In 1963 the B.K.K. Landfill, opened on 130 acres in the San Jose Hills in the City of West Covina. In 1971 the B.K.K. Landfill expanded to its present size of 583 acres. In 1972 40 acres of the landfill were permitted as a hazardous waste (Class I) unit. The Class I Unit was expanded to 140 acres in 1975. In November 1984, the B.K.K. Class I Unit stopped accepting hazardous waste and in March 1989, was formally closed. The closing process included an extensive landfill gas collection system and the placing of a low-permeability soil cap over 170 acres.

This cap covered the hazardous waste landfill and part of one adjacent Class III (solid waste) landfill. A separate Class III waste area of the B.K.K. Landfill is still permitted and is currently accepting waste.

In 1985 the State of California committed to complete three health studies of the B.K.K. Landfill: a cancer study, a reproductive outcomes study and a health risk assessment. In 1990, the cancer study, an initial pregnancy outcomes study and the interim health risk assessment were completed. A more comprehensive reproductive outcomes study is currently being prepared by the Department of Health Services (DHS).

At the time the interim report was being released to the public, the cancer potency of vinyl chloride was being reevaluated and a more potent value was expected. This would make the cancer risk estimated in the interim report an underestimate.

The final addendum report plus the interim report will complete the health risk assessment of the B.K.K. Landfill promised to the residents by the State of California.

Addendum Objective

The addendum report estimates the excess cancer risk associated with exposures to vinyl chloride detected at three monitoring stations located south-east and south of the closed Class I Unit of the B.K.K. Landfill.

Methodology

The addendum report uses the four components of a health risk assessment: hazard identification, toxicity assessment, exposure assessment, and risk characterization.

Hazard Identification

Vinyl chloride is the chemical of primary concern. Although other chemicals have been detected at the monitoring stations, these chemicals are also found in the South Coast air basin. It is not possible to tell how much of these chemicals are from the B.K.K. Landfill and how much are from other sources in the area.

Toxicity Assessment

To estimate the ability of vinyl chloride to cause cancer, OEHHA evaluated data from studies in humans, exposed at factories that made or used vinyl chloride, and from studies in laboratory animals (rats, mice, and hamsters).

The cancer risk is estimated using the cancer potency value for vinyl chloride, 20×10^{-3} per part per billion (ppb). The cancer potency is an estimate of the probability of developing cancer as a result of daily exposure for 70 years to one ppb of a chemical in air.

Exposure Assessment

The vinyl chloride monitoring data were collected by the South Coast Air Quality Management District (SCAQMD) from June 1981 - March 1990, and by the B.K.K. Corporation from September 1993 - September 1994.

Station A was located at Marlena & Nogales Streets, Station B at Lynn Court and Station MY at Myra Court. These areas were exposed to the highest detected levels of vinyl chloride. Although other residential areas near the B.K.K. Landfill were exposed to emissions from the landfill, the levels were not as high or as frequent.

Risk Characterization

Assuming that individuals lived near the monitoring stations for 30 years (1980-2009; a "reasonable maximum exposure scenario"), OEHHA estimates that there is an added 0.006 to 0.009 percent chance of developing cancer as a result of exposure to emissions of vinyl chloride from the closed Class I Unit of the B.K.K. Landfill.

Addendum Findings

There are four findings of the addendum report:

1. For individuals who lived near the monitoring stations during the 1980's, past exposures were higher than current exposures. The majority of the estimated excess cancer risk was incurred by 1983-1984.
2. The landfill gas collection system and the clay cap covering the closed Class I Unit have been successful in lowering vinyl chloride emissions to levels that pose little, if any, risk to nearby residents.
3. Regulatory agencies may want to include past exposures of vinyl chloride near the monitoring stations in subsequent health assessments of emissions from the landfill to ensure future risks do not add significantly to the past cumulative risk.
4. There is uncertainty associated with the assumptions used in the addendum report. The actual excess cancer risk is likely to be lower than these estimates and may be zero. Because the number of people who actually lived or are living near Stations A, B, or MY is small, it is unlikely that excess cases of cancer will occur. Each exposed individual may have an increased risk of developing cancer, however the added risk is small.

Questions and Answers

Q - What is the difference between the DHS Cancer Study and the OEHHA Health Risk Assessments?

A - The cancer study investigated cases of diagnosed cancers for the years 1972-82. The health risk assessments estimate the theoretical probability, or risk, of developing cancer.

Q - What is meant by "estimated excess lifetime cancer risk"?

A - "Estimated excess lifetime cancer risk" is an estimate of the risk for an individual above the background risk of developing cancer during a 70-year lifetime. An individual living in the United States has a 40 percent (0.4) chance of developing cancer throughout a 70-year lifetime. An individual living from 1980-2009 near Station A, for example, may have an added 0.007 percent chance of developing cancer as a result of emissions of vinyl chloride from the closed class I Unit of the B.K.K. Landfill.

Q - Has there been an increase in diagnosed cases of cancer since 1982?

A - No, the DHS Cancer Surveillance Section recently reviewed the 1988-93 tumor incidence data for a one mile area around the landfill. DHS's review found no indication of increased cancer incidence.

Copies of the final addendum report are available at the following address.

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Copies of the cancer study, the pregnancy outcomes study and the health risk assessments are available at the West Covina, Walnut and La Puente libraries.

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EXECUTIVE SUMMARY

This report is prepared as an addendum to the interim report, "Health Risk Assessment of the B.K.K. Landfill, West Covina, California. November 1990" (Department of Health Services-Hazardous Waste Toxicology Section [DHS-HWTS], 1990), and presumes the reader is familiar with the interim report. The format follows the four components of a health risk assessment: hazard identification, toxicity assessment, exposure assessment, and risk characterization. This report is an addendum because at the time the interim report was being released to the public, the carcinogenic potency of vinyl chloride was being reevaluated, and a more potent value was expected to be derived. The interim report stated that an addendum would be prepared which would use the revised potency value and additional ambient vinyl chloride monitoring data.

The objective of this addendum report is to quantitatively estimate the excess lifetime cancer risk associated with exposures to vinyl chloride near residential monitoring Stations A, B, and MY, located southeast and south of the closed Class I (hazardous waste) Unit of the B.K.K. Landfill.

In May 1981, vinyl chloride was first detected in excess of the state's ambient air quality standard of ten parts per billion (10 ppb) at the landfill perimeter. The interim report and this addendum report primarily focused on exposures to vinyl chloride because vinyl chloride is a known human carcinogen, and it is rarely detected in ambient air of the South Coast air basin. Vinyl chloride has clearly been shown to be associated with the Class I Unit of the B.K.K. Landfill.

The health risk assessment, conducted by the Office of Environmental Health Hazard Assessment (OEHHA), is possible because emissions of vinyl chloride reaching the residential community near the B.K.K. Landfill have been quantitatively studied since June 1981. The monitoring data were collected by a regulatory agency in residential neighborhoods almost daily for nearly a decade.

Other hazardous chemicals are known to be emitted from the Class I Unit of the B.K.K. Landfill. However, no long-term monitoring of those compounds in air around the B.K.K. Landfill was undertaken, and so evaluation of any exposure to those chemicals is difficult. Those chemicals are also found in background air in the air basin, and it is not possible, with the available data, to distinguish between the amount of those chemicals emitted from the landfill and the amount found in ambient air from other sources.

A summary of the sections of this report is given below.

HAZARD IDENTIFICATION: The B.K.K. Landfill opened in 1963. Wastes were classified in statute as "hazardous" and "nonhazardous" by the State of California in 1972. From 1972 until December 1984, the B.K.K. Landfill served as the primary commercial hazardous waste disposal site for the Los Angeles area. The Class I (hazardous waste) Unit of the landfill stopped accepting hazardous waste on November 30, 1984, and was closed in 1989. The B.K.K. Corporation has been operating a municipal solid waste landfill adjacent to the closed Class I Unit since 1987. Emissions of vinyl chloride from the closed Class I Unit are due to biodegradation of buried wastes containing chlorinated hydrocarbons and deposition of hazardous waste containing vinyl chloride. Other hazardous chemicals detected in ambient air are discussed in this section and in the exposure assessment section.

TOXICITY ASSESSMENT: Information is presented about scientific advances in the understanding of risks associated with exposure to vinyl chloride. Both epidemiologic (occupational) and experimental animal data are reviewed and used in this assessment. Vinyl chloride is classified by both the United States Environmental Protection Agency (USEPA) and the International Agency for Research on Cancer (IARC) as a known human carcinogen. Occupational exposures and doses in experimental animals were in parts per million (ppm). Residential exposures to vinyl chloride from the Class I Unit of the B.K.K. Landfill have been in ppb. (It takes 1,000 ppb to make 1 ppm.) Studies in experimental animals have shown evidence of greater sensitivity in the very young from exposure to vinyl chloride. USEPA interprets the sensitive period seen in animals as relating to children aged 0 to 5. Increased sensitivity from exposures at a young age is discussed in this section and in the risk characterization section.

EXPOSURE ASSESSMENT: Three sets of ambient vinyl chloride monitoring data are used in this addendum report: 1981-1982 data collected by South Coast Air Quality Management District (SCAQMD), with a detection limit of 10 ppb; 1983-1990 data collected by SCAQMD, with a detection limit of 2 ppb; and 1993-1994 data collected by the B.K.K. Corporation, with a detection limit varying from 0.005 to 0.010 ppb. SCAQMD's data were collected at three residential monitoring stations located southeast and south of the Class I Unit: Station A, located on the corner of Marlena and Nogales Streets; Station B, located on Lynn Court; and Station MY, located on Myra Court. B.K.K. Corporation's data were collected at three locations in close proximity to the former locations of Stations A, B, and MY. The 1981-1982 data were collected for 16 of the 24 months, the 1983-1990 data were collected almost daily, and the 1993-1994 data were collected every 12 to 14 days. Data collected for less than a 12-month period were assumed to be representative of a 12-month period. Photographs of the landfill operations during the early 1980s show activities such as the changing working face of the Class I Unit and the proximity of residential development. Seventy-two percent of the total 3.4 million tons of hazardous waste accepted at the Class I Unit was liquid hazardous waste, and approximately 85 percent of accepted fluids were disposed of at the working face. Odor studies conducted between 1978 and 1981 show that the most number of complaints came from residents on "M" and "L" named streets, located southeast and south of the Class I Unit, respectively. Residential development near Station A was completed in 1977; near Station MY, in 1979; and near Station B, in 1979. Data on other hazardous chemicals detected at Station A in 1982 are discussed, as are data on the same hazardous chemicals detected in the air basin in 1985. It is not possible, with the available data, to distinguish concentrations of these other hazardous chemicals that are due solely to emissions from the B.K.K. Landfill.

RISK CHARACTERIZATION: Assumptions regarding the ambient vinyl chloride monitoring data used in the risk calculations are presented. Exposures are assumed to have begun in 1980, since residential development in the area had been completed in 1979. A reasonable maximum exposure scenario (30-years) and an average exposure scenario (9-years) are calculated, rather than the worst-case scenario used in the interim report. A "reasonable maximum exposure" is the maximum exposure that is reasonably expected to occur, and is intended to be a conservative estimate, well above the average yet within the range of possible exposures. The unit risk value for vinyl chloride used in the calculations is the current California Environmental Protection Agency (Cal/EPA)

value of 20 x 10^{-5} per ppb, rather than the value of 0.69 x 10^{-5} per ppb used in the interim report. The unit risk is an upperbound estimate of the probability of developing cancer as a result of daily exposure over 70 years to 1 ppb of a substance in air. The Cal/EPA unit risk value was derived from a study in experimental animals (mice). Assuming the reasonable maximum exposure scenario of 30 years with exposures beginning in 1980, and assuming that individuals spend 80 percent of their time at home, the estimated excess (above background) lifetime cancer risk for an individual near the monitoring stations is as follows: at Station A, 7 x 10^{-5}; at Station B, 6 x 10^{-5}; at Station MY, 9 x 10^{-5}. Assuming an average exposure scenario for a 9-year period beginning in 1981 (covering the years of highest average annual concentrations) the estimated excess cancer risks are: at Stations A, 7 x 10^{-5}; at Station B, 5 x 10^{-5}; and at Station MY, 8 x 10^{-5}. Excess lifetime cancer risks using different assumptions for time spent at home, the average annual concentration of vinyl chloride in 1980, the substituted value for samples with nondetectable concentrations in the 1981-1982 data, the exposure duration, and the unit risk value are presented using Station A as an example. When values of 62, 76 or 96 percent time spent at home are assumed, estimates of excess cancer risk are between 5 and 8 x 10^{-5} at Station A. When the value of 5 ppb is substituted for the average annual concentration in 1980 and for samples with nondetectable concentrations in the 1981-1982 data at Station A, the estimate of excess cancer risk is 8 x 10^{-5}; when the value of 0 ppb is substituted, the estimate of excess cancer risk is 6 x 10^{-5}. When a worst-case exposure duration (70 years; 1980-2046) is assumed, there is no change in the risk estimate at Station A. When unit risk values derived from studies in male workers exposed to vinyl chloride are assumed, estimates of excess cancer risk are between 0.9 and 1 x 10^{-5} at Station A. The population excess cancer burden (that is, the excess number of cases of cancer that may occur in a defined population who all have the same individual excess lifetime cancer risk) is not estimated because not all of the population around the landfill were or are exposed to the concentrations of vinyl chloride detected at Stations A, B, and MY, and it is not possible to obtain a reliable estimate of exposure to other residential areas.

This addendum health risk assessment provides an assessment of past, present, and future risks for individuals living near Stations A, B, and MY from exposures to ambient fugitive emissions of vinyl chloride associated with the closed Class I Unit of the B.K.K. Landfill. There are four findings of this report:

1. From the available monitoring data, the single year of highest detected exposure to vinyl chloride was 1981. The maximum detected concentration of vinyl chloride at Station A in 1981 was 50 ppb, compared to 0.16 ppb in 1994. The average annual concentration at Station A in 1981 is estimated to have been 7.2 ppb, compared to an estimate of 0.05 ppb in 1994. For individuals who lived near Stations A, B, and MY during the 1980's, past exposures to vinyl chloride were higher than current exposures. The cumulative individual excess lifetime cancer risk suggests that for exposures beginning in 1980, the majority of the cancer risk was incurred by 1983-1984, and that there is little change (on the order of 15 to 22 percent) in the cumulative risk after 1986.

2. The landfill gas collection system, which was expanded over time, and the cap covering the closed Class I Unit have been successful in lowering the frequency and level of vinyl chloride emissions to levels that pose little, if any, risk to nearby residents.

3. Regulatory agencies with responsibility for remediation or control of the B.K.K. Landfill may want to include past exposures of vinyl chloride near Stations A, B, and MY in subsequent health assessments of emissions from the landfill to ensure future risks do not add significantly to the past cumulative risk.

4. In spite of using best available scientific knowledge, there is considerable uncertainty associated with the estimated exposures, the carcinogenic potency value of vinyl chloride, and young-age sensitivity methodology. The estimated excess cancer risks calculated in this addendum report are upperbound estimates, based on the assumptions, methodology, limitations and uncertainties specified. The actual excess cancer risk is likely to be lower than these estimates and may be zero. Because the number of people who actually lived or are living near Stations A, B, and MY is small, it is unlikely that excess cases of cancer will occur. Each exposed individual may have an increased risk of developing cancer, however, the added risk is small.

INTRODUCTION

This report is prepared as an addendum to the interim report, "Health Risk Assessment of the B.K.K. Landfill, West Covina, California, November 1990" (DHS-HWTS, 1990), and presumes the reader is familiar with the interim report. The format follows the four components of a health risk assessment: hazard identification, toxicity assessment, exposure assessment, and risk characterization.

This report is an addendum because at the time the interim report was being released to the public, the carcinogenic potency of vinyl chloride was being reevaluated by DHS and the Air Resources Board (ARB). The unit risk is a measure of carcinogenic potency and an upperbound estimate of the probability of developing cancer as a result of daily exposure over 70 years to one ppb of a substance in air. It was expected that the reevaluation would indicate vinyl chloride was more potent than previously thought, which would make the cancer risk estimated in the interim report an underestimate and out-of-date. The interim report stated that an addendum would be prepared which would use the revised unit risk value for vinyl chloride and additional ambient vinyl chloride monitoring data. This addendum report is not required by any state or federal statute, regulation, or agency.

The original impetus for the health risk assessment of the Class I Unit of the B.K.K. Landfill dates from 1985. At that time, DHS committed to undertake three health studies in response to residents' concerns about potential health hazards associated with living near the B.K.K. Landfill. The health studies were: a cancer study, a health risk assessment, and a reproductive outcomes study. A cancer study investigates cases of diagnosed cancers whereas a health risk assessment estimates the theoretical probability (the "risk") of developing cancer. This addendum report plus the interim report complete the health risk assessment of the Class I Unit of the B.K.K. Landfill in West Covina.

Objective, Methodology, and Scope of the Addendum Health Risk Assessment

The objective of this addendum report is to quantitatively estimate the "excess" lifetime cancer risk associated with exposures to vinyl chloride at residential monitoring Stations A, B, and MY. "Excess" refers to the risk above the background risk in the United States of developing cancer during a 70-year lifetime. Within the United States, the probability of developing cancer is 4 in 10, or 40 percent (Miller *et al.*, 1993). This addendum report estimates risks above the background level.

The methodology of this addendum report follows risk assessment guidelines set forth by USEPA (USEPA, 1989a; 1989b; 1991a; 1991b; 1992a). These guidelines are used so that the addendum report will be consistent with standard methodology, and so that its findings can be compared to findings of other health risk assessments of the B.K.K. Landfill. Changes in USEPA's recommended guidelines followed in this addendum report include the use of reasonably maximum and average exposure scenarios, and use of half the detection limit for samples with nondetectable concentrations. The interim report had used a worst-case exposure scenario, and the value of the detection limit for samples with nondetectable concentrations. **TABLE 1** highlights similarities and differences between the interim report and this addendum report.

TABLE 1

COMPARISON OF THE 1990 INTERIM AND 1996 ADDENDUM HEALTH RISK ASSESSMENT OF THE CLASS I UNIT OF THE B.K.K. LANDFILL, WEST COVINA, CALIFORNIA

Section	1990 Interim Report	1996 Addendum Report
HAZARD IDENTIFICATION		
• *Chemical of Primary Concern*	Vinyl chloride	Vinyl chloride
• *Other Chemicals Detected in Ambient Air*	7 known or suspected human carcinogens: vinyl chloride (known), benzene (known), trichloroethylene, perchloroethylene, chloroform, *1,1-dichloroethylene, and 1,2-dichloroethane	6 known or suspected human carcinogens: vinyl chloride (known), benzene (known), trichloroethylene, perchloroethylene, chloroform and 1,2-dichloroethane
TOXICITY ASSESSMENT		
• *Unit Risk Value*	0.69×10^{-5} per ppb	20×10^{-5} per ppb
• *Non-Cancer Health Effects*	Estimated using Chronic Reference Doses	Not applicable
EXPOSURE ASSESSMENT		
• *Vinyl chloride monitoring data*		
◦ *South Coast Air Quality Management District (SCAQMD)*	1983 - 1987	June 1981 - December 1982 January 1983 - March 1990
◦ *BKK Corporation*	Not applicable	September 1993 - September 1994
• *Samples With Nondetected Concentrations*	Nondetected concentrations given a value equal to the detection limit	Nondetected concentrations given a value equal to 1/2 the detection limit
• *Exposure Scenario(s)*	Worst-Case (70 years)	Reasonably Maximally Exposed (30 years) Average Exposed Individual (9 years)
RISK CHARACTERIZATION		
• *Individual Excess Lifetime Cancer Risk*	Estimated at Station MY using 1987 ambient vinyl chloride monitoring data Estimated at Station A using 1982 - 1987 ambient vinyl chloride monitoring data	Estimated at Stations A, B and MY using SCAQMD and BKK monitoring data (1981 - 1994)
	Estimated for the 1982 Expanded Monitoring Program (ambient air)	Discussed in Exposure Assessment section
	Estimated for the 1984 Extended Monitoring Program (indoor air)	Updated risk calculation in Appendix G
	Estimated using the Air Resources Board model of 1987 vinyl chloride data	Modeling program discussed in Appendix F
• *Population Excess Cancer Burden*	Estimated for the population within 1 mile of the landfill (40,000 people)	Not applicable

* 1, 1-dichloroethylene is no longer classified as a suspected human carcinogen by the California Environmental Protection Agency.

Typically, risk assessments required by regulatory agencies estimate current and future risk only. The methodology builds in overestimation of exposures to prevent underestimation of potential risks. Mitigation measures can then reduce current and future exposures if risk estimates are not acceptable. USEPA guidance for Superfund sites notes that "exposure assessments may consider past, present, and future exposures. Current and past scenarios should be based on actual exposure conditions, future scenarios on potential exposure conditions. Typically, Superfund risk assessments focus on current and future exposures, but past exposures may be important for selected sites" (USEPA, 1989b). The guidance recommends the Agency for Toxic Substances and Disease Registry (ATSDR) take the lead in evaluating current health effects from past exposures. ATSDR, created in 1980, is the federal agency authorized to conduct public health assessments at sites subject to the Resource Conservation and Recovery Act (RCRA). ATSDR defines a public health assessment as "an evaluation of environmental contamination data, community health concerns, and health outcome data" (ATSDR, 1994). Of 69 public health assessments completed by ATSDR in 1994, approximately half were for sites with documented past exposure.

Th B.K.K. Landfill is not a Superfund site. Between 1980 and 1981, USEPA granted "interim status" to the operation of the Class I Unit (see CH2M Hill, 1988). After subsurface landfill gas migrated from the Class I Unit and triggered the emergency evacuation of 19 homes along the southern border in July 1984, ATSDR evaluated the landfill (ATSDR, 1984). Because the duration of exposure to subsurface landfill gas was determined not to have exceeded seven years (the age of the affected homes), ATSDR concluded that an epidemiologic study was not indicated at that time, and DHS continued as the lead agency for health issues.

Of concern regarding the B.K.K. Landfill are the following:

- Ambient air exposures are to vinyl chloride, a known human carcinogen; vinyl chloride is one of the few chemicals for which there are both human data and experimental animal studies, which indicate carcinogenic potential;

- Vinyl chloride is clearly associated with the closed Class I Unit of the landfill; wastes containing vinyl chloride were deposited in the Class I Unit until June 1981;

- Vinyl chloride is without detectable background contribution in the South Coast air basin.

OEHHA's health risk assessment is possible because vinyl chloride was monitored in residential neighborhoods (Stations A, B, and MY) almost daily for nearly a decade by SCAQMD. Monitoring in residential neighborhoods eliminates the uncertainty associated with extrapolating from onsite to offsite locations. As technology advanced, the detection limit of vinyl chloride was lowered, and the data consistently met SCAQMD's internal quality control/quality assurance guidelines.

The scope of this addendum report is more refined than was the scope of the interim report. The refinement is due primarily to the iterative nature of risk assessments, and also to some changes in recommended guidance. The interim report was based on the exposure

characterization (CH2M Hill, 1988), and estimated excess cancer risk for individuals living within a one-mile radius of the landfill, using data from four air monitoring programs (collected between 1982-1987) and one air modeling program, and a 70-year (worst-case) exposure scenario. Congruent with the iterative nature of risk assessments, this addendum report focuses on exposures associated with detections of vinyl chloride at Stations A, B, and MY because these locations, southeast and south of the closed Class I Unit of the B.K.K. Landfill, have been identified as the most impacted residential neighborhoods, and long-term monitoring data are available. Exposures occurred at other residential locations around the landfill. However, detected concentrations of vinyl chloride at other residential locations around the landfill were neither as high nor as frequent as detected concentrations at Stations A, B, and MY, and long-term monitoring are not available for the other locations. This addendum report assumes exposures at Stations A, B, and MY began in 1980 because residential development near Stations A, B, and MY was completed in 1979. Long-term ambient vinyl chloride monitoring began in June 1981.

Only ambient (outdoor) fugitive emissions of vinyl chloride from the closed Class I Unit are evaluated in this addendum report. Complete characterization of the air pathway would also include a consideration of indoor air, reflecting exposure, if any, through subsurface landfill gas migration, and other effects that may make ambient and indoor concentrations differ. Subsurface landfill gas, as indicated by methane, did migrate from the landfill in 1984, and a limited indoor air sampling program for volatile organic compounds was conducted in selected homes. The limited indoor air sampling data is not included in the risk characterization of the long-term ambient vinyl chloride monitoring data because the short-term nature of the indoor monitoring is not compatible with the long-term monitoring. However, an updated risk estimate and more information about the short-term exposure that occurred in 1984 is presented in Appendix G. At this time, there is no evidence that subsurface landfill gas migration is a problem for homes in close proximity to the landfill. However, the B.K.K. Corporation will conduct a subsurface soil gas investigation under USEPA's RCRA Section 3008(h) Corrective Action Order on Consent to determine if a potential could exist for migration of subsurface soil gas even with of the landfill gas collection system. Exposures to additional hazardous chemicals were documented in the first health risk assessment of the B.K.K. Landfill (DHS-ARB- SCAQMD, 1983). These data and other data are evaluated in this addendum report.

Previous Health Risk Assessments of the B.K.K. Landfill

The first health risk assessment of the B.K.K. Landfill was conducted as a tri-agency study by DHS-ARB-SCAQMD and released in 1983 (DHS-ARB-SCAQMD, 1983). The study estimated excess lifetime cancer risk associated with exposures to concentrations of seven chemicals detected in ambient air at five locations around the landfill and one background location between July 19 to October 15, 1982. Exposures were assumed to have begun in 1976, when the county building inspector began to approve dwellings for occupancy near the landfill (DHS-ARB-SCAQMD 1983). The excess cancer risk for the limited time period was estimated to be approximately 5×10^{-5}.

The second health risk assessment conducted by the state was the interim health risk assessment (DHS-HWTS, 1990). In 1985, DHS initiated three health studies in response to residents' concerns: a cancer study, a health risk assessment, and a reproductive outcomes study. At a public meeting in December 1990, the cancer study, completed in two parts (Mack and Thomas, 1985; Mack and Pinder, 1988), the interim health risk assessment, and an initial

pregnancy outcomes study (Rust *et al.*, 1988) were released. A fact sheet summarizing the findings of the two-part cancer study and the interim health risk assessment was made available for residents (DHS-EETB, 1990). A copy of the fact sheet is contained in Appendix B. The two-part cancer study investigated cases of cancer diagnosed in residents who lived near the landfill between January 1972 through December 1982. The two-part study found no overall increase in the cancer rate among residents in the 11 census tracts studied, and no consistent pattern of occurrence to suggest that the B.K.K. Landfill was linked to individual types of cancer. Limitations of the study were explained in the fact sheet (see Appendix B). The interim health risk assessment estimated the theoretical probability of developing cancer for residents living near the landfill from exposure primarily to vinyl chloride in ambient air through 1987. The risk was estimated to be approximately 1 to 2 x 10^{-5}. The study of pregnancy outcomes in the 1978-1984 birth cohorts found elevated rates for some pregnancy outcomes (very low birth weight [less than 1,500 grams] and neonatal mortality [death in the first 28 days of life]). The findings of the pregnancy outcomes study stimulated a more comprehensive investigation by DHS, and that study is currently in the process of being completed.

The B.K.K. Corporation has completed three health risk assessments of the landfill, and is in the process of conducting one under the directive of the USEPA. The first two were prepared in 1986 as declarations filed in Superior Court of the County of Los Angeles on behalf of the B.K.K. Corporation. Those assessments recalculated the excess cancer risk associated with the seven chemicals detected in the 1983 tri-agency report, for the exposure period 1978-1985. In one report (Crump, 1986), the risk was estimated to be 0.25 x 10^{-5} (maximum likelihood estimate) and 1.1 x 10^{-5} (upper 95 percent confidence limit estimate); in the other report (Bogen and Smith, 1986), the risk was estimated to be 1.7 x 10^{-5} ("best" [50th percentile] estimate using a Monte Carlo analysis). A copy of each assessment was attached as appendices to the interim report. The third health risk assessment, prepared by ENVIRON Corporation in 1991 in fulfillment of the Air Toxics "Hot Spots" Information and Assessment Act (AB 2588) of 1987, is being reviewed by SCAQMD (ENVIRON Corporation, 1991). The "AB 2588" risk assessment is a regulatory risk assessment, which means that its purpose was to estimate current and future risks associated with the landfill (California Air Pollution Control Officers Association [CAPCOA], 1991). As per the Health and Safety Code Section 44361, OEHHA-Air Toxicology and Epidemiology Section (ATES) reviewed the AB 2588 risk assessment and submitted its comments to SCAQMD (OEHHA, 1993). The current health risk assessment required by USEPA will address evaluations of all environmental media: groundwater, soils, air and if possible, surface water. Evaluation of the air medium will include ambient air and subsurface soil gas pathways. The ambient air pathway will include a one-year sampling program of 19 volatile organic compounds, including vinyl chloride, at onsite and offsite locations (B.K.K. Corporation *et al.*, 1994).

Organizational Changes in the Department of Health Services Since the 1990 Interim Report

In July 1991, Governor Wilson created Cal/EPA. HWTS, which prepared the interim report, was moved from DHS to Cal/EPA. The Health Hazard Assessment Division of DHS became OEHHA. This addendum health risk assessment is prepared by OEHHA-HWTS.

The Environmental Epidemiology and Toxicology Branch (EETB) of DHS is now the Environmental Health Investigations Branch (EHIB), in the Division of Environmental and

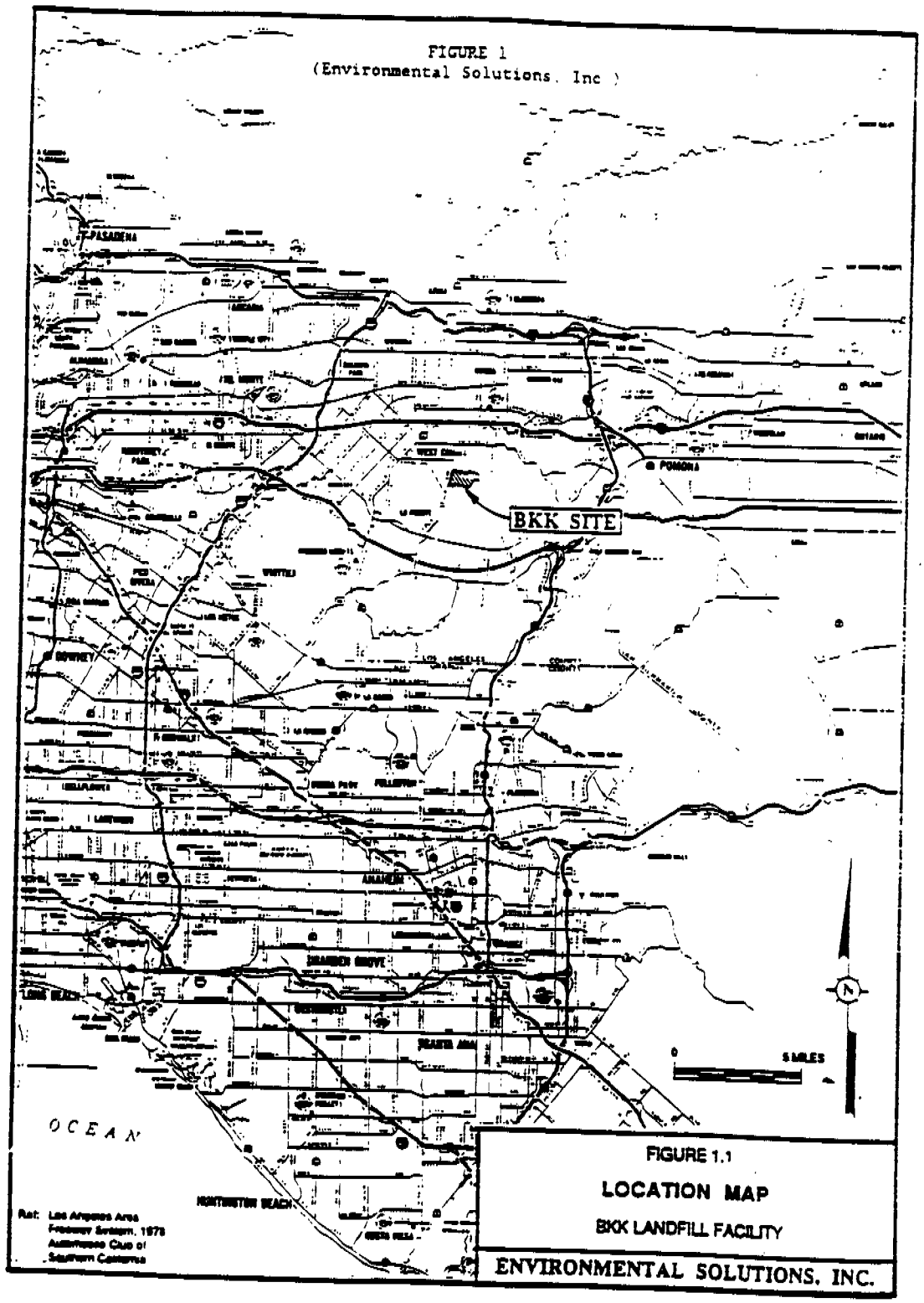
Occupational Disease Control (DEODC) of DHS. EHIB is completing the more comprehensive reproductive outcomes study.

Appendices to the Addendum Health Risk Assessment

Appendices to this report contain the following: Appendix C, updated toxicity tables from the 1990 interim report; Appendix D, statistical evaluation of the 1983-1990 ambient vinyl chloride monitoring data collected by SCAQMD; Appendix E, 1990 population census tract data for the vicinity of the landfill; Appendix F, an air dispersion model of the 1983-1990 ambient vinyl chloride monitoring data; Appendix G, updated risk estimates for the 1984 indoor air monitoring program evaluated in the interim report; Appendix H, a copy of a letter from DHS-Cancer Surveillance Section regarding review of recent tumor incidence data; Appendix I, a copy of the notice of release of the draft addendum report; and Appendix J, OEHHA's response to comments on the draft addendum report.

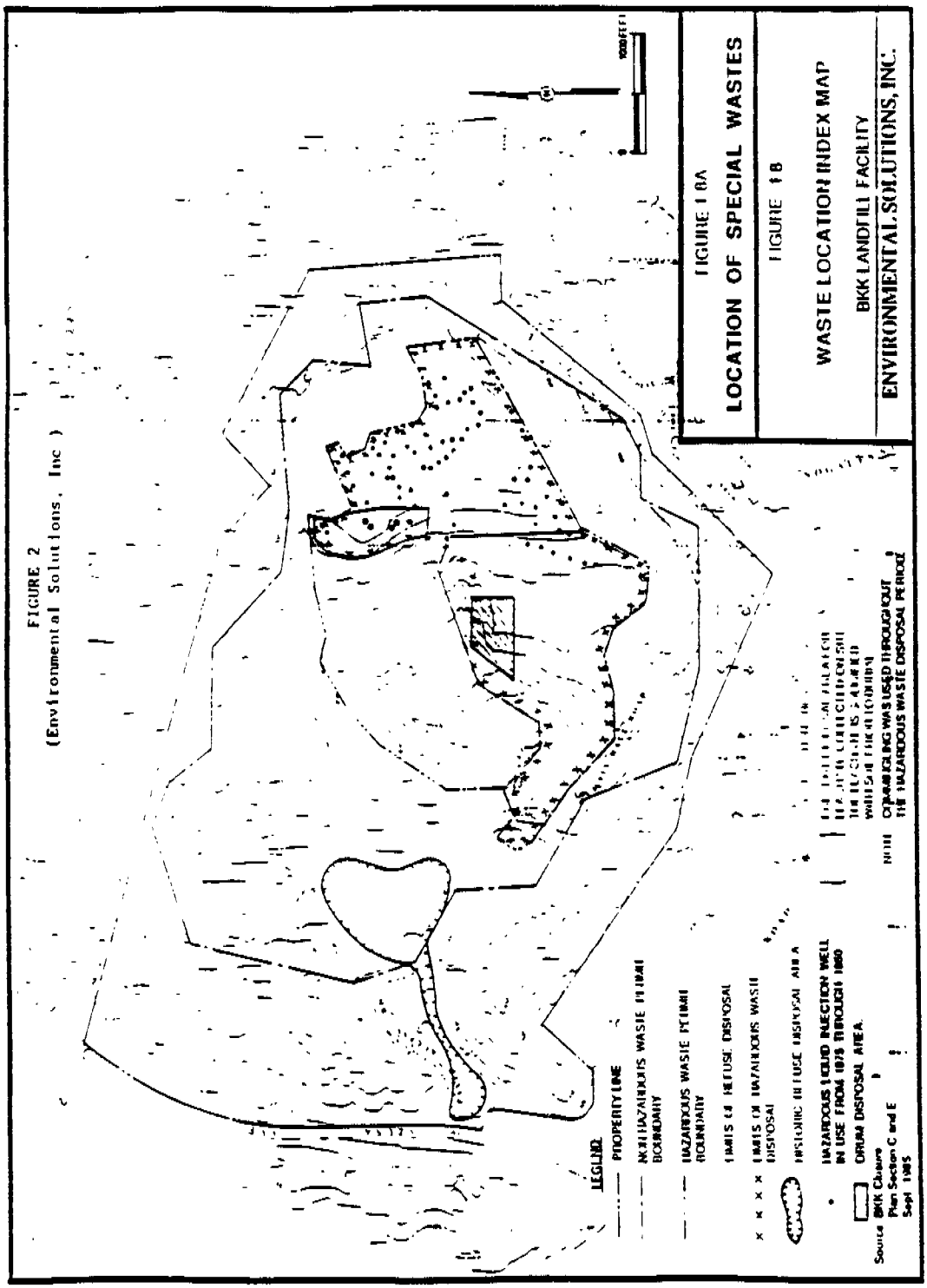
FIGURE 1 and **FIGURE 2**, from the interim report, show the location of the B.K.K. Landfill and the closed Class I Unit, respectively.

FIGURE 1



Source: Environmental Solutions, Inc., Figure 1.1, "BKK Landfill Site Assessment and Mitigation Work Plan, Book I, Subsurface: Site Characterization," March 15, 1988, p. 1-2.

FIGURE 2



Source: Environmental Solutions, Inc., Figure 1.8, "BKK Landfill Site Assessment and Mitigation Work Plan, Book I, Subsurface: Site Characterization," March 15, 1988, p. 1-16.

CMA 114327

HAZARD IDENTIFICATION

This section provides information about vinyl chloride disposal in the Class I Unit, vinyl chloride emissions from the Class I Unit, other hazardous chemicals detected in ambient air around the landfill, and regulatory agencies involved with the landfill.

Vinyl Chloride in the Class I Unit

While other chemicals are known to be emitted from the B.K.K. Landfill, vinyl chloride is the chemical of concern because it is a known human carcinogen and has clearly been shown to be associated with the landfill. Rarely detected in ambient air in the South Coast air basin, vinyl chloride was monitored almost daily by SCAQMD at residential locations south of the landfill between June 1981 and March 1990.

Emissions of vinyl chloride from the landfill are presumed to be due to two processes: biodegradation of buried wastes containing chlorinated hydrocarbons, and deposition of hazardous waste containing vinyl chloride in the Class I Unit. The landfill opened in 1963, approved by the City of West Covina and the Los Angeles Regional Water Quality Control Board (RWQCB). In 1972, the State passed the Hazardous Waste Control Act. The Act required wastes to be classified as "hazardous" or "nonhazardous." From 1972 until November 30, 1984, the B.K.K. Landfill served as the primary commercial hazardous waste disposal site for the Los Angeles area. Records of hazardous waste disposal at the landfill date from 1975 (CH2M Hill, 1988).

An estimate of the total amount of waste containing vinyl chloride deposited at the landfill has not been identified. Four facilities in the South Coast air basin handled vinyl chloride, and all four disposed of waste containing vinyl chloride in the Class I Unit at the B.K.K. Landfill (ARB, 1982a). Opening years of operations for the facilities were: Keysor-Century in Saugus, 1958; Stauffer Chemical in Carson, 1959 (closed in 1982); B.F. Goodrich Company in Carson, 1960 (closed in 1985); and Union Carbide in Torrance, 1972. Stauffer Chemical produced vinyl chloride until 1982 (ARB Part A, 1990; SCAQMD, 1983). Stauffer Chemical sent wastes from its ethylene dichloride and vinyl chloride operations to the Class I Unit from at least 1975 to mid-1981; in 1979, Stauffer disposed of approximately 50 tons of waste containing vinyl chloride and larger amounts of waste containing other halogenated compounds (ARB, 1983). Waste from its ethylene dichloride plant contained on the average 45 percent ethylene dichloride and 30 percent 1,1,2-trichloroethane (ARB, 1982b). These two chemicals can biodegrade to vinyl chloride (Vogel *et al.*, 1987).

Seventy-two percent (72%) of the total 3.4 million tons of hazardous wastes deposited in the Class I Unit between 1975 and 1984 was liquid hazardous waste (CH2M Hill, 1988). Liquids were disposed of at the working face, buried in drums, or injected into a total of 96 boreholes drilled into buried waste. Injection well (borehole) records identify truckloads of ethylene dichloride waste deposited in 1975, 1978, and 1979 (CH2M Hill, 1988). Approximately 85 percent of accepted fluids were disposed of at the working face and 10 percent in the boreholes (The Janes Network, 1994).

FIGURE 3 is a graphic presentation of the tons of hazardous waste deposited annually in the Class I Unit between January 1975, when records became available, and November 30, 1984, when the unit stopped accepting hazardous waste. Explanations for the dips in the figure are as follows:

- The dip in the figure for 1981 is attributed to the prohibition in June of waste containing vinyl chloride following the detection at the facility perimeter of vinyl chloride above the California ambient air quality standard of 10 ppb;

- The decline in 1984 is attributed to regulatory restrictions on the disposal of liquid wastes, and to the halting of hazardous waste disposal (except for asbestos) on November 30, 1984.

Vinyl Chloride Emissions From the Class I Unit

The landfill gas collection system is one of the ongoing site activities which has reduced the frequency and level of vinyl chloride emissions over time. Gas collection wells and a pilot gas burner and venting system were initially installed at the B.K.K. Landfill in 1975 (CH2M Hill, 1988). In 1980, the system consisted of 15 wells and 2 gas flares. An accelerated gas control program was started in July 1984 after subsurface landfill gas migration triggered evacuation of 19 homes along the southern border of the landfill (see Appendix G). By 1985 the landfill gas collection system included approximately 100 vertical gas extraction wells, 150 perimeter gas control wells, 198 perimeter gas monitoring probes, 4 horizontal gas well systems, and 2 flare stations with a total of 8 gas flares. As of 1994, the system consists of approximately 2,053 vertical gas extraction wells, 317 horizontal gas extraction wells, 170 perimeter gas monitoring probes, and the 2 flare stations with a total of 10 flares. Some of the wells and probes are located on the Class III (nonhazardous) portion of the landfill. The Integrated Waste Management Board (IWMB) and SCAQMD monitor the landfill gas collection system.

Maintenance of the cap on the closed Class I Unit is another factor in controlling fugitive emissions of vinyl chloride from the landfill. In 1992, B.K.K. Corporation and the Department of Toxic Substances Control (DTSC) reached a settlement agreement for violations of state hazardous waste laws and regulations found during a 1990 inspection (Cal/EPA, 1992). Failure to properly maintain the cap, designed to prevent rainwater from leaking in and landfill gas from escaping out, constituted one of the violations. On June 28 and 29, 1994, a multi-agency team consisting of 17 representatives from USEPA, DTSC, SCAQMD, IWMB, and the Local Enforcement Agency (LEA, City of West Covina) inspected the closed Class I Unit in response to the LEA's concerns about continuous detections of vinyl chloride in ambient air. Grid sampling on the surface of the closed Class I Unit over the two days found a total of 15 samples with readings of total hydrocarbons in excess of the instantaneous total hydrocarbon limit of 500 ppm (SCAQMD, 1994b). The total hydrocarbon limit was established under SCAQMD's "District Rule 1150.1," adopted in 1985 for the purpose of reducing gaseous emissions from active landfills (SCAQMD, 1985). SCAQMD issued two Notices to Comply to the B.K.K. Landfill for the elevated surface emissions; reinspection by SCAQMD two weeks later found the surface emissions in compliance. Although the B.K.K. Corporation has been working toward improving the vegetative and clay cover of the closed Class I Unit, conclusions of the multi-agency inspection included a recommendation for routine and consistent maintenance activities (USEPA, 1994a).

FIGURE 3

TONS OF HAZARDOUS WASTE DEPOSITED IN B.K.K. LANDFILL
WEST COVINA, CALIFORNIA

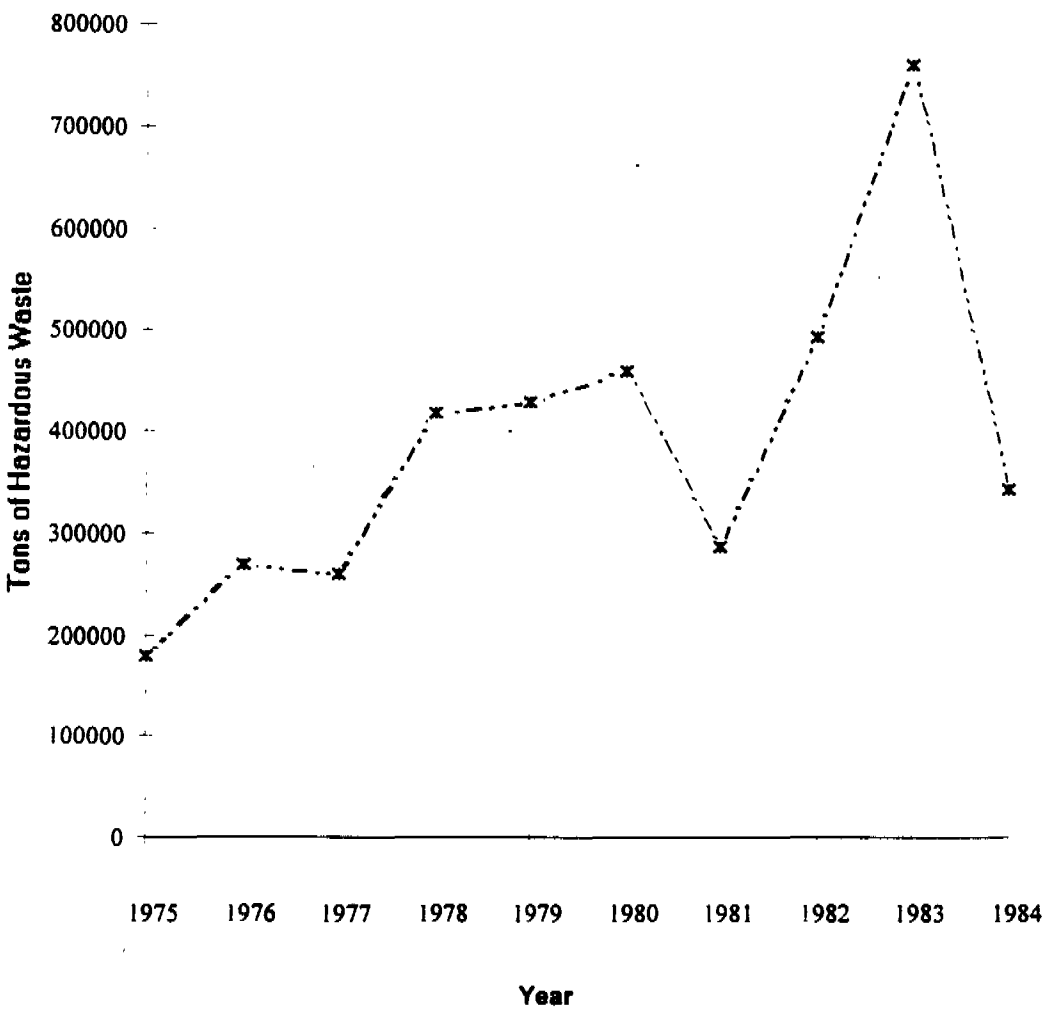


Figure derived from Table 1.4 "BKK Landfill Site Assessment and Mitigation Work Plan, Book I, Subsurface
e Characterization," Environmental Solutions, Inc., March 15, 1988, pgs. 1-22 and 1-23.

CMA 114330

Ambient air monitoring at the perimeter of the landfill for concentrations of total organic compounds and any toxic air contaminant (such as vinyl chloride) in the landfill gas control system was also required under SCAQMD's Rule 1150.1. Semiannual monitoring was required of the B.K.K. Corporation. Based on the Rule 1150.1 monitoring, the B.K.K. Corporation published a Proposition 65 Warning for detections of vinyl chloride along the southern perimeter of the landfill on August 29, 1993, in the *San Gabriel Valley Tribune* (B.K.K. Corporation, 1993b). (Proposition 65 is California legislation [Health and Safety Code Section 25249.5 *et seq.*] regarding exposures and discharges of known carcinogens and reproductive toxicants. For carcinogens, Proposition 65 requires a warning for exposures in excess of an individual estimated cancer risk of 1×10^{-5}. For vinyl chloride, the 1×10^{-5} risk level of 3 micrograms per day under Proposition 65 regulations [California Code of Regulations, Title 22, Section 12705] equates to a concentration in air of 0.05 ppb.) In September 1993, the B.K.K. Corporation began a voluntary ambient air sampling program to collect data that would be used to refine the Proposition 65 warning (B.K.K. Corporation, 1993c). B.K.K. Corporation's Proposition 65 monitoring program evolved to include additional perimeter stations, dilution (residential) stations, and collocated (duplicate) stations, and increased the frequency of monitoring to every 12 to 14 days. The B.K.K. Corporation also sought to lower the detection limit for vinyl chloride (B.K.K. Corporation, 1993a). Since 1993, the B.K.K. Corporation reports achieving a detection limit varying from 0.005 to 0.010 ppb. USEPA has stated that the analytical procedure appears to be acceptable to USEPA for the purposes of ambient air monitoring at the B.K.K. Landfill (USEPA, 1994e). SCAQMD reviewed the modified protocol for vinyl chloride analysis and found it acceptable (SCAQMD, 1994a).

USEPA also required control of emissions of vinyl chloride from the closed Class I Unit. Beginning in October 1993, USEPA required the B.K.K. Corporation to develop a Response Action Plan (RAP) which would implement actions within 30 days to reduce vinyl chloride emissions whenever ambient air sampling results showed detections of vinyl chloride equal to or greater than 0.5 ppb (USEPA, 1994f). Response actions included sealing cracks in the cap of the closed Class I Unit, and pumping liquids from gas collection wells.

Other Hazardous Chemicals Detected in Ambient Air

The earliest ambient air monitoring data of the B.K.K. Landfill is contained in the 1980 report, "Second Interim Report, Investigation of Odorous and Volatile Compounds for BKK Class I Landfill Site in the City of West Covina," prepared by the University of Southern California (USC) for the B.K.K. Corporation. Vinyl chloride was not among the more than 20 compounds analyzed; benzene and chloroform were identified as the carcinogens of concern (USC, 1980). The data were collected during active, open-face hazardous waste disposal. They were reviewed by CH2M Hill in the exposure characterization report and found to have limitations and uncertainties in laboratory analytical accuracy and quality assurance which precluded their use as a primary data set to assess human exposure (CH2M Hill, 1988).

The 1982 ambient air program conducted by the three agencies (DHS-ARB-SCAQMD, 1983) was designed to monitor nine compounds known or suspected to be human carcinogens. In addition to vinyl chloride, the following chemicals were monitored: benzene (also a known human carcinogen), chlorobenzene, trichloromethane (chloroform), 1,1-dichloroethene (vinylidene chloride), trans-1,2-dichloroethene, trichloroethene, perchloroethene, and 1,2-dichloroethane (ethylene dichloride). Trans-1,2-dichloroethene and chlorobenzene were

not detected. Vinylidene chloride is no longer considered a carcinogen by Cal/EPA (Standards and Criteria Work Group [SCWG], 1994).

The carcinogens detected in 1982 have been detected in landfill gas at the inlets to the landfill flares. Data from flare emissions and source tests were reviewed by CH2M Hill in the exposure characterization (CH2M Hill, 1988). Flare emissions data were determined to be inadequate for use in exposure assessment because of lack of replicate sampling, split sampling, and simultaneous ambient air sampling. The source tests data were found to be inadequate because of limited sampling time (3-hour tests), which may not reflect a full 24-hour period.

The carcinogens detected at Station A in 1982 are among 12 selected volatile organic compounds monitored by ARB and/or SCAQMD in the air toxics monitoring program, begun in 1985 (SCAQMD, 1987). The air toxics monitoring program provides information on background levels for the selected compounds in the Los Angeles air basin. Data from ARB's El Monte Station were included in the interim report because the El Monte Station appeared to be more representative of ambient air conditions around the B.K.K. Landfill. However, vinyl chloride and 1,2-dichloroethane were not monitored at the El Monte Station. Monitoring in 1985 at SCAQMD's Azusa Station, located approximately 3.5 miles to the north/northwest of the B.K.K. Landfill, included the six carcinogens also detected at Station A in 1982. These two data sets are discussed in the exposure assessment section of this addendum report.

In December 1994, the B.K.K. Corporation began a one-year ambient air monitoring program for 19 volatile organic compounds at onsite and offsite locations around the facility. These data are being reviewed by USEPA for the risk assessment it is requiring the B.K.K. Corporation to conduct.

Regulatory Agencies Involved With the B.K.K. Landfill

Regulatory agencies involved with activities of the landfill are: the USEPA, for corrective actions at the entire 583-acre facility; DTSC, for the closed 170-acre Class I Unit; SCAQMD, for control of gaseous emissions from the facility; RWQCB, for discharges from the facility affecting groundwater or surface water; IWMB and the City of West Covina, for solid waste issues at the facility. ARB provides technical support to the agencies, as needed.

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TOXICITY ASSESSMENT

This section explains the toxicity values used in the risk characterization, and adds information on how increased knowledge has lead to changes in the assessment of the carcinogenicity of vinyl chloride.

Toxicity Values

Toxicity values are numerical expressions of a chemical's dose-response relationship, that is, the amount of a chemical (dose) that induces an effect (response). Toxicity values are developed for noncarcinogenic and carcinogenic effects.

Reference doses (RfDs) are used when exposure occurs by the ingestion route. Since the 1990 interim report, USEPA developed reference concentrations (RfCs) for exposures by the inhalation route. Both RfDs and RfCs are used as measures of a chemical's noncarcinogenic toxicity. RfDs and RfCs are not expressed as probabilities or risks but rather as thresholds below which it is unlikely that even sensitive populations will experience adverse health effects from exposure to a specific chemical.

A slope factor is the upperbound estimate of the probability of a response per unit intake of a chemical over a lifetime, expressed in the units "(milligrams per kilogram per day [mg/kg-d])$^{-1}$." The unit risk is the probability of a response per unit concentration of a substance in the medium where human contact occurs. Both the slope factor and the unit risk are measures of a chemical's carcinogenic activity. For ambient air exposures, the unit risk is an estimate of the probability of developing cancer as a result of daily exposure over 70 years to 1 ppb of a substance in air. The unit risk is expressed as "(ppb)$^{-1}$."

Updated toxicity information for carcinogens detected in environmental media around the landfill is contained in Appendix C.

History of the Assessment of Vinyl Chloride Toxicity

Understanding of adverse health effects associated with exposure to vinyl chloride, and the "dose" necessary to induce the effects, has advanced over the years. Vinyl chloride is one of the few chemicals for which there are both epidemiologic studies and experimental animal data. The following chronology is presented to show the change over time of awareness about acute effects, chronic effects, carcinogenic effects, and developmental/reproductive effects.

Vinyl chloride, a synthetic organic chemical, is a gas at ambient temperature and pressure, with a density twice that of air. First prepared in 1833, commercial preparation of vinyl chloride in the United States began in 1939 (United States Department of Labor [USDOL], 1974). The primary use of vinyl chloride is in the production of polyvinyl chloride (PVC). The industry is divided into three segments: monomer production, polymer production, and fabrication. Production of the monomer is a large-scale continuous process, involving only a few firms. Conversion to the polymer or copolymer is an incomplete batch process in which not all of the monomer is reacted (Barnes, 1976). Until 1974, high exposures to workers occurred during

evacuation of the residual monomer gas, and during cleaning of the polymerization tank walls. Current regulatory standards and manufacturing processes substantially reduce or eliminate worker exposure to vinyl chloride. Little exposure occurred in the fabrication segment of the industry, where the finished PVC product is made by extrusion, injection molding, and calendering processes.

Initially, vinyl chloride was regarded as a material of low human toxicity. In the 1930s, its use as a surgical anesthetic was considered, with narcotic properties being seen at levels around 8,000,000-10,000,000 ppb. However, its high flammability, and the cardiac and circulatory disturbances noted in animals and humans, precluded its use in surgical anesthesia (Patty, 1987).

Acute, narcotic effects from exposure to vinyl chloride were the initial toxic end points of concern. The first occupationally associated deaths attributed to vinyl chloride were reported in the literature in 1960 (Danziger, 1960). The deaths were attributed to inhalation of vinyl chloride, presumably at levels above 10,000,000 ppb.

In 1961, an indication of a chronic hazard was observed primarily in workers who cleaned polymerization tank vessels. They experienced a wide range of symptoms which became known as "vinyl chloride disease" and included: Raynaud's phenomenon (fingers blanch and experience numbness and discomfort upon exposure to cold), acro-osteolysis (resorption of the terminal bones of the fingers and/or toes), joint and muscle pain, enhanced collagen deposition (the major protein of the white fibers of connective tissue, cartilage, and bone), stiffness of the hands, and scleroderma-like skin changes (thickening of the skin with adhesions to underlying tissue). These symptoms were observed at exposure levels below 50,000 ppb. Studies in the 1980s identified a role of the immune system in workers with vinyl chloride disease, and determined that susceptibility to vinyl chloride disease was increased in the presence of specific genes; the immunologic effects of vinyl chloride were observed in mice exposed to 10,000 ppb (ATSDR, 1993). No case of vinyl chloride disease has been reported since 1974 (ATSDR, 1990).

In 1974, three employees from one PVC manufacturing company in the United States were identified as having died from a rare type of liver cancer, angiosarcoma of the liver (Creech and Johnson, 1974; Falk *et al.*, 1974). A fact-finding investigation by the Occupational Safety and Health Administration (OSHA) uncovered additional deaths due to angiosarcoma of the liver among workers in three similar companies. Between 1961 and 1977, 23 cases of liver angiosarcoma were reported among approximately 20,000 vinyl chloride workers in the United States (ARB Part B, 1990).

Studies were undertaken worldwide (Sweden, England, Italy, Japan, Germany, Canada, Norway, France, and Russia) to assess mortality among workers exposed to vinyl chloride. By 1985, at least 17 epidemiologic studies relating vinyl chloride exposure to the incidence of various cancers had been completed. Most of the studies were a retrospective cohort design in which cases were identified 15 to 29 years after first exposure. Since monitoring of vinyl chloride was not routinely conducted before 1975, exposure concentrations were estimated based on job classification and length of employment. A worldwide registry of histologically confirmed cases of liver angiosarcoma resulting from exposure to vinyl chloride identified 120 cases between 1974 to 1986, of which at least 44 percent (53/120) were employed as polymerization tank wall cleaners (ATSDR, 1990). The worldwide registry, although incomplete,

tabulates information about cases known to have occurred in men xposed to vinyl chloride that have been reported by individual manufacturing companies, trade associations, and occupational health organizations in Europe, North America, and Japan (Forman *et al.*, 1985).

Studies in the 1980s examined additional types of cancer and noncancer effects. "Other cancers that have shown a statistically significant increase in mortality among vinyl chloride workers, in at least some studies, included cancer of the brain and central nervous system, the lung and respiratory tract, and the lymphatic/hematopoietic system" (ATSDR, 1993).

The earliest experimental animal study on the carcinogenicity of vinyl chloride was reported in 1971 in a study of rats exposed by inhalation (Viola *et al.*, 1971). Subsequent studies have shown that "vinyl chloride is carcinogenic in mice, rats, and hamsters when given orally and by inhalation. Vinyl chloride has been found to cause tumors in a dose-related manner at several sites, including liver, lung and mammary gland. The oncogenic response appears to be a function of the site, vinyl chloride concentration, tumor type, species of animal, and route of administration" (ARB Part B, 1990). In the liver, different types of tumors (liver angiosarcoma, hepatocellular carcinoma) have been seen following inhalation exposures. Hepatocellular carcinomas were observed only in rats, and the incidence of this tumor was significantly elevated when exposures started early in life (Maltoni *et al.*, 1981; Drew *et al.*, 1983; Cogliano and Parker, 1992). Hepatocellular carcinoma was induced in one study when exposures were begun at eight months of age (Drew *et al.*, 1983). A study in newborn and adult rats on the effect of age on induction of pre-neoplastic hepatic lesions showed a well defined period restricted to the early lifetime of the animals (approximately day 7 to 21) during which the liver is most sensitive to carcinogenic effects of vinyl chloride (Laib *et al.*, 1985a; 1985b).

Cancer is considered to be a multistage process wherein a healthy cell is transformed into a malignant one by a series of somatic mutations. The mechanisms by which vinyl chloride exerts its carcinogenic effects have been elucidated from studies in both cultured cells and experimental animals; vinyl chloride is metabolized to 2-chloroethylene oxide, which binds covalently to deoxyribonucleic acid (DNA) causing mutations (ATSDR, 1993; ARB Part B, 1990). Mathematical models derived from the multistage theory of carcinogenesis assist in estimating which stages in the process of tumor development are affected by a particular chemical. Effects on an early stage suggest the chemical "initiates" the cancer process (that is, that the chemical causes the initial event), while effects on a late stage suggest the chemical "promotes" tumor development. Both the epidemiologic (occupational) and experimental animal data indicate that vinyl chloride affects an early stage of carcinogenesis. Epidemiologic studies have found that workers exposed to vinyl chloride before age 25 (Wong et al., 1991) and between the ages of 25 and 34 (Chen and Blancato, 1989; Pirastu *et al.*, 1990) had a higher risk of liver cancer death than workers exposed after age 35. In animal studies, "across all the species and endpoints examined, the evidence consistently suggests that vinyl chloride primarily affects the initial stage in tumor development," and to a lesser degree, contributes to late-stage tumor progression (Brown and Hoel, 1986).

Exposure to vinyl chloride in the workplace environment was not controlled by any regulatory program during the 1940s and early 1950s. The first recommendations for an occupational exposure limit in the United States were issued in 1955 at a level of 500,000 ppb (Wu et al., 1989). Concern about the narcotic effects of vinyl chloride and the risk of developing acro-osteolysis caused reduction in exposure limits in England in the 1960s (Forman *et al.*, 1985).

One estimate of typical exposures to vinyl chloride monomer among the more highly exposed workers at polymerization plants is: 1,000,000 ppb prior to 1955; from 300,000 to 500,000 ppb during 1955 through 1970; and from 100,000 to 200,000 ppb during 1970 to 1974 (Wu *et al.*, 1989). In 1974, OSHA promulgated a 1,000 ppb occupational standard (USDOL, 1974).

In the late 1970s, several studies were undertaken to determine whether there were any cases of liver angiosarcoma in residential neighborhoods adjacent to vinyl chloride production or polymerization facilities prior to occupational controls (Doll, 1988): a study in Sweden and the former Yugoslavia found none; a study in Great Britain found 1 over a 12-year period but exposure was not attributed to the facility because the individual lived near the plant for only 6 years; a study in New York found 4 cases over an 18-year period in people who lived within 1 mile of fabrication plants or a polymerization plant. The cases in New York were diagnosed between 1958 and 1975, and occurred after 15 or more years of residence, suggesting an association with the facilities (Brady *et al.*, 1977). Doll notes that "This disease [angiosarcoma of the liver] is normally so rare that, in the absence of specific exposure to one of the known causes (vinyl chloride, thorium dioxide, and arsenic in pesticides and medicines), the annual incidence is on the order of $1\text{-}2 \times 10^{-7}$. In these circumstances the discovery of even one case in a man living close to a factory in which vinyl chloride was used in the days before exposure was tightly controlled, may be regarded as presumptive evidence of the effect of environmental pollution." It is reported that concentrations of vinyl chloride within one kilometer of plants handling vinyl chloride in 1975 were on the order of 10 to 40 ppb, which is one-ten thousandth of the estimated concentration that caused the observed occupational incidence (Doll, 1988; Baxter *et al.*, 1977).

"Epidemiological studies of families of vinyl chloride workers or communities having vinyl chloride processing facilities suggested the possibility of an increased incidence of birth defects and spontaneous abortions among people at risk; however, subsequent reviews of these studies have concluded that there is inadequate evidence to link environmental or paternal exposure to vinyl chloride with birth defects or spontaneous abortions in humans" (ARB Part B, 1990). "Vinyl chloride crosses the placenta of experimental animals. Some data indicates it may act as a transplacental carcinogen" (ARB Part B, 1990). Studies in experimental animals have shown developmental toxicity consisting of decreased litter size and fetal weight, delayed ossification, and dilated ureters; however, these effects occurred at levels which also produced maternal toxicity, so it is not clear if the effects on the fetus are due to maternal toxicity or to vinyl chloride (ATSDR, 1993).

Reproductive effects reported in male workers exposed to vinyl chloride are decreased sexual function (androgen levels) and libido; exposure levels were not given and concomitant exposure to other chemicals could not be ruled out in those studies (ATSDR, 1993). A study by Bi *et al.* of male rats exposed by inhalation to vinyl chloride found a statistically significant increase in damage of testicular seminiferous tubules at concentrations of 100,000 ppb; decreased testicular- and increased liver-to-body weight ratios at 100,000 ppb; and no observed adverse effects at 10,000 ppb (Bi *et al.*, 1985). USEPA, DTSC, and OEHHA developed an action level of 25 ppb for indoor air exposures to vinyl chloride near Operating Industries, Inc., a Superfund site in southern California, based on no observed adverse effects in rats at 10,000 ppb (Hiatt *et al.*, 1993). ATSDR developed a minimum risk level (MRL) of 2 ppb for inhalation exposures of 15 to 364 days duration, based on the increased liver-to-body-weight ratio in rats (ATSDR, 1993).

Vinyl chloride has been identified as a mutagen in bacteria, yeast, and animal systems. Workers exposed to less than 15,000 ppb, however, did not show higher levels of chromosome breaks or aberrations than those in the control group (ARB Part B, 1990).

New Unit Risk Value and Proposition 65

OEHHA-ATES reviewed epidemiologic (occupational) studies and animal cancer bioassays for the purpose of developing a unit risk value for ARB as required by the Toxic Air Contaminant Program (Health and Safety Code Section 39650 *et seq.*). The unit risk was derived using the linearized multistage model (described in detail in ARB Part B, 1990). **TABLE 2** lists the 95 percent upper confidence limits (UCLs) range of cancer risk estimates for vinyl chloride derived by OEHHA-ATES. Three values are shaded:

- The high end of the range estimate; the theoretical, upperbound, individual (human) excess lifetime cancer risk of 20×10^{-5} per ppb, based on studies in female mice that developed lung carcinoma;
- The low end of the range estimate; the theoretical, upperbound, individual (human) excess lifetime cancer risk of 2.5×10^{-5} per ppb, based on studies in male workers who developed liver cancer only (liver angiosarcoma and primary liver cancer); and
- The middle of the range estimate; the theoretical, upperbound, individual (human) excess lifetime cancer risk of 4.5×10^{-5} per ppb, based on combined studies in male workers who developed liver, brain, or lung cancers.

A unit risk based only on human studies would range between 2.5×10^{-5} per ppb, for liver cancer only (liver angiosarcoma and primary liver cancer), and 4.5×10^{-5} per ppb, for liver, brain, and lung cancers combined.

OEHHA-ATES stated the "Evaluation of animal tumorigenicity data indicates that vinyl chloride's carcinogenic potency is dependent on sex, tumor site, and age of exposure. Taking all these factors into account, staff conclude that the best estimate to use in order to assure the public health is the top of the range of the animal UCLs of risk, 20×10^{-5} $(ppb)^{-1}$" (ARB Part B, 1990).

By using default conversion factors (that is, assuming an individual weighs 70 kilograms and breathes 20 cubic meters of air a day) and assuming daily lifetime exposure, the unit risk is equivalent to a slope factor of 0.27 $(mg/kg\text{-}day)^{-1}$. Low dose extrapolation models other than the linearized multistage model would likely produce lower or higher estimates of cancer potency, depending on the assumptions used in the model (that is, a sublinear model would yield a lower estimate of cancer potency and a supralinear model would yield a higher estimate). Preference for use of the linearized multistage model in estimating the potency of vinyl chloride is consistent with the generally recognized mechanism of action by which vinyl chloride exerts its carcinogenic effects.

In December 1990, ARB adopted the unit risk value for vinyl chloride recommended by OEHHA-ATES, and identified vinyl chloride as a toxic air contaminant.

In 1992, OEHHA established 3 micrograms per day (µg/day) as the no significant risk level (NSRL) for vinyl chloride under Proposition 65. Proposition 65 is California legislation regarding exposures and discharges of known carcinogens and reproductive toxicants. For carcinogens, Proposition 65 requires a warning for exposures in excess of an individual estimated lifetime cancer risk of 1×10^{-5}. The NSRL represents a daily intake level over a 70-year lifetime that is calculated to result in a risk of 1×10^{-5}. The NSRL of 3 µg/day was derived from the unit risk value adopted by ARB, and equates to an ambient air level of 0.05 ppb vinyl chloride.

TABLE 2
(Office of Environmental Health Hazard Assessment
Air Toxicology and Epidemiology Section)

RANK ORDERING OF ESTIMATES OF HUMAN RISK BY CATEGORY

Rank	Experiment	Strain[a]/Species, Sex	Tumor	Individuals[b]	Unit Risk[c] ppb^{-1}
1	Drew	sw/mouse, female	lung carcinoma	228	$20.x10^{-5}$
2	BT-9,15[d]	sd/rat, female	liver angiosarcoma	380	$18.X10^{-5}$
3	Bi	wi/rat, male	liver angiosarcoma	78	$13X10^{-5}$
4	Drew	fi/rat, female	liver angiosarcoma	353	$8.4x10^{-5}$
5	BT-9,15	sd/rat, male	liver angiosarcoma	298	$6.5x10^{-5}$
6	BT-1,2	sd/rat, female	liver angiosarcoma	313	$4.9x10^{-5}$
7	Waxweiler	oc/human, male	liver+brain+lung	1294	$4.5x10^{-5}$
8	Bi	wi/rat, male	lung angiosarcoma	78	$4.5x10^{-5}$
9	Drew	fi/rat, female	hepatocellular carcinoma	353	$4.4x10^{-5}$
10	Drew	fi/rat, female	mammary	354	$4.2x10^{-5}$
11	BT-1,2	sd/rat, female	mammary	394	$3.7x10^{-5}$
12	Waxweiler	oc/human, male	liver	1294	$2.6x10^{-5}$

a: sw - CDI Swiss, sd - Sprague-Dawley, wi - Wistar, fi - Fischer, oc - occupational cohort.
b: Number of all individuals entered in the analysis, exposed and unexposed.
c: Calculated as an upper confidence limit with units ($mg/kg\text{-}d^{-1}$) and converted to units of concentration (ppb^{-1}) using default conversion factors. The unit risk is an estimate of the probability of being diagnosed with cancer as a result of daily exposure over 70 years to 1 $\mu g/m^3$ of a substance in air.
d: BT - Maltoni's Bentivoglio (Bologna, Italy) project on long-term carcinogenicity bioassays of vinyl chloride.

Source: California Air Resources Board, "Technical Support Document, Part B, Proposed Identification of Vinyl Chloride as a Toxic Air Contaminant," October 1990, Stationary Source Division, Sacramento, California, pages 8-11.

Sensitivity to Vinyl Chloride From Exposures at a Young Age

In 1989, V.J. Cogliano of USEPA described in a memorandum a new assessment of the cancer risk from vinyl chloride (USEPA, 1992b). Cogliano identified a public health concern for young-age exposures based on a sensitive period seen in rats exposed by inhalation to vinyl chloride (Maltoni *et al.*, 1981; Drew *et al.*, 1983; Laib *et al.*, 1985a, 1985b). The studies in rats showed that the carcinogenic effects from exposure to vinyl chloride depend on both age at initial exposure and duration of exposure, and therefore, suggest that children may face higher risks than adults for exposures of a given duration. USEPA interprets the sensitive period seen in rats as relating to children aged 0-5. The incremental cancer risk from breathing vinyl chloride throughout this age range may be equal and in addition to the cancer risk from breathing vinyl chloride throughout adulthood. This means that, for a given air concentration of vinyl chloride, an individual exposed during the first six years of life (age 0-5) incurs the full conventional (70-year) lifetime of risk, and an individual exposed from birth for 70 years incurs double the conventional lifetime risk. Short-term exposures for adults over 30, however, carry less than the conventional lifetime risk, according to USEPA's interpretation of the experimental animal data. **TABLE 3** shows USEPA's estimates of risk for initial 6-year (age 0-5) and subsequent 4-year (age 6-9, *etc.*) apportioned lifetime exposures to vinyl chloride. The risk values in TABLE 3 are based on USEPA's slope factor for vinyl chloride of 0.29 $(mg/kg\text{-}d)^{-1}$, which equates to a unit risk of 23×10^{-5} per ppb. (Note that the USEPA slope factor is almost identical to the Cal/EPA slope factor of 0.27 $[mg/kg\text{-}d]^{-1}$).

USEPA's 1991 interim guidance for Superfund includes a brief note on Cogliano's method of assessing apportioned lifetime exposures to vinyl chloride in the discussion of toxicity values for short-term exposures (USEPA, 1991a). In 1992, Cogliano published his interpretations of the experimental animal data for vinyl chloride (Cogliano and Parker, 1992). The report concludes that vinyl chloride provides an example of a chemical for which both toxicologic and pharmacokinetic evidence exists to override the default assumption of an equal risk over a lifetime under all age exposure conditions. In the 1994 edition of its Health Effects Assessment Summary Tables (HEAST), USEPA emphasizes that the slope factor and unit risk values for vinyl chloride do not reflect state-of-the-art science for vinyl chloride (USEPA, 1994b; 1994c), indicating that they do not reflect sensitivity to vinyl chloride from exposures at a young age, as identified by Cogliano.

In 1993, USEPA, DTSC, and OEHHA co-authored a presentation which applied this young-age sensitivity methodology to action levels for relocation criteria at Operating Industries, Inc., a Superfund site in southern California (Hiatt *et al.*, 1994).

There is good evidence that experimental animals are more sensitive to developing cancer as adults when exposed to vinyl chloride at a young age, compared to those exposed only as adults. However, the assessment methodology used by USEPA is relatively new and has not been fully evaluated or accepted for use in this type of health risk assessment. As mentioned, the unit risk value developed by OEHHA (20×10^{-5} per ppb) accounts for age specific sensitivity, and is used in this addendum report.

TABLE 3
(United States Environmental Protection Agency)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY'S ESTIMATES OF RISK FOR APPORTIONED LIFETIME EXPOSURES TO VINYL CHLORIDE AT VARIOUS CONCENTRATIONS

Estimated Excess Lifetime Cancer Risk to Humans From a 4 Year Exposure:

	Apportioned Lifetime Risk					
Age*	1 ppm	100 ppb	10 ppb	1 ppb	0.1 ppb	0.2 ppb
0 to 5	2.3 E-1	2.3 E-2	2.3 E-3	2.3 E-4	2.3 E-5	4.6 E-5
6 to 9	5.8 E-2	5.8 E-3	5.8 E-4	5.8 E-5	5.8 E-6	1.2 E-5
10 to 13	4.6 E-2	4.6 E-3	4.6 E-4	4.6 E-5	4.6 E-6	9.3 E-6
14 to 17	3.5 E-2	3.5 E-3	3.5 E-4	3.5 E-5	3.5 E-6	7.0 E-6
18 to 21	2.3 E-2	2.3 E-3	2.3 E-4	2.3 E-5	2.3 E-6	4.6 E-6

* Age range during 4 year exposure period.

Source: United States Environmental Protection Agency (USEPA), "Cancer Risk Estimates for Vinyl Chloride." Memorandum dated September 28, 1992 from Jim Cogliano, Chief, Carcinogen Assessment Statistics and Epidemiology Branch, Office of Health and Environmental Assessment, Washington, D.C., to Arnold Den, Science Advisor, Region IX, San Francisco, California, with September 26, 1989 memorandum attached.

Risk estimates are based on USEPA's cancer slope factor for vinyl chloride of 0.29 $(mg/kg\text{-}d)^{-1}$.

CMA 114341

EXPOSURE ASSESSMENT

This section reviews the types, frequency, magnitude, and duration of residential exposures to vinyl chloride from the B.K.K. Landfill based on ambient vinyl chloride monitoring data, activities at the landfill, and odor complaints from residents around the landfill. In this section, the ambient vinyl chloride monitoring data are divided into four periods: 1981-1982 data, 1983-1990 data, combined 1981-1990 data, and 1993-1994 data. The data collected during the decade of the 1980s are displayed several ways (tables, line graphs, combined graphs, frequency graphs) in order to view exposures from different perspectives. Exposures to other hazardous chemicals detected in ambient air are also evaluated in this section.

Ambient Vinyl Chloride Monitoring Data

1981-1982 Data

In October 1980, odor complaints from residents living near the landfill prompted SCAQMD to monitor for organic compounds in the vicinity of the facility; in May 1981, vinyl chloride was detected above the ambient air quality standard of 10 ppb (DHS-ARB-SCAQMD, 1983). In June 1981, SCAQMD began monitoring at six locations. **TABLE 4** shows the number of days the ambient air quality standard was exceeded at each of the six locations, the maximum concentration detected at each of the six locations, and the number of valid sample days per month (SCAQMD, 1982a). Twenty-four hour integrated bag samples were collected and analyzed by gas chromatograph-flame ionization with a detection limit of 10 ppb (SCAQMD, 1982b; 10 ppb is equivalent to 0.01 ppm). Residence 1 became Station A, located at the intersection of Mariena and Nogales Streets, 450 feet southeast of the landfill property line. Residence 2 became Station B, located on Lynn Court, immediately adjacent to the southern property line. **FIGURE 4**, from the interim report, shows the locations of Stations A, B, and MY.

Monitoring continued at Stations A and B in 1982. The detection limit for vinyl chloride was lowered from 10 ppb to 2 ppb during the 90-day monitoring program conducted by the agencies, DHS-ARB-SCAQMD. The 1981-1982 data were reviewed in the exposure characterization report and deemed not to be useful for exposure assessment because of the detection limit of 10 ppb (CH2M Hill, 1988). They were not included in the interim report because of the change in the detection limit during the monitoring period.

However, this addendum report spans long-term monitoring data in which the detection limit for vinyl chloride continues to be lowered as technology advances. Detections at or above the detection limits are assumed to be real detections in each monitoring period.

Average annual concentrations in this addendum report are calculated using the arithmetic mean, rather than the geometric mean used in the interim report. USEPA considers "the arithmetic mean is appropriate regardless of the pattern of daily exposures over time or the type of statistical distribution that might best describe the sampling data. The geometric mean of a set of sampling results, however, bears no logical connection to the cumulative intake that

TABLE 4
(South Coast Air Quality Management District)

1981 VINYL CHLORIDE CONCENTRATION, PPM AT BKK VICINITY

LOCATION	MONTHS	DAYS[a]	MAX[b]	DAYS OF VALID DATA
Suburban Water Company 163	May	0	<.01	1
	June	0	< 01	26
	July	0	< 01	30
	Aug	0	<.01	29
	Sept[c]	0	< 01	3
Suburban Water Company 164	June	0	< 01	21
	July	0	<.01	30
	Aug	0	< 01	29
	Sept	0	<.01	3
Residence 1	June	10	.03	25
	July	9	.03	30
	Aug	8	.03	29
	Sept	5	.01	22
	Oct	9	.04	30
	Nov	9	.02	24
	Dec	12	.02	29
Residence 2	June	14	.05	25
	July	9	.02	30
	Aug	12	.02	29
	Sept	9	03	22
	Oct	12	.03	30
	Nov	11	.03	24
	Dec	7	.02	29
Residence 3	June	3	.01	25
	July	0	<.01	5
Residence 4	July	3	.01	25
	Aug	9	.02	29
	Sept	0	<.01	3

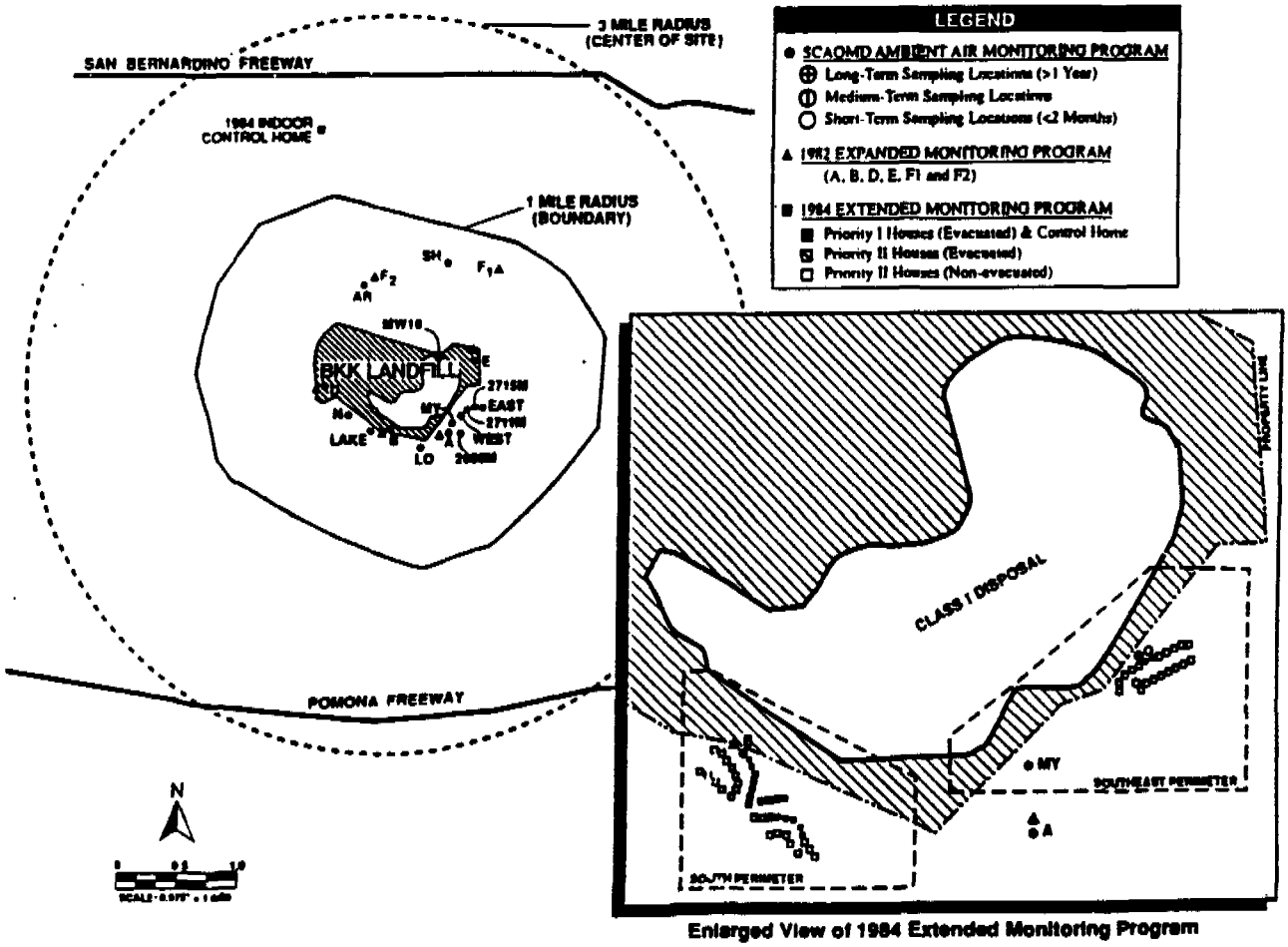
a) Days - Number of days exceeding state standard, VC ≥ .01 ppm, 24-hour average.
b) Max - Single highest daily average in ppm.
c) Sampling was discontinued on Sept. 3, 1981 at all locations except at Residence 1 and 2.

Source: South Coast Air Quality Management District, "Vinyl Chloride in the South Coast Air Basin, May 1982," El Monte, California, p. 18.
Note: PPM = Parts per million; 1 ppm = 1,000 parts per billion (ppb); 0.05 ppm = 50 ppb.
See TABLE 5 for monthly and annual average concentrations of vinyl chloride. Residence 1 = Station A; Residence 2 = Station B.

FIGURE 4
(CH2M Hill)

LOCATION OF SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT'S
AMBIENT VINYL CHLORIDE MONITORING STATIONS A, B, AND MY

FIGURE 7: Air Monitoring Programs, BKK Landfill, West Covina, CA (Based on CH2MHill Fig. 5-21 and Plate 15)



Enlarged View of 1984 Extended Monitoring Program

Source: CH2M Hill, "BKK Landfill Exposure Characterization Report, West Covina, California," August 15, 1988, Volume I, p. 5-165 (Figure 5.21) and Volume II, Plate 15.

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would result from long-term contact with the site contaminants, and it may differ appreciably from - and be much lower than - the arithmetic mean" (USEPA, 1992a). Calculating average annual concentrations when a certain portion of the data are below the detection limit requires exchanging samples with nondetectable concentrations for a value. USEPA recommends substituting half the detection limit for samples with nondetectable concentrations (USEPA, 1989b). When more than half the data are samples with nondetectable concentrations, assigned values may result in biased or imprecise estimates (Hornung and Reed, 1990). There is uncertainty associated with the substituted value since nondetected concentrations could range from zero to just below the detection limit. The "true" level cannot be known.

TABLE 5 supplements the information in TABLE 4 by providing monthly and annual arithmetic average concentrations of vinyl chloride at Stations A and B during 1981-1982. The data were collected for 16 of the 24 months. SCAQMD's detection limit between June 1981 through September 1982 was 10 ppb, and between October through December 1982, 2 ppb. Results of the data collected between October 16 and December 31, 1982, were reported in terms of the ambient air quality standard (10 ppb) rather than the detection limit (2 ppb). Consequently, the number of samples with nondetectable concentrations in this portion of the data are likely over-reported.

Approximately 60 percent of the data in 1981 and 80 percent of the data in 1982 were samples with nondetectable concentrations. Substituting a value of half the detection limit (that is, a value of 5 ppb) for samples with nondetectable concentrations in this data set may result in estimated average concentrations that artificially reflect the detection limit. When a value of 1 ppb (half the detection limit for the 1983-1990 data) is substituted in the 1981-1982 data, the estimated average annual concentrations for 1982 are more consistent with the 1983 average annual concentrations. This suggests that 1 ppb is a more reasonable substitution value for the 1981-1982 data. This assumption may underestimate exposures since some samples may have had concentrations of vinyl chloride below 10 ppb but above 2 ppb. In the 1983 data, the percent of samples with nondetectable concentrations at the detection limit of 2 ppb decreased to 53-56 percent. The average values in TABLE 5 were calculated using 1 ppb for samples with nondetectable concentrations.

FIGURE 5 shows the percent of days during 1981 and 1982 when vinyl chloride was detected above the ambient air quality standard of 10 ppb at Station A or B (ARB, 1983).

TABLE 5

1981-1982 AVERAGE VINYL CHLORIDE CONCENTRATIONS IN AMBIENT AIR (ppb)
BKK LANDFILL, WEST COVINA, CALIFORNIA

		1981	1982
Station A (Residence 1)	January	--	2.0
	February	--	2.5
	March	--	2.5
	April	--	--
	May	--	--
	June	10.5	--
	July	4.4	8.1
	August	7.0	4.9
	September	7.0	3.2
	October	8.2	3.1
	November	9.2	1.7
	December	4.1	2.8
Annual Average		**7.2**	**3.4**
Station B (Residence 2)	January	--	3.3
	February	--	3.1
	March	--	3.4
	April	--	--
	May	--	--
	June	7.5	--
	July	4.7	1.0
	August	5.0	2.2
	September	3.1	1.9
	October	6.9	3.5
	November	8.4	2.1
	December	6.6	5.0
Annual Average		**6.0**	**2.8**

Source: Letter regarding South Coast Air Quality Management District's continuous sampling and analysis project for vinyl chloride monomer. Letter dated April 9, 1982 from Edward Camarena, Director of Enforcement, Headquarters, South Coast Air Quality Management District, El Monte, to Mr. Joseph Pantalone, California Air Resources Board, Research Division, Sacramento, California.

A value of 1 ppb was substituted for samples with nondetectable concentrations. Detection limits: 6/81-9/82, 10 ppb; 10/82-12/92, 2 ppb.

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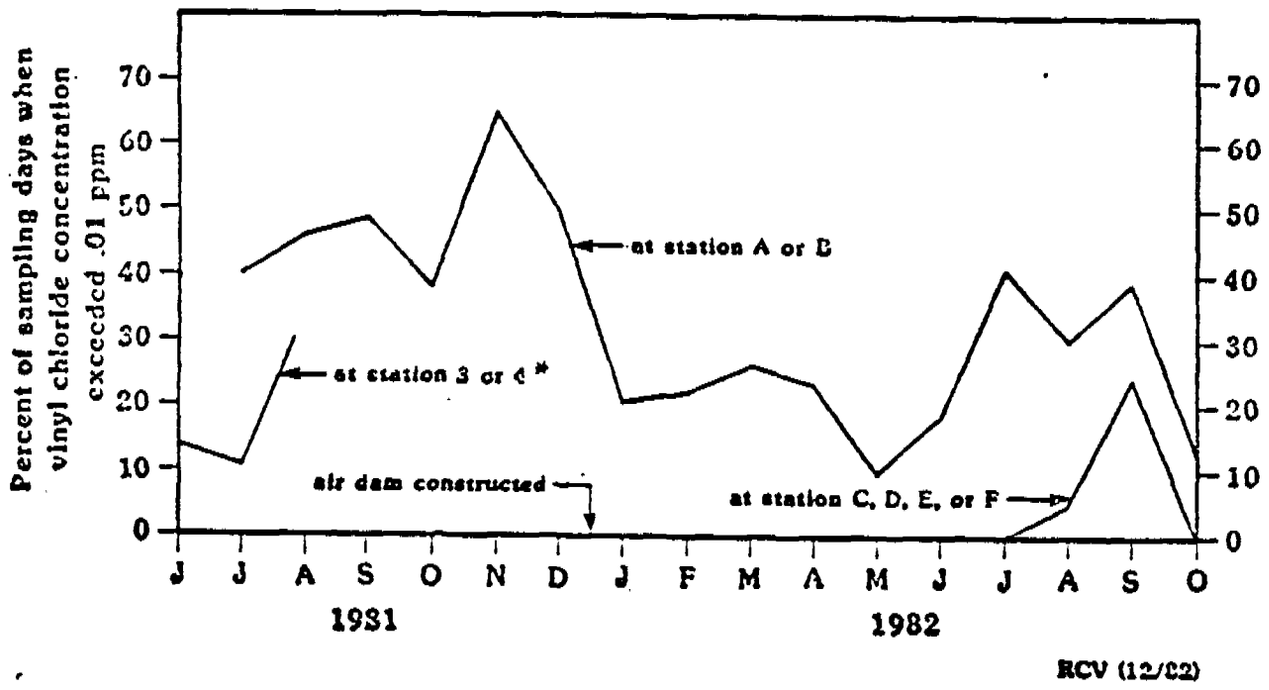
FIGURE 5
(Air Resources Board)

1981-1982 AMBIENT CONCENTRATIONS OF VINYL CHLORIDE
NEAR BKK LANDFILL



Source: Air Resources Board, Figure 1 of "Developments in the BKK Landfill Situation," Memorandum dated January 12, 1983 from James J. Morgester, Chief, Enforcement Division to James Boyd, Executive Officer. Taken from BKK Landfill files, Stationary Source Division, Sacramento, California.

CMA 114347

1983-1990 Data

SCAQMD continued almost daily ambient air monitoring for vinyl chloride at residential Stations A and B with a detection limit of 2 ppb beginning in January 1983. On June 27, 1984, SCAQMD established Station MY, located on Myra Court, 150 feet southeast of the B.K.K. Landfill property line (FIGURE 4).

SCAQMD continued monitoring for vinyl chloride at these three residential stations through March 1990. The detection limit for vinyl chloride changed during that period as follows: between 1983 through 1987, the detection limit was 2 ppb; from January 1988 through September 1989, 1.4 ppb; and between October 1989 through March 1990, 2 ppb. Although SCAQMD's laboratory could achieve a lower detection limit in 1988, the time to process the analysis increased. SCAQMD considered the advantage of a quicker processing time outweighed the small increment in sensitivity, and so returned to using the 2 ppb detection limit. The three residential monitoring stations were dismantled after March 1990 because vinyl chloride was not detected at or above 2 ppb at any of the three stations for two consecutive months.

Beginning in 1987, the frequency of samples with nondetectable concentrations was more than 50 percent at all 3 stations. Substituting the value of half the detection limits for samples with nondetectable concentrations beginning in 1987 may overestimate the average annual concentrations.

TABLE 6 presents the average annual and maximum concentrations of vinyl chloride detected at Stations A, B, and MY between January 1983 through March 1990. Average annual concentrations ("means") were calculated using three different values for samples with nondetectable concentrations: mean 1 assumed one-half the detection limits; mean 2 assumed zero ppb; mean 3 assumed the detection limits. Although Station MY operated for only the last 6 months of 1984, data collected at Station MY between August through December 1984 show 153 days of vinyl chloride monitoring, for which the average concentration was 5.43 ppb.

FIGURE 6 displays the average annual concentrations of vinyl chloride detected at Stations A, B, and MY between January 1983 and March 1990. **FIGURE 7** displays on a monthly basis the frequency of vinyl chloride detected at or above 2 ppb at Stations A, B, and MY.

Appendix D contains a statistical evaluation of SCAQMD's 1983-1990 ambient vinyl chloride monitoring data.

TABLE 6
(Ogden Environmental and Energy Services Co., Inc.)

1983-1990
AVERAGE ANNUAL VINYL CHLORIDE CONCENTRATIONS (PPB)
NEAR BKK LANDFILL

	Monitoring Station A				Monitoring Station B				Monitoring Station MY			
	Max Value	Mean 1	Mean 2	Mean 3	Max Value	Mean 1	Mean 2	Mean 3	Max. Value	Mean 1	Mean 2	Mean 3
1983	19 0	2 84	2.32	3.37	16 0	2.98	2.42	3 54	-	-	-	-
1984	21.0	4.17	3.76	4 57	23.0	3.93	3 56	4 30	-	-	-	-
1985	16 0	3 45	3.02	3.87	15 0	2 29	1.74	2.84	20 0	5.36	5.07	5 66
1986	11.0	1 92	1 26	2.58	8.0	1.33	0 49	2 18	14.0	3 02	2 57	3 46
1987	7 0	1 67	0.94	2 40	9.0	1.16	0 25	2 07	15 0	2.60	2.04	3.15
1988	7.4	1.30	0.77	1.83	9.4	0 76	0.08	1.44	15.0	2 48	2 10	2.87
1989	2.4	0 79	0.03	1.56	2 4	0 78	0.01	1 56	3.6	0.92	0.21	1 62
1990(a)	1 0*	1.00*	0.00	2.00	1.0*	1 00*	0 00	2 00	2 2	1.04	0.07	2.01

Means 1, 2, and 3 calculated with the following assumptions regarding below detectable vinyl chloride measurements:

Mean 1 - assume one-half detection limit
Mean 2 - assume zero ppb.
Mean 3 - assume detection limit.

For monitoring periods 1983-1987 and 1990, reported instrument detection limit was 2 0 ppb.
For January 1988 through September 1989, detection limit was 1 4 ppb.
(a) Data collected only in January, February, and March during 1990.

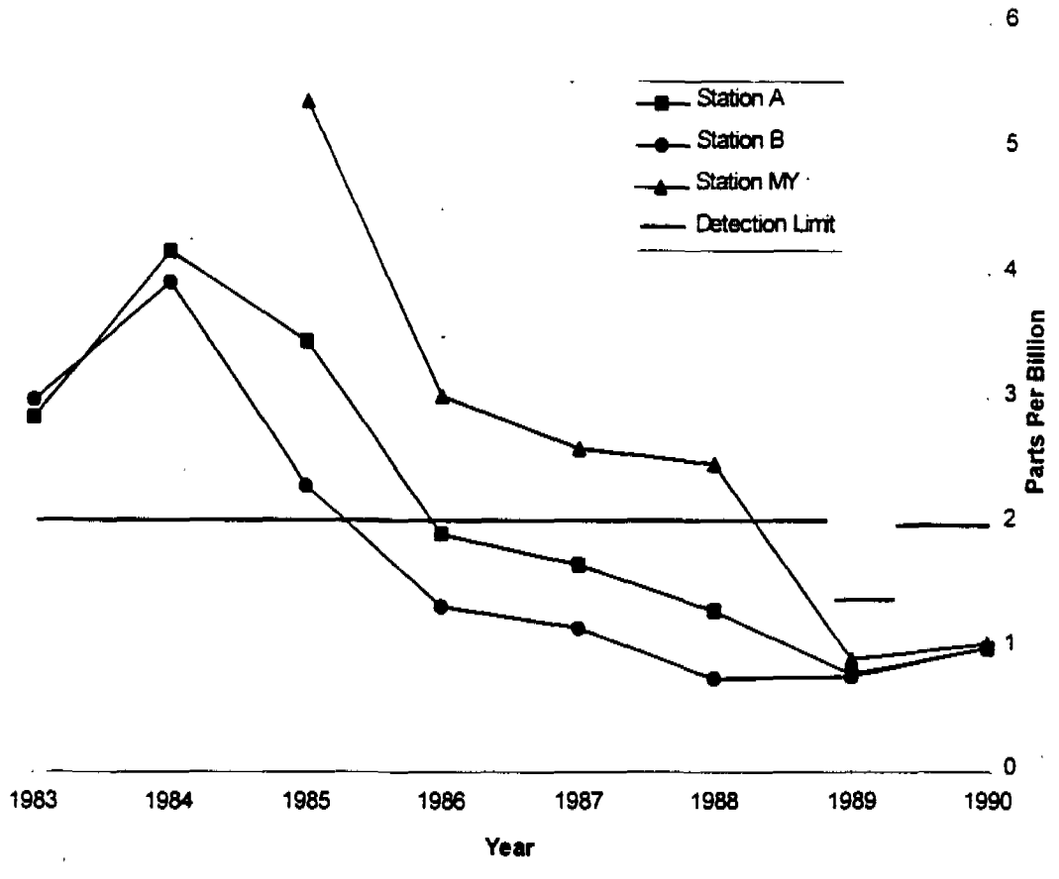
* All nondetects (asterisks not in original report)

Source: "Table 4-3, Air Quality Modeling for the BKK Landfill Air Toxic Exposure Study," Ogden Environmental and Energy Services Co., Inc., November 1992, San Diego, California , pg. 32. Derived from South Coast Air Quality Management District's daily ambient vinyl chloride monitoring data, January 1983 through March 1990.

Although Station MY operated for only the last 6 months of 1984, data collected at Station MY between August and December 1984 show 153 days of vinyl chloride monitoring, with an average concentration of 5.43 ppb.

FIGURE 6

1983-1990 AVERAGE ANNUAL VINYL CHLORIDE CONCENTRATIONS (PPB)
NEAR BKK LANDFILL, WEST COVINA, CALIFORNIA



Source: Figure derived from Table 6 of this draft addendum report "1983-1990 Average Annual Vinyl Chloride Concentrations (ppb) Near BKK Landfill." The source for Table 6 was "Air Quality Modeling for the BKK Landfill Air Toxic Exposure Study," Ogden Environmental and Energy Service Co., Inc., November 1992, San Diego, California.

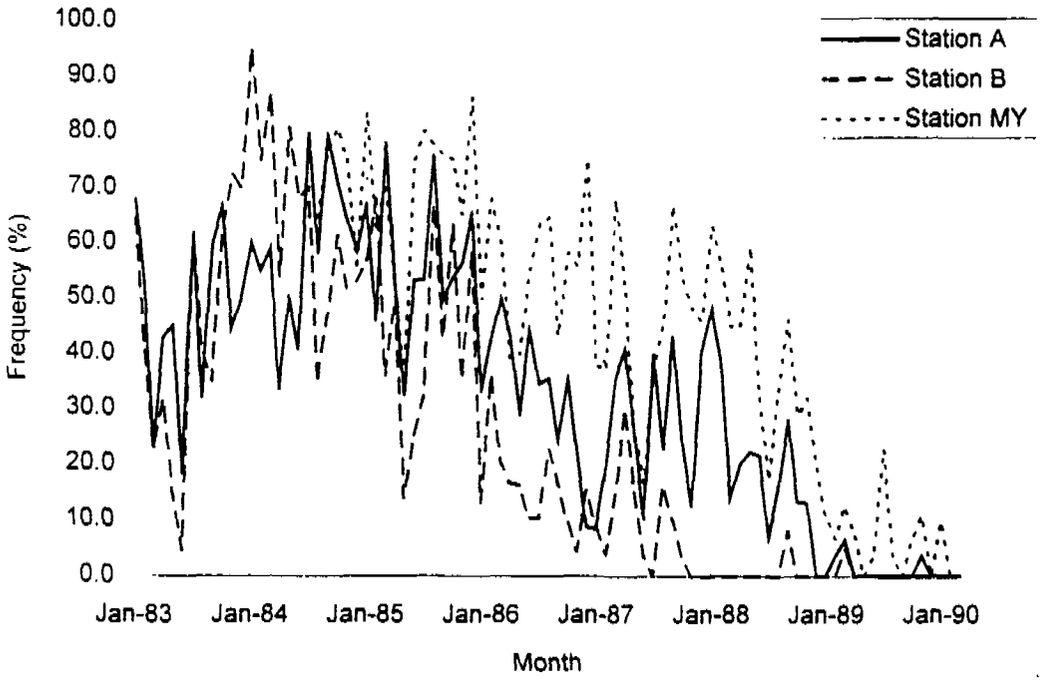
The slight increase in 1990 reflects the fact that 1 ppb was substituted for nondetects in the data, and data was only collected in January, February, and March of 1990.

Nondetected concentrations were given the value of half the detection limit. Detection limit: 1983-1987, 2 ppb; January 1988-September 1989, 1.4 ppb; October 1989-March 1990, 2 ppb.

CMA 114350

FIGURE 7

FREQUENCY OF VINYL CHLORIDE DETECTED AT OR ABOVE 2 PPB, 1983-1990
BKK LANDFILL, WEST COVINA, CALIFORNIA



Source: Figure derived from South Coast Air Quality Management District's ambient vinyl chloride monitoring data, January 1983 through March 1990.

CMA 114351

Combined 1981-1990 Data

TABLE 7 shows the number of samples with nondetectable concentrations per number of days sampled at Stations A, B, and MY for SCAQMD's combined 1981-1990 ambient vinyl chloride monitoring data.

FIGURE 8 displays the frequency of vinyl chloride detected at or above the ambient air quality standard of 10 ppb at Stations A and B between 1981 through 1989. **FIGURE 9** shows on a monthly basis the maximum measured vinyl chloride concentrations detected at Stations A, B, and MY between 1983 and 1990. The last year in which detections of vinyl chloride at or above 10 ppb occurred at each station are as follows:

- at Station A, in 1986, once in January (10 ppb) and once in February (11 ppb);
- at Station B, once in May 1985 (15 ppb);
- at Station MY, nine days in 1988. (See also next data group for one detection above 10 ppb in June 1991.)

Control measures at the landfill were effective in reducing vinyl chloride emissions over time. By 1989, vinyl chloride was detected at or above the detection limit of 2 ppb only 31 times at Station MY, 4 times at Station A, and once at Station B. During the first three months of 1990, vinyl chloride was only detected three times at or above 2 ppb; all three detections occurred at Station MY and in January.

TABLE 7

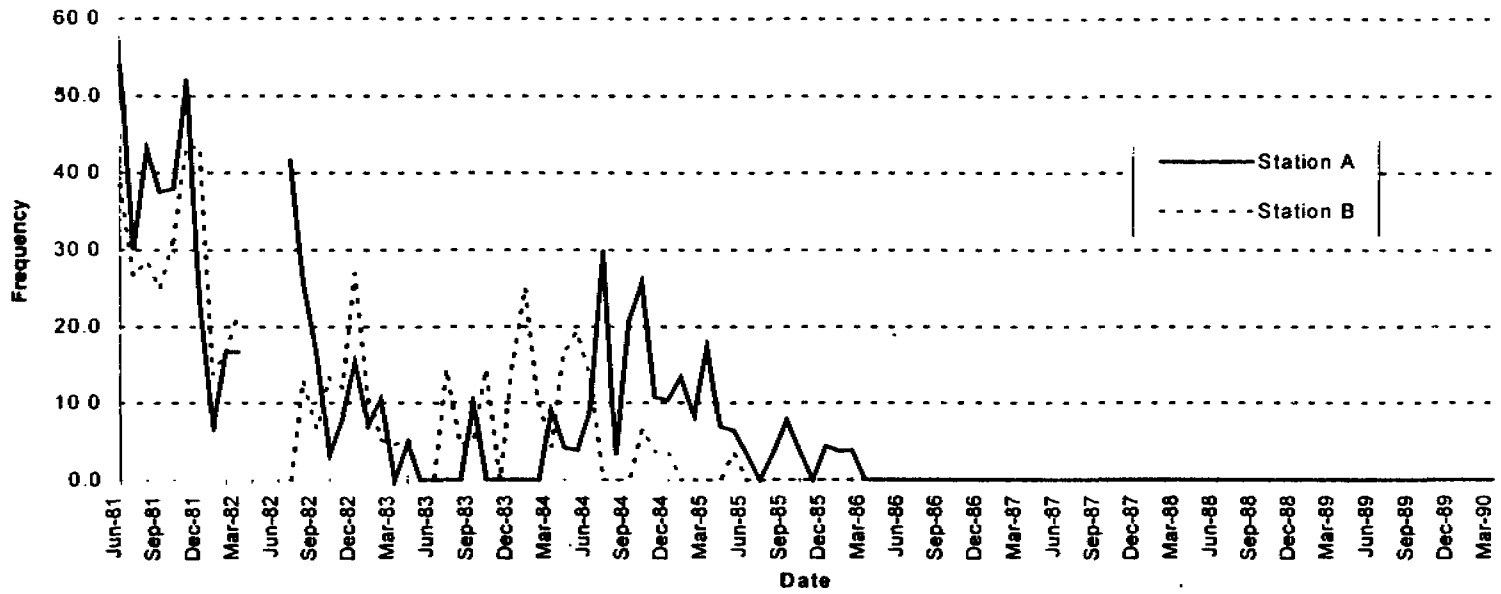
NUMBER OF SAMPLES WITH NONDETECTABLE VINYL CHLORIDE CONCENTRATIONS PER TOTAL NUMBER OF DAYS SAMPLED
1981-1990 SCAQMD AMBIENT VINYL CHLORIDE MONITORING DATA
BKK LANDFILL, WEST COVINA, CALIFORNIA

	Station A			Station B			Station MY		
	# Non-detects	# Days Sampled	Percent %	# Non-detects	# Days Sampled	Percent %	# Non-detects	# Days Sampled	Percent %
1981	116	191	60	125	191	65	-	-	-
1982	181	225	80	185	224	83	-	-	-
1983	134	254	53	142	252	56	-	-	-
1984	123	303	41	113	304	37	-	-	-
1985	137	321	43	160	289	55	90	307	29
1986	222	337	66	284	338	84	152	344	44
1987	246	338	73	305	335	91	191	345	55
1988	240	317	76	316	327	96	180	329	55
1989	345	349	99	325	326	99	312	344	91
1990	88	88	100	81	81	100	85	88	97

SCAQMD = South Coast Air Quality Management District
Detection Limits = 6/81-9/82, 10 ppb; 10/82-12/87, 2 ppb; 1/88 - 9/89, 1.4 ppb; 10/89 - 3/90, 2 ppb

FIGURE 8

FREQUENCY OF VINYL CHLORIDE DETECTED AT OR ABOVE 10 PPB, 1981-1989
BKK LANDFILL, WEST COVINA, CALIFORNIA

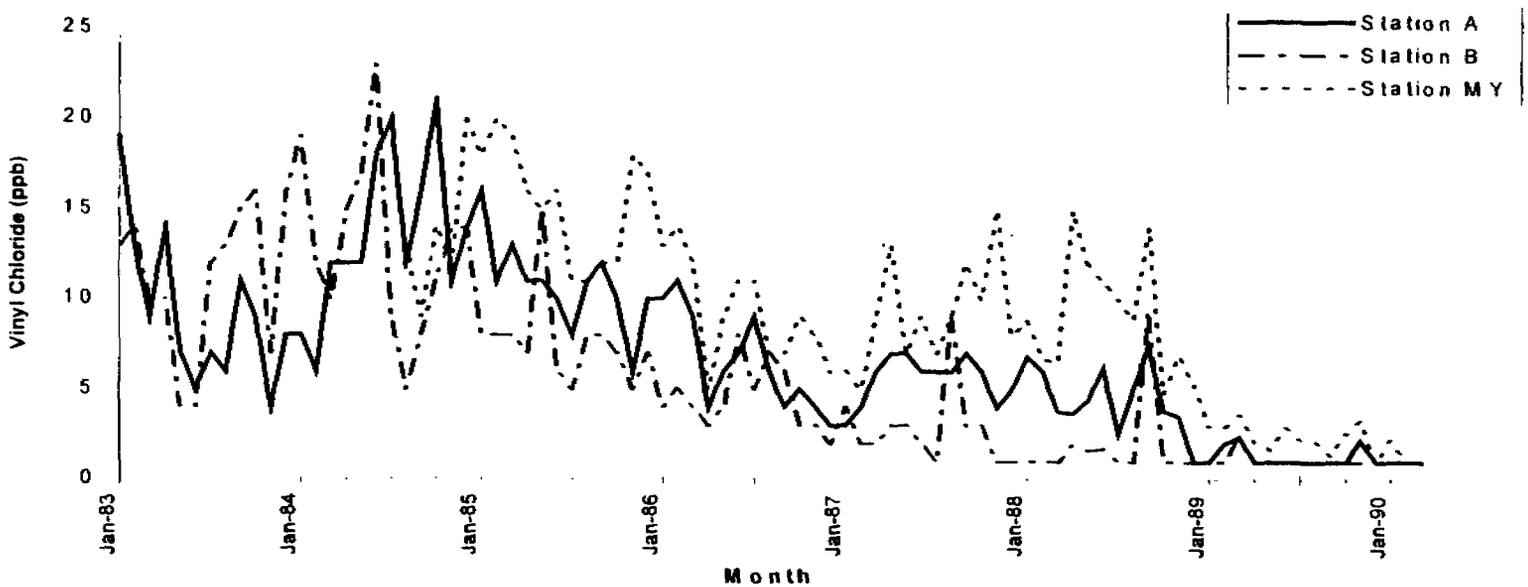


Source: Figure derived from South Coast Air Quality Management District's ambient vinyl chloride monitoring data, January 1983 through March 1990.

CMA 114354

FIGURE 9

MAXIMUM MEASURED VINYL CHLORIDE LEVEL EACH MONTH, 1983-1990
BKK LANDFILL, WEST COVINA, CALIFORNIA



Source: Figure derived from South Coast Air Quality Management District's ambient vinyl chloride monitoring data, January 1983 through March 1990

CMA 114355

1993-1994 Data

Between 1990, when SCAQMD discontinued the almost daily ambient monitoring for vinyl chloride at residential locations, and 1993, when the B.K.K. Corporation began the Proposition 65 ambient vinyl chloride monitoring every 12 to 14 days, data was collected under 2 regulatory programs: USEPA's Site Assessment and Mitigation (SAM) Workplan (Stirrat, 1991) and SCAQMD's Rule 1150.1.

- Under an order issued by USEPA in 1986, the SAM workplan required the B.K.K. Corporation to determine the extent of contamination on and off the landfill. In the ambient air monitoring component of the workplan, samples were collected for a total of 33 days between December 1989 and August 1990, and analyzed for 20 volatile organic compounds. The detection limit for vinyl chloride was reported to be 0.1 ppb. Data for the station located at Myra Court showed that of the total 33 samples, vinyl chloride was detected in 21 samples as follows: above 0.1 ppb, 14 times; above 1 ppb, 5 times; above 2 ppb, twice, with a maximum detection of 2.73 ppb in June 1990 (Stirrat, 1990). USEPA's validation of the data concluded that the detected results and quantitation limits for vinyl chloride were estimates "and usable for limited purposes only"; additionally, "the detected results and quantitation limits for many of the target analytes in all samples were qualified as the laboratory did not run initial calibration curves" (ICF Technology Incorporated, 1992).

- Ambient air monitoring according to SCAQMD's Rule 1150.1 found vinyl chloride south of the landfill as follows: offsite at Myra Court at 10.2 ppb in June 1991; onsite near Nogales Street at 1.38 ppb in January and 1.40 ppb in June, 1992; and onsite at Nogales Street at 0.12 ppb in February 1993.

Based on the ambient air data collected at the perimeter of the landfill under SCAQMD's Rule 1150.1, the B.K.K. Corporation published a warning in the *San Gabriel Valley Tribune* on August 29, 1993, for detections of vinyl chloride above the Proposition 65 NSRL (B.K.K. Corporation, 1993b).

During 1993-1994, the B.K.K. Corporation added several offsite stations to monitor concentrations, collected 24-hour ambient air samples every 12 to 14 days, and updated its Proposition 65 vinyl chloride monitoring data quarterly. **TABLE 8** is B.K.K. Corporation's monitoring data for the period September 1993 through September 1994 (BKK Corporation, 1994). USEPA has stated that the analytical procedure appears to be acceptable (USEPA, 1994e), and SCAQMD found the modified protocol acceptable (SCAQMD, 1994a). **FIGURE 10** shows the locations of the B.K.K. Corporation's Proposition 65 monitoring stations. Stations #3 ("Nogales End") and #6 ("Lynn Court") are perimeter monitoring stations located on the landfill. They are used to represent the locations of SCAQMD's residential monitoring Stations MY and B, respectively. This may overestimate exposures since some dilution in the ambient air concentration would be expected between the perimeter and the residential locations. Station #10 ("Amar-at-Nogales") is used to represent the location of SCAQMD's residential monitoring Station A. This may underestimate the exposure since Amar is south of Marlena on Nogales, where Station A had been located. (Note: There was no station #8 or #9 in the Proposition 65 monitoring program.)

The detection limit for B.K.K. Corporation's data varies from 0.005 to 0.010 ppb. In TABLE 8, only values above 0.005 ppb are detected concentrations. Samples with nondetectable concentrations are given a value of half the detection limit by the B.K.K. Corporation for each day and station. The number of samples with nondetectable concentrations per total number of days sampled at each of the three stations is as follows: #10, "Amar-at-Nogales" (Station A), 4 nondetects in 17 days sampled (18 percent); #6, "Lynn Court" (Station B), 13 nondetects in 28 days sampled (46 percent); and #3, "Nogales End" (Station MY), 12 nondetects in 35 days sampled (31 percent).

TABLE 8
(BKK Corporation)

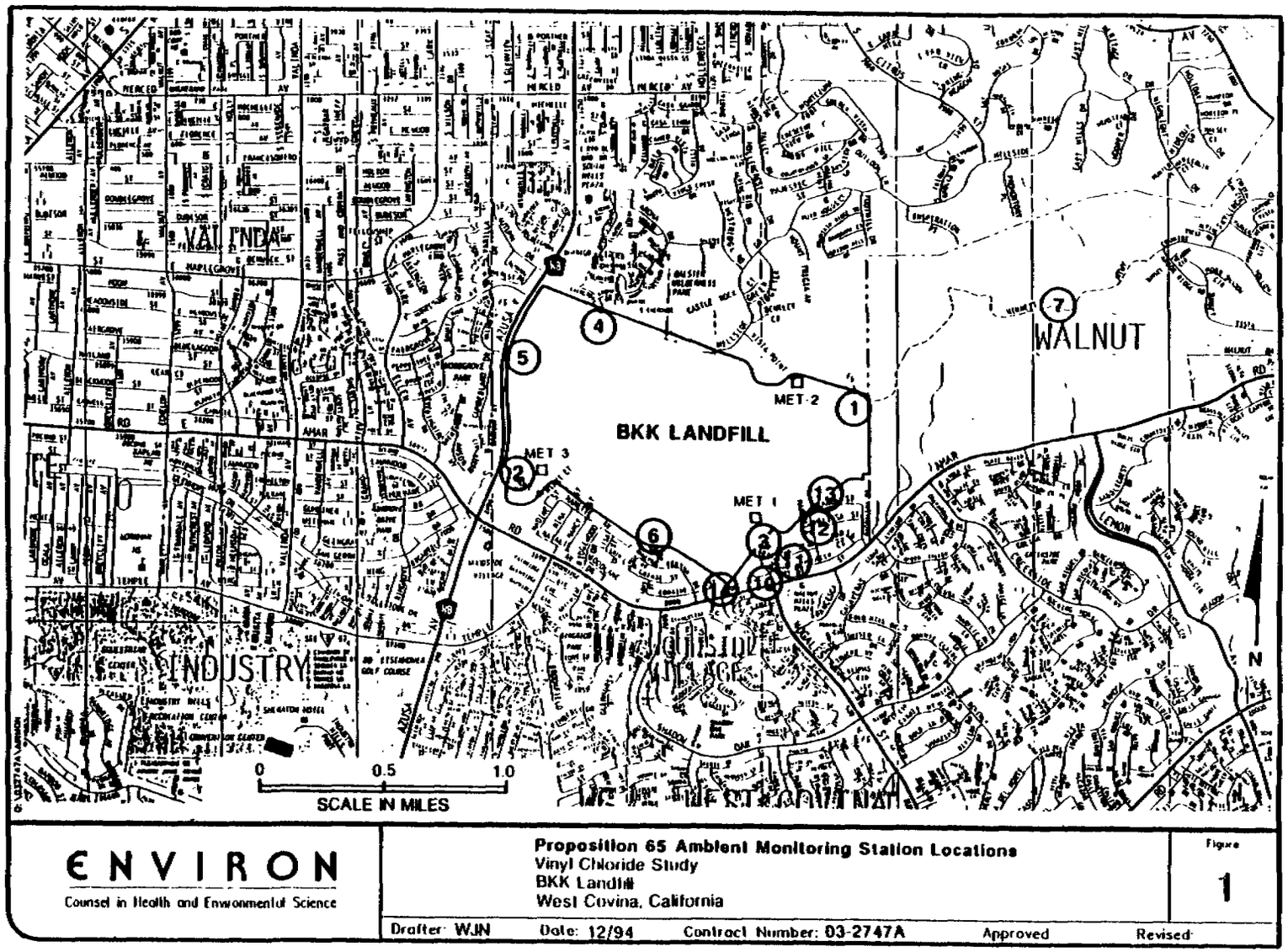
BKK LANDFILL
PROPOSITION 65 VINYL CHLORIDE MONITORING PROGRAM
Cumulative Average Last 12 Months

(Parts per billion volume/volume)

Sample Date	Sample Station & Location											
	1 Microwave Tower	2 Azusa Spillway	3 Nogales End	4 NW Gate	5 West Borrow	6 Lynn Court	10 Amar at Nogales	11 Mariena	12 Melissa at Marcella	13 Miranda Fence	14 Loraine Street	7 Walnut Village
14-Sep-93		0.0025	0.0025									
15-Sep-93		0.0025	0.0240									
16-Sep-93	0.0025	0.0025	0.0025	0.0025	0.0140	0.0025						0.0025
20-Sep-93		0.0025	0.0040									
22-Sep-93		0.0900	0.0035									
28-Sep-93	0.0560	0.0025	1.4000	0.0050	0.0040	0.0035						0.0035
12-Oct-93	0.0025	0.0025	0.0025	0.0025	0.0090	0.0025						0.0025
21-Oct-93	0.0030	0.0030	0.0035		0.0025	0.0030						0.0025
08-Nov-93	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025						0.0025
15-Nov-93	0.0025	0.0025	0.0030	0.0030	0.0025	0.0025						0.0030
07-Dec-93	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025						0.0025
16-Dec-93	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025						0.0025
29-Dec-93	0.0025	0.0260	0.5100	0.0035	0.0150	0.1000						0.0025
11-Jan-94	0.0140	0.0250	0.7100	0.0045	0.0150	0.0470						0.0035
25-Jan-94	0.0045	0.0190	0.5800	0.0030	0.0140	0.0660						0.0045
03-Feb-94	0.0035	0.0040	0.0380	0.0040	0.0130	0.0700						0.0030
11-Feb-94			0.3200									
16-Feb-94	0.0045	0.0040	0.1400	0.0045	0.0025	0.0720	0.0520					0.0040
01-Mar-94	0.0030		0.3400			0.0660	0.1600	0.0690	0.0580	0.0600		0 0030
15-Mar-94	0.0035	0.0260	0.2700			0.0540	0.0250	0.0590	0.0490	0.0120		0.0040
29-Mar-94	0.0045	0.0040	0.3300			0.0360	0.0620	0.0460	0.0390	0.0180	0.0640	0.0040
12-Apr-94	0.0040	0.0130	0.3100			0.0240	0.0790	0.0520	0.0210	0.0180	0 0760	0.0045
26-Apr-94	0.0035	0.0040	0.0250			0.0370	0.0035	0.0030	0.0040	0.0260	0.0040	0.0030
10-May-94	0.0040	0.0030	0.0170			0.0040	0.0040	0.0040	0.0035	0.0150	0.0050	0.0045
24-May-94	0.0040	0.0040	0.0050			0.0040	0.0045	0.0050	0.0030	0.0040	0.0055	0.0035
07-Jun-94	0.0045	0.0760	0.5800			0.0390	0.0160	0.0770	0.0940	0.0860	0.0820	0.0045
21-Jun-94	0.0050	0.0045	1.0000			0.0040	0.1600	0.1500	0.0040	0.0045	0.0035	0.0045
28-Jun-94			0.3800									
05-Jul-94	0.0050		0.0380				0.0050	0.0050	0.0035	0.0330	0.0050	
19-Jul-94	0.0045	0.0040	0.1800			0.0025	0.0240	0.0650	0.0340	0.0310	0.0240	
03-Aug-94	0.0045	0.0290	0.4400			0.0260	0.0580	0.0480	0.0380	0.0340	0.0450	
16-Aug-94	0.0040	0.0055	0.1900			0.0125	0.0370	0.0560	0.0290	0.0050	0.0300	0.0030
30-Aug-94	0.0073	0.0035	0.0045			0.0035	0.0035	0.0035	0.0035	0.0110		0.0030
13-Sep-94	0.0040	0.0500	0.4100			0.0470	0.0840	0.0320	0.0320	0.1000	0.0590	0.0030
27-Sep-94	0.0150	0.0660	0.5700			0.0610	0.1100	0.0690	0.0260	0.0880	0.0920	0.0093
Avg. 24-hr	0.0063	0.0158	0.2526	0.0033	0.0076	0.0285	0.0522	0.0465	0.0276	0.0341	0.0381	ND
Avg. ug/day	0.33	0.82	13.13	0.17	0.40	1.48	2.71	2.42	1.43	1.77	1.98	ND
No. Samples	29	31	35	12	13	28	17	16	16	16	13	26
Risk	1.3E-06	3.2E-06	5.1E-05	6.7E-07	1.5E-06	5.7E-06	1.0E-05	9.3E-06	5.5E-06	6.8E-06	7.6E-06	

Source: "BKK Landfill 3008(h) Consent Order, EPA Docket No. RCRA-09-89-0019, Monthly Progress Report for September 1994, Attachment: BKK Landfill Proposition 65 Vinyl Chlonde Monitonng Program." Letter dated October 7, 1994, from Julia M. Bussey, Corporate Manager, Regulatory Affairs, BKK Corporation, Torrance, California to Ms. Carmen Santos, United States Environmental Protection Agency-Region IX, San Francisco, California. The detection limit varies from 0.005 to 0.010 ppb. Only values above 0.005 ppb are detected concentrations. Samples with nondetectable concentrations are given a value of half the detection limit by the B.K.K. Corporation for each day and station. The number of samples with nondetectable concentrations per total number of days sampled at the three stations is as follows: #10, "Amar-at-Nogales" (Station A), 4 nondetects in 17 days sampled (18%); #6, "Lynn Court" (Station B), 13 nondetects in 28 days sampled (46%); and #3, "Nogales End" (Station MY), 12 nondetects in 35 days sampled (31%). NOTE: There was no station #8 or #9.

FIGUR.
(B.K.K. Corporation-ENVIRON Corporation)



CMA 114358

Source: Memorandum dated December 21, 1994, from ENVIRON Corporation at the request of Julia M. Bussey, Corporate Manager, Regulatory Affairs, B.K.K. Corporation, Torrance, California, to the Office of Environmental Health Hazard Assessment, Sacramento, California. Note: There was no station #8 or #9 in the Proposition 65 monitoring program.

Site Activities

Approximately 72 percent of the total 3.4 million tons of hazardous waste disposed of at the landfill between 1975 and 1984 was liquid hazardous waste (CH2M Hill, 1988). Four disposal methods were practiced between 1972 and 1984 (CH2M Hill, 1988):

- Commingling: approximately 85 percent of accepted fluids were disposed of by this method between 1972 to May 1984 (The Janes Network, 1994); bulk liquid hazardous wastes were discharged into a cavity prepared in the daily working face and commingled with solid nonhazardous wastes at a ratio of approximately one-part liquid to two-parts refuse;

- Solidification: from May 1984 through November 1984, hazardous and nonhazardous liquid wastes were mixed with soil on approximately 15 acres of the top of the Class I Unit prior to burial; gas condensate generated onsite and contaminated groundwater from extraction wells were air stripped and then disposed of by this method until November 1987, at which time the leachate treatment plant began operating.

- Drum disposal: from 1972 through 1984, closed drums were stacked on pallets then covered with soil to a total height of five feet; drums containing acid, alkaline, or solvent waste were separated from each other by unspecified distances;

- Injection wells: from 1975 through 1980, waste acids, cyanide solutions, and organic fluids were injected into 96 boreholes ("injection wells") drilled into the buried waste; these wells were 10 feet wide holes, 120 feet deep; approximately 10 percent of accepted fluids were disposed of by this method.

In 1985, USEPA prepared an aerial photographic analysis of the B.K.K. Landfill (USEPA, 1985). Twelve photographs dating from 1964 through 1984 show the expansion of the landfill, and of the residential community around the landfill. The focus of USEPA's analysis was the identification of surface drainage, seepage, leachate, disposal practices, point discharges, and vegetation damage at the landfill. Of the four disposal methods practiced at the landfill between 1972 and 1984, all but the injection well method can be seen in the photographs. Onsite disposal operations seen in the photographs include the working face/active disposal area, standing liquid, and the liquid waste disposal (solidification) area. The following figures are selected from USEPA's aerial photographic analysis: **FIGURE 11**, October 20, 1980; **FIGURE 12**, September 2, 1983; and **FIGURE 13**, June 15, 1984.

Residential developments around the landfill prior to 1980, as captured in the aerial photographic analysis, were as follows: 1964, southwest, approximately 1,000 feet from the landfill; 1968, northwest and north; 1970, no new residential development evident; 1973, south of Amar Road; and 1976, north of Amar Road. Residential developments at Marlena and Nogales Streets, location of the future monitoring Station A, were begun in 1976 and completed in 1977; developments at Lynn Court and Myra Court were begun and completed in 1979 (City of West Covina, 1994).

FIGURE 11
(United States Environmental Protection Agency)

BKK SANITARY LANDFILL, OCTOBER 20, 1980

Source: "Aerial Photographic Analysis of the BKK Sanitary Landfill, West Covina, California," January 1985, United States Environmental Protection Agency, EPA Region 9, Environmental Monitoring Systems Laboratory, Las Vegas, Nevada, pg. 21. TS-AMD-8307a/3600.

FIGURE 12
(United States Environmental Protection Agency)

BKK SANITARY LANDFILL, SEPTEMBER 2, 1983

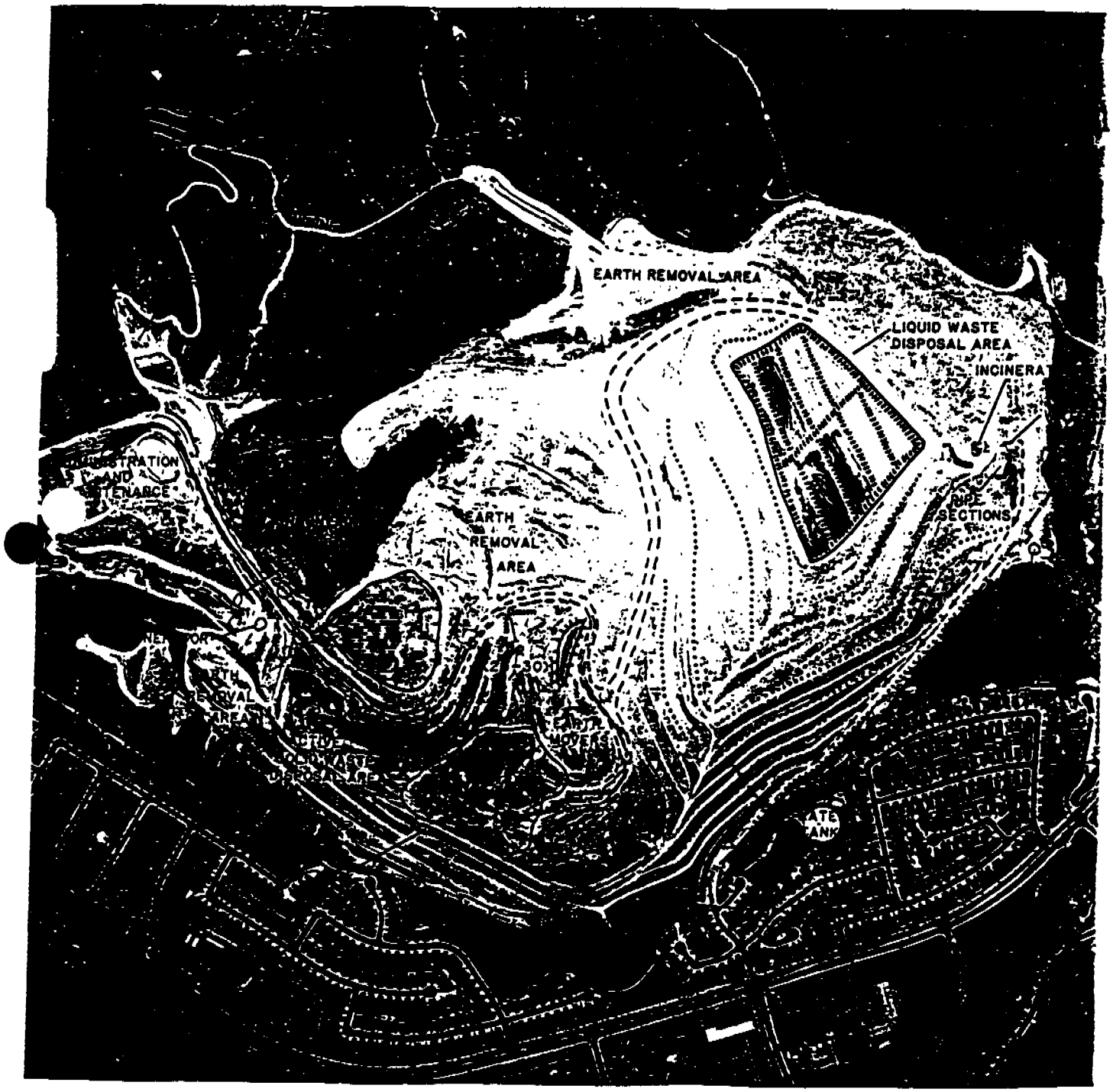
Source "Aerial Photographic Analysis of the BKK Sanitary Landfill, West Covina, California," January 1985, United States Environmental Protection Agency, EPA Region 9, Environmental Monitoring Systems Laboratory, Las Vegas, Nevada, pg. 23. TS-AMD-8307a/3600

Note: Incinerator = Flare Station

CMA 114361

FIGURE 13
(United States Environmental Protection Agency)

BKK SANITARY LANDFILL, JUNE 15, 1984



Source: "Aerial Photographic Analyses of the BKK Sanitary Landfill, West Covina, California," January 1985, United States Environmental Protection Agency, EPA Region 9, Environmental Monitoring Systems Laboratory, Las Vegas, Nevada, pg. 35. TS-AMD-8307a/3800.

Note: Incinerator = Flare Station

CMA 114362

Odor Complaints Data

Complaints of odors emanating from the B.K.K. Landfill were lodged by residents to both SCAQMD and the City of West Covina as early as 1969 (CH2M Hill, 1988). Detection of odors does not necessarily equate to exposure to hazardous substances. Odors do indicate downwind receptors and suggest the potential for exposure when hazardous chemical contaminants are also present (Ziem and Davidoff, 1992).

The first odor complaint recorded was in March 1969 to SCAQMD (SCAQMD, 1981). Odor complaints increased from 48 in 1971 to 57 in 1974. In 1974, the B.K.K. Corporation acknowledged complaints from residents situated to the north and northeast of the working face of the landfill (BKK Corporation, 1974). In 1979, B.K.K. Corporation contracted with the USC Environmental Engineering Program to investigate odors from the landfill. The study analyzed complaints received by the City during January 1, 1978 through May 16, 1979. The objectives of the study were to determine which areas were subject to odors, what climatological factors elicited an increase in complaints, identify sources of odor based on wind data, and select appropriate monitoring and sampling conditions for the next phase of the study. Results of USC's first interim report (USC, 1979) included the finding that of the 521 complaints received during the study period, a larger number were lodged during the initial four months of 1979 as compared to the same period in 1978. The majority of complaints were from residents of the "M" named streets, possibly because those residents were "closer to the working face of the B.K.K. Landfill" and at a "higher elevation consistent with the landfill" (USC, 1979). (It is assumed that the "M" named streets in the USC study included Miranda, Maria, Maureen, and Marlena Streets, developed in 1977. Marlena at Nogales Streets, location of future monitoring Station A, was developed by 1977; Lynn Court and Myra Court, locations of future monitoring Stations B and MY respectively, were developed in 1979 [City of West Covina, 1994].) USC's final report was released in February 1981 and concluded that odor problems were associated with the downslope air drainage into residential areas under nighttime meteorological conditions (USC, 1981).

In 1980, the City Council directed city staff to scientifically measure odor emissions from the landfill, so the city contracted with Eutek Inc. Eutek's study included an analysis of odor complaints by month, day of week, and hour of the day. Complaints recorded on Sundays and after-hours suggested a mechanism for odor transport other than the working face. "M" and "L" named streets were more frequently affected by odors from the landfill (Eutek, 1981).

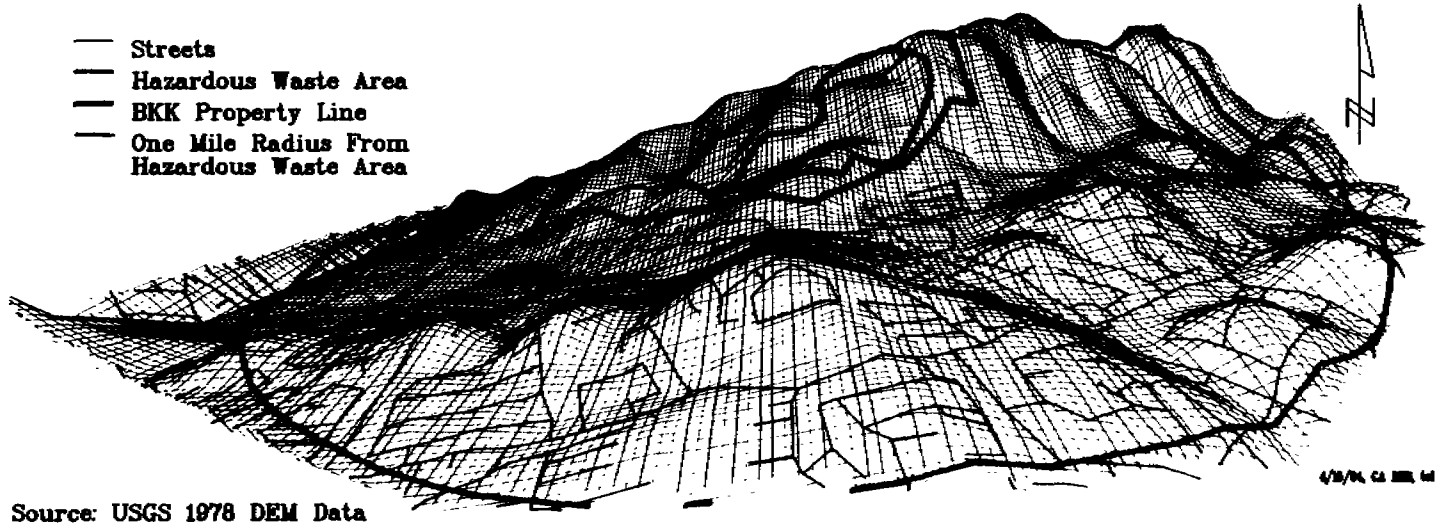
DHS-DEODC analyzed odor complaints data in conjunction with its pending reproductive outcomes study. The frequency and distribution of over 4,000 odor complaints made between 1980 and 1985 indicate that topography and meteorology contributed to odor complaints (Kharrazi *et al.*, 1993). **FIGURE 14** is a three-dimensional map of the closed Class I Unit and the residential neighborhood one-mile south of the landfill. **FIGURE 15** and **FIGURE 16** show the location of odor complaints per housing unit for the periods 1980-1982 and 1984-1985, respectively.

A 1983 tri-agency study monitored for nine chemicals. Monitoring stations were located onsite at the western and eastern ends of the landfill, and in residential areas around the landfill. The monitoring data indicated that the areas with the greatest frequency of odor complaints, that is the southeastern and southern residential areas, coincided with those areas where highest detections of vinyl chloride occurred. However, odor complaints are not necessarily considered to be an indicator of vinyl chloride exposure, since odors were reported in the residential area north of the landfill, but vinyl chloride was not detected in this area. (DHS-ARB-SCAQMD, 1983).

Updated census data for the residential community around the B.K.K. Landfill is presented in Appendix E of this report.

FIGURE 14
(Department of Health Services
Division of Environmental and Occupational Disease Control)

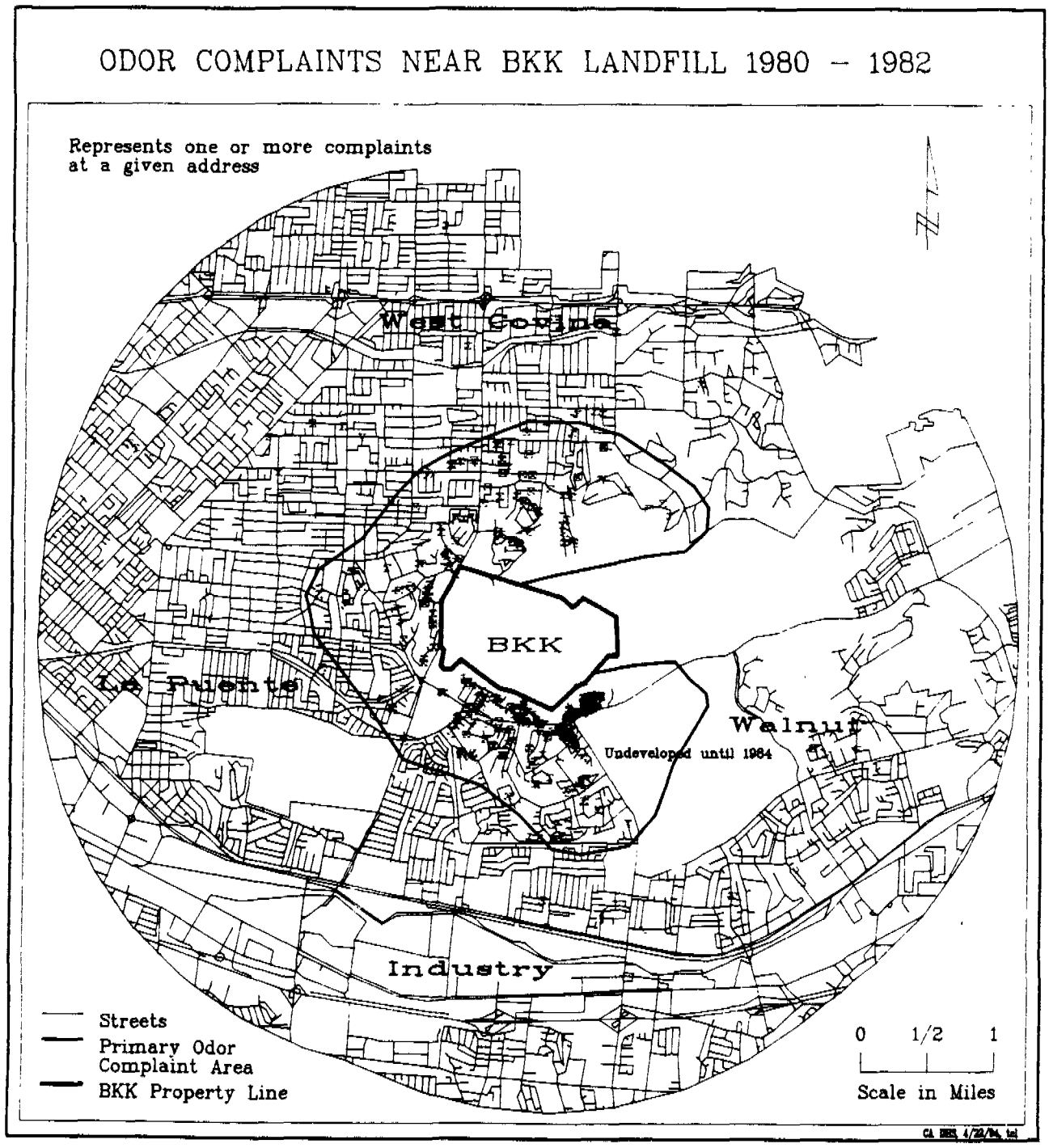
TOPOGRAPHY NEAR BKK LANDFILL, WEST COVINA, CA



CMA 114364

Courtesy of Martin Kharrazi, Ph D., Tim Lomas, and Rachel Broadwin, California Department of Health Services, Division of Environmental and Occupational Disease Control, Environmental Health Investigations Branch, Emeryville, California, April 1994

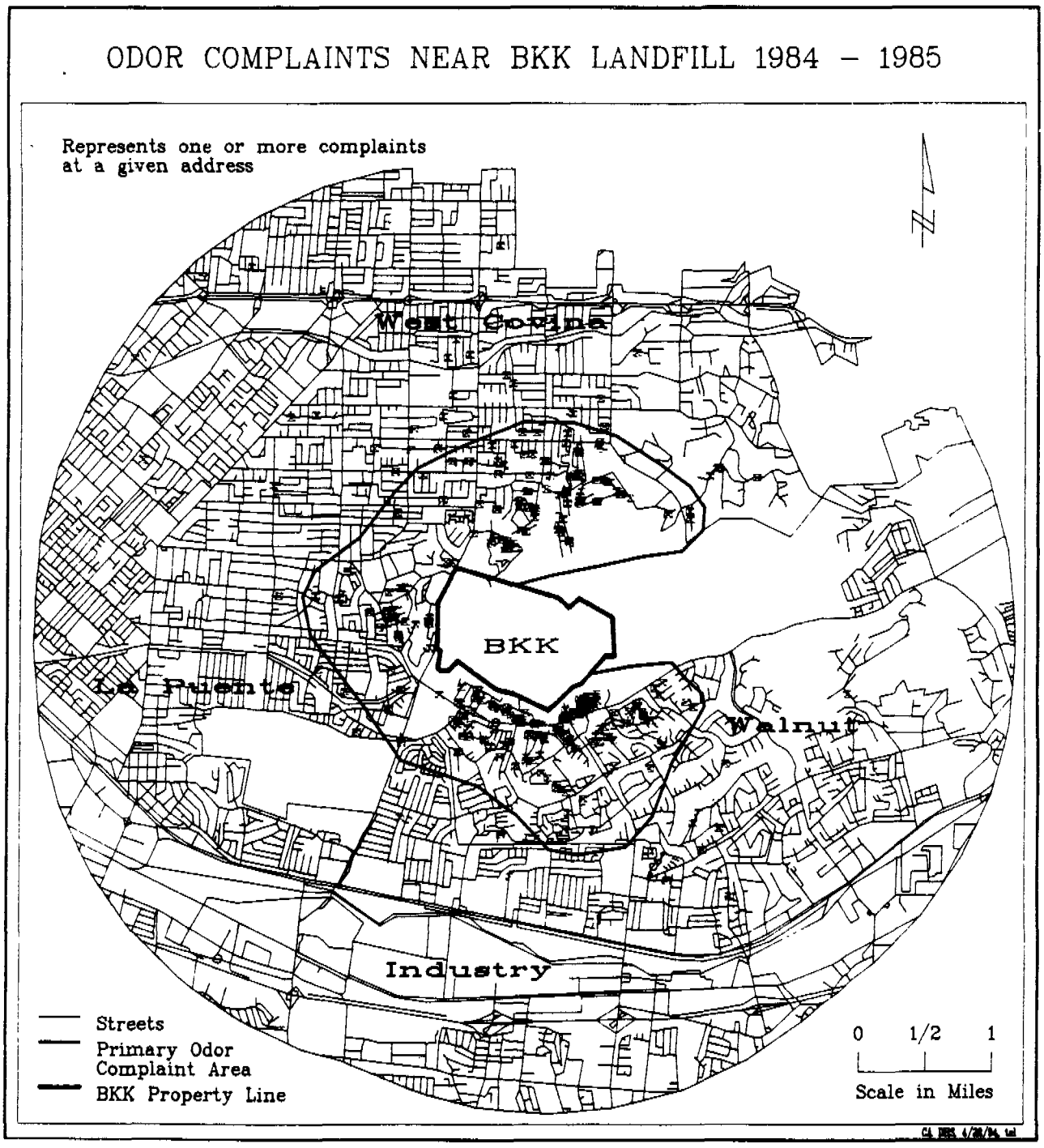
FIGURE 15
(Department of Health Services
Division of Environmental and Occupational Disease Control)



Courtesy of Martin Kharrazi, Ph.D., Tim Lomas, and Rachel Broadwin, California Department of Health Services, Division of Environmental and Occupational Disease Control, Environmental Health Investigations Branch, Emeryville, California, April 1994

CMA 114365

FIGURE 16
(Department of Health Services
Division of Environmental and Occupational Disease Control)



...esy of Martin Kharrazi, Ph.D., Tim Lomas, and Rachel Broadwin, California Department of Health Services, Division of Environmental and Occupational Disease Control, Environmental Health Investigations Branch, Emeryville, California, April 1994

CMA 114366

Other Hazardous Chemicals Detected in Ambient Air

As discussed in the Hazard Identification section of this report, the 1982 90-day ambient air monitoring program conducted by DHS-ARB-SCAQMD detected five other chemicals that are known or suspected to be human carcinogens: benzene (known), trichloroethylene, perchloroethylene, chloroform, and 1,2-dichloroethane (DHS-ARB-SCAQMD, 1983). Beginning in 1985, these chemicals were also monitored by ARB and/or SCAQMD as part of the air toxics monitoring program in the Los Angeles basin (SCAQMD, 1987). These data are not directly comparable because the data from the two sites were collected during two different years: Station A, in 1982 and Azusa Station, in 1985. Whether concentrations at Azusa Station were higher, lower, or the same in 1982 is not known. Consequently, there is insufficient information to estimate, in a manner similar to analysis of the ambient vinyl chloride monitoring data, risks associated with exposures to the other hazardous chemicals emitted from the Class I Unit of the B.K.K. Landfill. Use of flare data to project concentrations of the five other hazardous chemicals would involve additional assumptions, including extrapolation from the landfill to residential locations, and would not be consistent with USEPA's recommended guidance that past scenarios be based on actual exposure conditions (USEPA, 1989b).

In 1995 OEHHA reviewed limited 1994-1995 ambient air monitoring data regarding potential exposures to future inhabitants of Walnut Village, a residential neighborhood proposed for development east of the B.K.K. Landfill in the City of Walnut. Ambient air levels of benzene, perchloroethylene, chloroform, and carbon tetrachloride monitored at the B.K.K. Corporation's Proposition 65 monitoring stations #1, located at the northeast corner of the facility, and #7, located offsite to the northeast (see FIGURE 10) were compared to levels of the same chemicals found at four of SCAQMD's air toxics monitoring stations (Burbank, Hawthorne, Anaheim, and Azusa). From the available data OEHHA concluded that potential health risks to future inhabitants of Walnut Village would not differ significantly from the potential health risks experienced by inhabitants of other similar areas of the Los Angeles basin exposed to ambient air contaminants (OEHHA, 1995).

B.K.K. Corporation's December 1994 - December 1995 ambient air sampling program, required by USEPA, monitored for 19 volatile organic compounds. USEPA is reviewing these data for use in the risk assessment it is requiring the B.K.K. Corporation to conduct.

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RISK CHARACTERIZATION

This section presents the assumptions used in the risk calculation, the individual excess lifetime cancer risk estimates, the exposed population, estimated excess lifetime cancer risks using different assumptions and methodology, limitations and uncertainties, and the findings of the 1995 addendum health risk assessment.

The interim report included risk estimates based on exposure concentrations generated from ARB's modeling of 1987 vinyl chloride concentrations at Stations A, B, and MY (ARB Part A, 1990). In 1991, OEHHA and DHS attempted to refine the air dispersion model used in the interim report. However, the work showed that the B.K.K. Landfill was more complex than could be accounted for in air dispersion models approved for regulatory use in 1991. Appendix F contains information about the air dispersion model developed for this project, and the limitations and uncertainties which precluded its use in this addendum report. This addendum report focuses on monitored data.

Assumptions Regarding the Beginning of Exposures at Stations A, B, and MY

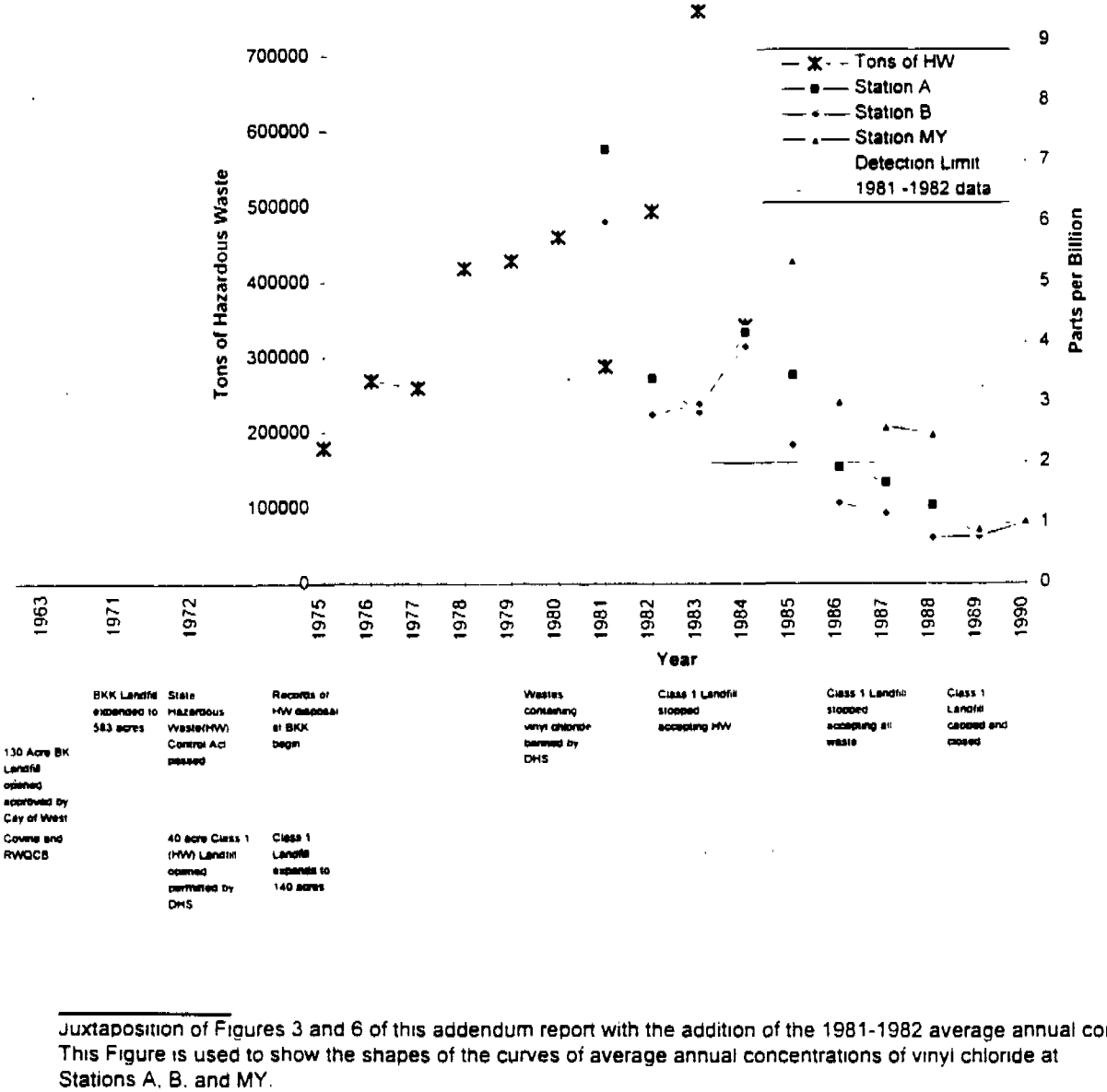
There are no monitoring data to quantify exposures at Stations A, B, and MY prior to June 1981. Odor complaints data indicate residents were reporting odors from the landfill as early as 1969, and specifically, at "M" and "L" named streets southeast and south of the Class I Unit beginning in 1978. These odor complaints indicate areas of downwind impact and suggest emissions from the Class I Unit were reaching nearby residents. Homes near Station A were built by 1977, and near Stations B and MY, by 1979. Onsite disposal practices at the time included commingling (open-face disposal) of liquid hazardous wastes.

It is assumed in this addendum report that exposures at Stations A, B, and MY began in 1980. Ascribing a value to exposures in 1980 at Stations A, B, and MY is done by looking at **FIGURE 17**. FIGURE 17 juxtaposes FIGURE 3 (tons of hazardous waste deposited in the landfill) with FIGURE 6 (average annual vinyl chloride concentrations in ppb detected between 1983 through 1990). The period of overlap is not long enough to determine if such a lag truly existed. By 1983, the landfill had been receiving wastes for 20 years. FIGURE 17 shows that:

- No emissions of vinyl chloride could have occurred before 1963, the year the landfill opened; exactly when emissions of vinyl chloride began is not known;
- The dip in the graph of hazardous waste deposited in 1981 is attributed to the prohibition of waste containing vinyl chloride after vinyl chloride was first detected above the ambient air quality standard of 10 ppb at the landfill perimeter;
- The shapes of the curves of average annual concentrations of vinyl chloride at Stations A, B, and MY in FIGURE 17 and the estimated average annual concentrations at Stations A and B in 1981-1982 from TABLE 5 suggest exposures prior to 1981.

FIGURE 17

TONS OF HAZARDOUS WASTE DEPOSITED,
AVERAGE ANNUAL VINYL CHLORIDE CONCENTRATIONS (PPB)
B.K.K. LANDFILL, WEST COVINA, CALIFORNIA



Juxtaposition of Figures 3 and 6 of this addendum report with the addition of the 1981-1982 average annual concentration This Figure is used to show the shapes of the curves of average annual concentrations of vinyl chloride at Stations A, B, and MY.
Detection Limits: 6/81/-9/82, 10 ppb; 10/82-12/87, 2 ppb; 1/88-9/89, 1.4 ppb; 10/89-3/90, 2 ppb.
Note: The increase in 1990 reflects the fact that 1 ppb was substituted for nondetects in the data, and that data were only collected for January, February, and March of 1990

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This information suggests that average annual concentrations of vinyl chloride in 1980 at Stations A, B, and MY were not likely zero. This addendum report assumes that average annual concentrations in 1980 at Stations A, B, and MY were 2 ppb. This may underestimate exposures, especially at Station MY.

Assumptions Regarding Ambient Vinyl Chloride Monitoring Data

Estimates of the individual excess lifetime cancer risk are based on the assumption that ambient air concentrations of vinyl chloride represent indoor air exposures (USEPA, 1989a).

The data used in the risk calculations are: SCAQMD's ambient vinyl chloride monitoring data from Stations A, B, and MY for the period June 1981 through March 1990; and the B.K.K. Corporation's Proposition 65 monitoring data from Stations "Amar-at Nogales," "Lynn Court," and "Nogales End" for the period September 1993-1994. The following assumptions are made regarding the data:

1981-1982 Data

- 1981-1982 average annual concentrations at Stations A and B are based on data collected for 7 of the 12 months in 1981, and 9 of the 12 months in 1982. The average concentrations for each set are assumed to be representative of a 12-month exposure (see TABLE 5);

- Station MY, located 150 feet southeast of the landfill property line, was established in June 1984. Values for the period 1980-1982 are assumed to be equal to the average annual concentrations at Station A, located 450 feet southeast of the landfill property line (see FIGURE 4). This may be an underestimation since Station MY was closer to the Class I Unit than Station A, and the levels and frequency of vinyl chloride detections were consistently higher at Station MY when both stations were in service;

- A value of 1 ppb is substituted for samples with nondetectable concentrations. (The detection limit between June 1981 through September 1982 was 10 ppb, and between October through December 1982, 2 ppb.)

1983-1990 Data

- The 1983 average annual concentration at Station MY is assumed to be 2.8 ppb, that is, equal to Station A's 1983 average annual concentration (see TABLE 6). This may be an underestimation. For the years 1985 through 1988, TABLE 6 shows that concentrations at Station MY were 1.6- to 1.9-fold higher than concentrations at Station A, and TABLE 7 shows that Station MY had between 25 to 33 percent fewer samples with nondetectable concentrations ;

- The 1984 average annual concentration at Station MY is assumed to be 5.4 ppb, that is, the average concentration detected by SCAQMD between August through

December at Station MY. The average concentration of the 5-month period is assumed to be representative of the 12-month exposure;

- The detection limit between January 1983 through December 1987 was 2 ppb; between January 1988 through September 1989, 1.4 ppb; and between October 1989 through March 1990, 2 ppb. A value of one-half the detection limit is substituted for samples with nondetectable concentrations.

1993-1994 Data

- It is assumed that the B.K.K. Corporation's Proposition 65 vinyl chloride monitoring data are acceptable for quantitative assessment; USEPA has stated that the analytical procedure appears to be acceptable to USEPA (USEPA, 1994e) and SCAQMD found the modified protocol acceptable (SCAQMD, 1994a). The detection limit varied from 0.005 to 0.010 ppb (see TABLE 8);

- It is assumed that the B.K.K. Corporation's Proposition 65 monitoring stations "Amar-at-Nogales" (residential station), "Lynn Court" (perimeter station), and "Nogales End" (perimeter station) represent SCAQMD's Stations A, B, and MY respectively. Because of the difference in locations, this assumption may lead to an overestimation for Stations B and MY, and may lead to an underestimation for Station A;

- Average annual concentrations for the 5-year period 1990-1994 are assumed to be equal to the average annual concentrations detected in the September 1993-1994 Proposition 65 monitoring.

1995-2009 Data

- Average annual concentrations for the period 1995-2009 are assumed to remain the same as the September 1993-1994 average concentrations. Changes in site activities, corrective actions, and technological advancements over the 15-year period may invalidate this assumption. Monitoring data collected by the B.K.K. Corporation in 1995 show decreasing vinyl chloride concentrations.

Individual Excess Lifetime Cancer Risk Estimates

The "individual excess lifetime cancer risk" is an estimate of the risk for an individual above the background risk of developing cancer during a 70-year lifetime.

In the United States, a new estimate of the normal background risk of developing cancer has been made since 1990 and it is greater than the value reported in the interim report from approximately 1 in 3 individuals (DHS-HWTS, 1990) to 4 in 10 individuals (Miller *et al.*, 1993); that is, approximately 40,000 in 100,000 individuals will develop cancer during their lifetime.

Exposure Scenario

The interim report estimated risk based on a "worst-case" exposure scenario. "Worst-case" represents a hypothetical individual and an extreme set of conditions, which will usually not be observed in an actual population. This addendum report estimates risks assuming a "reasonable maximum" exposure (RME) scenario and an "average" exposure scenario. USEPA defines the RME as "the maximum exposure that is reasonably expected to occur at a site," and is intended to be a conservative estimate, well above the average yet within the range of possible exposures (USEPA, 1989a).

Average concentrations are used in the risk equation. Use of average concentrations rather than the 95 percent upper confidence limit (UCL) value to estimate the RME is based on the following: 1) the monitoring stations were at the point of exposure; and 2) USEPA guidance states that "data sets with 20 to 30 samples provide fairly consistent estimates of the mean (i.e., the 95 percent UCL is close to the sample mean)....in general, the UCL approaches the true mean as more samples are included in the calculation (USEPA, 1992a)." As shown in TABLE 7, the minimum number of samples collected by SCAQMD in one year was 191. Thus, the 95 percent UCL is represented by the average annual concentrations.

USEPA cites national statistics on the number of years an individual resides at one location, and indicates an upperbound (90th percentile) of 30 years and an average (50th percentile) of 9 years. USEPA recommends a 30-year exposure duration when calculating the RME and a 9-year exposure duration when calculating the average exposure (USEPA, 1989a).

Risk Equation

The equation for the risk estimate is as follows:

$$ECR = Cvc \times YR \times EF \times UR \times 1/LT$$

where

ECR = Excess lifetime cancer risk
Cvc = Concentration of vinyl chloride in air (ppb)
YR = Years of residence associated with exposure
EF = Exposure factor, i.e., percent of time at home (80%)
UR = Unit risk value for vinyl chloride (20×10^{-5} per ppb)
LT = Human individual lifetime in years (70-years)

The interim report estimated an individual excess lifetime cancer risk of 1 to 2×10^{-5}, based on concentrations of vinyl chloride detected in SCAQMD ambient vinyl chloride monitoring data collected between 1982 through 1987, a worst-case exposure scenario (70 years residence), 80 percent time at home, and a unit risk value of 0.69×10^{-5} per ppb.

TABLE 9 is the estimates of individual upperbound excess lifetime cancer risks of this addendum report, assuming a reasonable maximum exposure scenario (30 years, 1980-2009). The estimates are based on concentrations of vinyl chloride detected at Stations A, B, and MY in SCAQMD data (June 1981 through March 1990) and the B.K.K. Corporation data (September 1993-1994), 80 percent time at home, and the unit risk value of 20×10^{-5} per ppb. Exposure values have been rounded to two significant figures, and risk estimates to one significant figure. The individual excess lifetime cancer risk is estimated per a specified period in order to delineate the risk per exposure year(s). Years with higher exposure concentrations are associated with a greater portion of the lifetime risk. The cumulative risk is an estimate of the past risk incorporated into the current and future risk estimate.

TABLE 9

INDIVIDUAL EXCESS LIFETIME CANCER RISK FROM EXPOSURE
TO AMBIENT FUGITIVE VINYL CHLORIDE EMISSIONS FROM THE CLASS I UNIT OF THE
BKK LANDFILL, WEST COVINA, CALIFORNIA
REASONABLE MAXIMUM EXPOSURE SCENARIO (30 YEARS)

STATION	EXPOSURE PERIOD	CONCENTRATION (ppb)	DURATION (yrs)	EXCESS RISK PER EXPOSURE PERIOD	CUMULATIVE EXCESS RISK
A	1980*	2.0	1	$5x10^{-6}$	$5x10^{-6}$
	1981	7.2	1	$2x10^{-5}$	$2x10^{-5}$
	1982	3.4	1	$8x10^{-6}$	$3x10^{-5}$
	1983	2.8	1	$6x10^{-6}$	$4x10^{-5}$
	1984	4.2	1	$1x10^{-5}$	$5x10^{-5}$
	1985	3.5	1	$8x10^{-6}$	$5x10^{-5}$
	1986	1.9	1	$4x10^{-6}$	$6x10^{-5}$
	1987	1.7	1	$4x10^{-6}$	$6x10^{-5}$
	1988	1.3	1	$3x10^{-6}$	$6x10^{-5}$
	1989	0.8	1	$2x10^{-6}$	$7x10^{-5}$
	1990-1994	0.05	5	$6x10^{-7}$	$7x10^{-5}$
	1995-2009	0.05	15	$2x10^{-6}$	$7x10^{-5}$
B	1980*	2.0	1	$5x10^{-6}$	$5x10^{-6}$
	1981	6.0	1	$1x10^{-5}$	$2x10^{-5}$
	1982	2.8	1	$6x10^{-6}$	$3x10^{-5}$
	1983	3.0	1	$7x10^{-6}$	$3x10^{-5}$
	1984	3.9	1	$9x10^{-6}$	$4x10^{-5}$
	1985	2.3	1	$5x10^{-6}$	$5x10^{-5}$
	1986	1.3	1	$3x10^{-6}$	$5x10^{-5}$
	1987	1.2	1	$3x10^{-6}$	$5x10^{-5}$
	1988	0.8	1	$2x10^{-6}$	$5x10^{-5}$
	1989	0.8	1	$2x10^{-6}$	$6x10^{-5}$
	1990-1994	0.03	5	$3x10^{-7}$	$6x10^{-5}$
	1995-2009	0.03	15	$1x10^{-6}$	$6x10^{-5}$
MY	1980*	2.0	1	$5x10^{-6}$	$5x10^{-6}$
	1981	7.2	1	$2x10^{-5}$	$2x10^{-5}$
	1982	3.4	1	$8x10^{-6}$	$3x10^{-5}$
	1983	2.8	1	$6x10^{-6}$	$4x10^{-5}$
	1984	5.4	1	$1x10^{-5}$	$5x10^{-5}$
	1985	5.4	1	$1x10^{-5}$	$6x10^{-5}$
	1986	3.0	1	$7x10^{-6}$	$7x10^{-5}$
	1987	2.6	1	$6x10^{-6}$	$7x10^{-5}$
	1988	2.5	1	$6x10^{-6}$	$8x10^{-5}$
	1989	0.9	1	$2x10^{-6}$	$8x10^{-5}$
	1990-1994	0.25	5	$3x10^{-6}$	$8x10^{-5}$
	1995-2009	0.25	15	$9x10^{-6}$	$9x10^{-5}$

Calculated by ECR = Cvc x YR x EF x UR x 1/LT where:

- ECR = Excess lifetime cancer risk
- Cvc = Concentration of vinyl chloride in air (ppb)
- YR = Years of residence associated with exposure
- EF = Exposure frequency, i e , percent of time at home (80%)
- UR = Unit risk value for vinyl chloride ($20x10^{-5}[ppb]^{-1}$)
- 1/LT = Human lifetime (70 years)

Exposure values have been rounded to two significant figures, and risk values, to one significant digit

*1980 concentration = 2 ppb, assumed; no data available.

1981-1982 concentrations = SCAQMD ambient vinyl chloride monitoring data Nondetected concentrations given the value of 1 ppb.

1983-1989 concentrations = SCAQMD ambient vinyl chloride monitoring data. Nondetected concentrations set at 1/2 the detection limit (detection limit 1983-1987, 2.0 ppb; detection limit 1988-9/89, 1.4 ppb; detection limit 10/89-3/90, 2.0 ppb)

1990-1994 concentrations = B K K Corporation's Proposition 65 Vinyl Chloride Monitoring Program, 9/93-9/94 Nondetected values set at 1/2 the detection limit. The detection limit varied from 0 005 to 0.010 ppb

Using the assumptions and methodology of this risk characterization, the estimated excess cancer risks are:

- at Station A, approximately 7×10^{-5};
- at Station B, approximately 6×10^{-5}; and
- at Station MY approximately 9×10^{-5}.

These excess cancer risks mean that for an individual living from 1980 to 2009 near Station A, for example, the theoretical upperbound risk of developing cancer is increased from 0.4 (the background cancer risk in the United States) to 0.40007 as a result of exposure to emissions of vinyl chloride from the closed Class I Unit of the B.K.K. Landfill.

Another way of expressing this excess cancer risk is: an individual living in the United States has a 40 percent (0.4) chance of developing cancer throughout a 70-year lifetime. For an individual living from 1980 to 2009 near Station A, there is an added 0.007 percent chance of developing cancer as a result of exposure to emissions of vinyl chloride from the closed Class I Unit of the B.K.K. Landfill.

Assuming an average exposure scenario (nine years) and the average annual concentrations for the years 1981-1989 (the years of highest detected average annual concentrations), the estimated excess cancer risks are:

- at Station A, 7×10^{-5};
- at Station B, 5×10^{-5}; and
- at Station MY, 8×10^{-5}.

The assumptions and methodology of this risk characterization suggest that:

- The year 1981 was the year of highest risk, based on available monitoring data;
- The majority of the excess lifetime cancer risk was incurred by 1983-1984;
- Current and future estimates of risk associated with the B.K.K. Landfill which do not incorporate past exposures at Stations A, B, and MY would not fully characterize the risks potentially incurred by individuals who lived near those stations since 1980 (30 year exposure scenario), or during the decade of the 1980s (9-year exposure scenario).

Exposed Population

In addition to the individual excess lifetime cancer risk, health risk assessments may estimate the population excess cancer burden. The population excess cancer burden is an estimate of the excess number of cases of cancer that may occur in a defined population who all have the same individual excess lifetime cancer risk. This addendum report does not estimate a population excess cancer burden because not all the population around the landfill were or are exposed to the concentrations of vinyl chloride detected at Stations A, B, and MY, and it is not possible to obtain a reliable estimate of exposure to other residents.

The number of potentially exposed individuals near Stations A, B, and MY since 1980 or during the decade of the 1980s is unknown, but considered to be too small for the probability that even one excess case of cancer will develop as a result of exposure to emissions of vinyl chloride from the closed Class I Unit of the B.K.K. Landfill. As an example, there would need to be

approximately 14,000 exposed individuals living near Station A between 1980 and 2009 for one excess case of cancer to develop, based on the individual excess lifetime cancer risk as estimated in this addendum report. Because the number of individuals near Station A is much smaller than 14,000, it is unlikely that excess cases of cancer will occur. However, each exposed individual may have an increased risk of developing cancer.

Recently, DHS-Cancer Surveillance Section (DHS-CCS) reviewed the 1988-93 tumor incidence data for a one mile area around the landfill. DHS's review found no indication of increased cancer incidence (DHS-CSS, 1995; see Appendix H).

Consequently, there is a need for a different way of evaluating the effect of the exposures at Stations A, B, and MY. Stratification of risks by year of exposure, as done in this addendum report (see TABLE 9), accomplishes this by highlighting the different risks associated with exposures in and over time, namely:

- Risks for individuals who will have lived near Station A, B, or MY from 1980 to 2009 (6 to 9 x 10^{-5});
- Risks for individuals who lived near Station A, B, or MY only during the decade of the 1980s (6 to 8 x 10^{-5});
- Risks for individuals near Station A, B, or MY between 1990 and 1994 (3 x 10^{-6} to 3 x 10^{-7}); and
- Risks for individuals near Stations A, B, or MY between 1995 and 2009 (1 to 9 x 10^{-6}).

These different risks would not be evident in risk assessments which address only current and future risks. The stratification of risks by year of exposure is consistent with USEPA's position that past exposures may be important for selected sites (USEPA, 1989b).

Estimated Excess Lifetime Cancer Risk Using Different Assumptions and Methodology

TABLE 10 shows the excess cancer risk at Station A, as an example, using different assumptions for time spent at home, the average annual concentration of vinyl chloride in 1980, the substituted value for samples with nondetectable concentrations in the 1981-1982 data, the exposure duration, and the unit risk value.

Time Spent At Home

The interim report and this addendum report assumed individuals spend 80 percent of their time at home. USEPA assumes individuals spend 350 days per year at home, or 96 percent time at home (USEPA, **1991b**). Using USEPA's percent time spent at home in the risk equation suggests slightly higher estimates of excess cancer risk: at Station A, for example, 8 x 10^{-5} . There are also California-specific values. ARB funded a statewide survey of activity patterns for the purpose of obtaining representative data for modeling population exposures and designing appropriate monitoring strategies. Activity patterns of residents over 11 years of age were surveyed during 1987-1988 (ARB, 1991; Jenkins *et al.*, 1992), and children 11 years and younger, during 1989-1990 (Phillips *et al.*, 1991). Average data rather than frequency distributions were presented. Results showed that, on the average, Californians spend 87 percent of their time indoors, 7 percent in enclosed transit, and 6 percent outdoors. Of the 87 percent time indoors, 62 percent is spent as indoors at home for individuals over 11 years of age, and 76 percent, as indoors at home for children 11 years and younger. Using ARB's percent time spent at home suggests slightly lower estimates of excess cancer risk: at Station A, 5 x 10^{-5} using 62 percent and 6 x 10^{-5} using 76 percent. These different assumptions

TABLE 10

ESTIMATED EXCESS CANCER RISK AT STATION A USING DIFFERENT ASSUMPTIONS

Station A

	Time Spent at Home	Average Annual Concentration 1980	Substituted Value for Samples with Non-Detectable Concentrations 1981-1982 Data	Exposure Duration (years)	Vinyl Chloride Unit Risk	Cumulative Excess Cancer Risk*
Different Time[a] Assumptions	62%	2 ppb	1 ppb	30	20×10^{-5}	5×10^{-5}
	76%	2 ppb	1 ppb	30	20×10^{-5}	6×10^{-5}
	80%	2 ppb	1 ppb	30	20×10^{-5}	7×10^{-5}
	96%	2 ppb	1 ppb	30	20×10^{-5}	8×10^{-5}
Different Substituted[b] Value Assumptions	80%	0 ppb	0 ppb	30	20×10^{-5}	6×10^{-5}
	80%	5 ppb	5 ppb	30	20×10^{-5}	8×10^{-5}
Different Duration[c] Assumptions	80%	2 ppb	1 ppb	70	20×10^{-5}	7×10^{-5}
Different Unit Risk[d] Assumptions	80%	2 ppb	1 ppb	30	2.5×10^{-5}	0.9×10^{-5}
	80%	2 ppb	1 ppb	30	4.5×10^{-5}	1×10^{-5}
Assumptions Used in Addendum Report	**80%**	**2 ppb**	**1 ppb**	**30**	**20×10^{-5}**	**7×10^{-5}**

* Note - Cumulative Excess Cancer Risk after replacement of shaded assumptions in column 1, 2 or 3, but retaining all other assumptions for 1983 to 2009 average annual concentrations as shown in TABLE 9.

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[a] 62% = for individuals over 11 years of age, taken from the Air Resources Board, "Activity Patterns of California Residents," Final Report, 1991, Research Division, Sacramento California;

76% = for children 11 years and younger, taken from Phillips, T.J., Jenkins, P.L., and Mulberg, E.J., "Children in California: Activity Patterns and Presence of Pollutant Sources," 1991, Air Resources Board, Sacramento, California;

80% = Office of Environmental Health Hazard Assessment (OEHHA) value; also assumed in "Health Risk Assessment of the B.K.K. Landfill, West Covina, California, Interim Report November 1990," Department of Health Services-Hazardous Waste Toxicology Section, Emeryville, California;

96% = United States Environmental Protection Agency value, taken from "Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual, Supplemental Guidance, 'Standard Default Exposure Factors' Interim Final 1991," OSWER Directive: 9285. 6-03. Office of Emergency and Remedial Response, Toxics Integration Branch, Washington, D.C.

[b] 0 ppb = lowest possible value
1 ppb = OEHHA's assumed value for samples with nondetectable concentrations of vinyl chloride in the 1981-1982 data
2 ppb = OEHHA's assumed average annual concentration of vinyl chloride at Station A in 1980, as described in the addendum report
5 ppb = value based on half the detection limit for vinyl chloride in 1980

[c] worst case duration = for comparison with the 1990 interim report

[d] 2.5×10^{-5} per ppb = value derived by OEHHA from a study of vinyl chloride workers who developed liver cancer
4.5×10^{-5} per ppb = value derived by OEHHA from combined studies of vinyl chloride workers who developed liver, brain, or lung cancers
20×10^{-5} per ppb = current unit risk value derived by OEHHA from a study in mice that developed lung cancer

regarding percent time spent at home suggest stimates of excess cancer risk which, at Station A, are approximately 29 percent below or 14 percent above the estimate obtained using the primary assumption in this addendum report for percent time spent at home (see TABLE 10).

Average Annual Concentration in 1980 and Substituted Value for Samples With Nondetectable Concentrations in the 1981-1982 Data

There is uncertainty associated with the average annual concentration assumed for exposures in 1980, and with the substituted value for samples with nondetectable concentrations. Nondetected concentrations could range from zero to just below the detection limit. The "true" level cannot be known. The reasonable maximum exposure scenario as estimated in TABLE 9 assumes an average annual concentration in 1980 of 2 ppb, and the substituted value of 1 ppb for samples with nondetectable concentrations in the 1981-1982 data. If a value of 5 ppb (half the detection limit achievable in 1980-1982) were substituted for the average annual concentration in 1980 and for samples with nondetectable concentrations in the 1981-1982 data, estimates of excess cancer risks are increased: at Station A, 8×10^{-5}. If a value of zero were substituted for the average annual concentration in 1980 and for samples with nondetectable concentrations in the 1981-1982 data, estimates of excess cancer risks are decreased: at Station A, 6×10^{-5}. These different assumptions regarding the average annual concentration in 1980 and the substituted value in the 1981-1982 data give estimates of excess cancer risk approximately 14 percent above or below estimates obtained using the primary assumption for the average annual concentration in 1980 and for samples with nondetectable concentrations in the 1981-1982 data (see TABLE 10).

Exposure Duration

Under a 70-year exposure duration, the average annual concentration at Station A in 1995 is assumed to be constant for the 55-year period, 1995 to 2050. Because the exposure in 1995 is low, there is no change in the risk estimate at Station A assuming either a 30-year or a 70-year duration (see TABLE 10). This estimate differs from that of the 70-year duration risk estimate of th interim report because of the following differences: different assumed year for the beginning of exposures; different exposure concentrations for the years 1980-1982 and the years after 1987; and different unit risk values for vinyl chloride (see TABLE 11). The current 70-year duration estimate is based on updated data and toxicity information.

Unit Risk Values

The Cal/EPA unit risk value of 20×10^{-5} per ppb is an upperbound estimate of the carcinogenic activity of vinyl chloride in humans, derived from studies in female mice that developed lung carcinoma. TABLE 2 also shows two unit risk values based only on human studies. The unit risk values derived from human data have the advantage that they do not have to incorporate an adjustment for interspecies variation. However, they have limitations which include use of studies of only male workers and use of estimates of exposure based on job descriptions rather than actual exposure monitoring data. The unit risk value 2.5×10^{-5} per ppb is derived from studies of vinyl chloride workers who developed angiosarcoma of the liver; the unit risk value derived from combined studies of vinyl chloride workers who developed liver, lung, or brain cancers is 4.5×10^{-5} per ppb. Assuming the unit risk value 2.5×10^{-5} per ppb, the excess cancer risks (calculated for the period 1980 to 2009, using the average annual concentrations shown in TABLE 9) decrease: at Station A, 0.9×10^{-5}. Assuming the unit risk value 4.5×10^{-5} per ppb, the excess cancer risk at Station A is 1×10^{-5}. These different assumptions regarding the unit risk value suggest estimates of excess cancer risk approximately 87 percent lower than estimates assuming the current Cal/EPA unit risk value (see TABLE 10).

Sensitivity From Exposures at a Young Age

The methodology suggested by USEPA to estimate risk to vinyl chloride from exposures at a young (0 to 5) age assumes that, for a given constant exposure level, an individual exposed during the first six years of life incurs the full conventional (70-year) lifetime risk, and that an individual exposed from birth for 70 years incurs double the conventional lifetime risk. This increased sensitivity to exposures at a young age does not mean that a child will develop cancer but that when the exposed child becomes an adult, her or his risk of developing cancer is increased. Although referred to in USEPA's guidance documents, the methodology for assessing risk from exposures to vinyl chloride at a young age is still pending final adoption by USEPA. Further discussion is provided in the "Limitations and Uncertainties" section

Summary of Health Risk Assessments Conducted To-Date Regarding the B.K.K. Landfill

TABLE 11 summarizes results of the health risk assessments conducted to-date regarding the B.K.K. Landfill. Different assumptions, methodologies, and data bases were used. Estimated risks associated with exposures to vinyl chloride range from 0.04×10^{-5} to 9×10^{-5}.

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TABLE 11

SUMMARY OF HEALTH RISK ASSESSMENTS OF THE BKK LANDFILL, WEST COVINA, CALIFORNIA

Report	Assumptions	Methods	Excess Cancer Risk
1 "Ambient Air Monitoring and Health Risk Assessment for Suspect Human Carcinogens Around the BKK Landfill in West Covina" Department of Health Services(DHS)/Air Resources Board(ARB)/South Coast Air Quality Management District(SCAQMD), 1983	• No dilution in measured concentrations at stations A, B, C, D, E, F • Exposure for 24 hrs/day, 365 days/year, for the 7 years (1976-1982) • 100% absorption in the lungs • Exposure to 7 chemicals: vinyl chloride, benzene, trichloroethylene, perchloroethylene, 1,1-dichloroethylene, 1,2-dichloroethane, and chloroform • Unit risk for vinyl chloride 1.3 x 10^{-5} per ppb	Individual excess lifetime cancer risk and population excess cancer burden	5 x 10^{-5} (all 7 chemicals)
2. "Estimates of Carcinogenic Risk in the Vicinity of the BKK Landfill" K.S. Crump & Co., 1986	• Average air concentrations from 12 SCAQMD stations and 4 University of Southern California stations • Exposure for 24 hrs/day, 365 days/year, for the 8 year period 1978-1985 • Exposure to 7 chemicals (see above list) • Unit risk value for vinyl chloride: 1 x 10^{-5} per ppb	Maximum likelihood estimate (MLE) and upper 95% confidence limit estimate (UCL)	0.25 x 10^{-5} (MLE) 1.1 x 10^{-5} (UCL) (all 7 chemicals) 0.1 - 0.2 x 10^{-5} (vinyl chloride)
3. "Risk Assessment for Exposure to Ambient Air in the Vicinity of the BKK Landfill in West Covina, 1978-1985" Bogen and Smith, 1986	• 50% dilution in measured concentrations at stations A, B, D, F • Exposure for 15 hrs/day, 365 days/year, for the 7 years (1978-1985) • 50% absorption in the lungs • Exposure to 7 chemicals (see above list) • Unit risk value for vinyl chloride: 1 x 10^{-5} per ppb	Monte Carlo potency analysis method and Monte Carlo procedure, best estimate	1.7 x 10^{-5} (all 7 chemicals) 0.04 x 10^{-5} (vinyl chloride)
4 "Health Risk Assessment of the BKK Landfill, West Covina, California, Interim Report" DHS-Hazardous Waste Toxicology Section (DHS-HWTS), 1990	• No dilution in monitored concentrations at stations A, B, and MY; 1982-1987 SCAQMD data • Exposure for 24 hrs/day, 292 days/year for 70 years (1976-2046) • 100% absorption in the lungs • Exposure to vinyl chloride primarily • Unit risk value for vinyl chloride: 0.69 x 10^{-5} per ppb	Worst case scenario; individual excess lifetime cancer risk and population excess cancer burden	1 - 2 x 10^{-5}
5. "Risk Assessment in Fulfillment of AB2588 Requirements for SCAQMD Facility No. 55449" ENVIRON, 1991	-- Under review by SCAQMD --	--	--
6. "Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions from the Class I Unit of the BKK Landfill, West Covina, California" Office of Environmental Health Hazard Assessment-HWTS, 1996	• No dilution in monitored vinyl chloride concentrations at stations A, B, and MY; data sets: 1981-1982 SCAQMD, 1983-1990, SCAQMD; 1993-1994, BKK Corporation • Exposure for 24 hrs/day, 292 days/year, for 30 years (1980 to 2009) • Exposure to vinyl chloride primarily • Unit risk for vinyl chloride: 20 x 10^{-5} per ppb	Reasonable maximum exposure scenario; individual excess lifetime cancer risk	Station A: 7 x 10^{-5} Station B: 6 x 10^{-5} Station MY: 9 x 10^{-5}

Limitations and Uncertainties

The estimated risks calculated in this report are upperbound estimates based on the assumptions, methodology, limitations, and uncertainties specified. Other methods (maximum likelihood estimates, best estimates, Monte Carlo estimates) may yield different risk estimates. Most of the assumptions used in this report tend to overestimate the risk. Normally, the actual risks from a site would be no more than, and likely less than (if not zero), the estimates given in this type of assessment. Limitations and uncertainties associated with the data and methodology of this addendum report are the following:

Hazard Identification

- Other hazardous chemicals known to be emitted from the B.K.K. Landfill include benzene (also a known human carcinogen), trichloroethylene, perchloroethylene, chloroform, and 1,2-dichloroethane. These chemicals were not quantitated to the same extent as vinyl chloride, and have also been detected in background air in the South Coast air basin. It is not possible with the available data to distinguish between the amount of those chemicals emitted from the Class I Unit of the B.K.K. Landfill and the amount found in ambient air from other sources.

Toxicity Assessment

- The unit risk value for vinyl chloride (20×10^{-5} per ppb) was estimated using the linear multistage model. This model extrapolates the incidence of the carcinogenic effect observed at high exposure levels, in this case, in the ppm range, to what may be expected at much lower exposure levels, in this case, more than 1,000 times lower (ppb). The unit risk value is the upperbound 95 percent confidence limit of the maximum likelihood estimate. This model may underestimate the unit risk value but is more likely to overestimate it. There is the possibility that the unit risk value is zero at low ppb levels;

- The unit risk value is based on an animal study. Other values calculated from data obtained in occupational studies were lower. However, the occupational studies were only of male workers and the exposure levels were not actually measured;

- There is evidence that very young animals are more sensitive to the carcinogenic effects of vinyl chloride. USEPA recommends age-attributable risk estimations for exposures to vinyl chloride. The unit risk value developed by OEHHA accounts for age specific sensitivity.

Exposure Assessment

- There are no ambient vinyl chloride monitoring data for the year 1980. It is an assumption of this addendum report that exposures at Stations A, B and MY began in 1980. Residential development near the monitoring stations was completed in 1979, and odor complaints data from 1978 to 1981 suggest that residents on the "M" and "L" named streets were subject to downwind exposures before the residential monitoring program began;

- The 1981-1982 ambient vinyl chloride monitoring data were collected for 16 of the 24 months, and the average concentrations of the available data for each year were assumed to be representative of a 12-month period. This may overestimate exposure;

- Results of the data collected between October 16 and December 31, 1982 are available in terms of the ambient air quality standard (10 ppb) rather than the detection limit (2 ppb). Consequently, the number of samples with nondetectable concentrations in this portion of the data are likely over-reported. A value of 1 ppb was substituted for samples with nondetectable concentrations in the 1981-1982 data; this may underestimate exposure;

- During the period 1983-1990, ambient air monitoring was conducted only in residential areas to the southeast and south of the landfill. These data may have identified the highest detectable ambient levels of vinyl chloride but they do not provide information for other residential areas around the landfill;

- Monitoring at Station MY began in June 1984. Average annual concentrations at Station A for the years 1981 through 1983 are used to represent exposures at Station MY for those three years. This may underestimate exposures at Station MY because it is closer to the landfill than Station A and higher levels were consistently reported for Station MY when both stations were in service;

- The majority of samples in SCAQMD's 1981-1990 ambient vinyl chloride monitoring data were samples with nondetectable concentrations (21 of 26 data sets, see TABLE 7). The substitution of 1 ppb for samples with nondetectable concentrations is likely to underestimate exposures in the 1981-1982 data since some of the samples may have had concentrations of vinyl chloride below 10 ppb but above 2 ppb. The substitution of 1 ppb for samples with nondetectable concentrations in the 1983-1990 data is likely to overestimate exposures;

- The analytical procedure for B.K.K. Corporation's 1993-1994 Proposition 65 monitoring data has been accepted by SCAQMD and appears to be acceptable to USEPA.

Risk Characterization

- Using a substituted value greater than zero for samples with nondetectable concentrations yields a risk estimate that will always be above zero, whether there were actual exposures or not;

- The assumption that 2 ppb is a reasonable estimate for an average annual concentration in 1980 may underestimate exposure because of the average annual concentrations of vinyl chloride at Stations A and B in 1983, and the estimated average annual concentrations of vinyl chloride at Stations A and B in 1981-1982;

- Actual indoor air levels of vinyl chloride are not known. Indoor air monitoring was conducted only in October 1984 and only in ten homes along the southern border of the landfill. The assumption that ambient air and indoor air concentrations are equal may either underestimate or overestimate exposure;

- The length of residency and percent time at home are general assumptions which tend to overestimate exposures. They are not based on actual information from residents living near the monitoring stations.

- 1995 monitoring data collected by the B.K.K. Corporation shows an average annual concentration of 0.12 ppbv onsite at the Nogales End station. This suggests that vinyl chloride concentrations are decreasing, and the associated risk is also decreasing.

- There is uncertainty associated with the vinyl chloride unit risk value used in the addendum report. Using USEPA's apportioned lifetime risk estimates to account for increased sensitivity from exposures at a young age (TABLE 3) suggests a 14-fold higher risk at Station A than the estimate obtained in this addendum report. Using a physiologically-based pharmacokinetic model for vinyl chloride (Appendix J, comments from the Chemical Manufacturers Association, article by Reitz *et al.*, 1995) suggests a 140-fold lower risk at Station A then the estimate obtained in this addendum report.

  Risk estimation is a constantly evolving science and changes as new information and data are accepted by risk assessors. The USEPA apportioned lifetime risk estimate and the physiologically-based pharmacokinetic model for vinyl chloride are proposed newer ways to look at risk from vinyl chloride. This addendum report used a currently established and accepted estimate of risk for vinyl chloride.

Findings of the Addendum Health Risk Assessment

This addendum health risk assessment provides an assessment of past, present, and future risks for individuals living near Stations A, B, and MY from exposures to ambient fugitive emissions of vinyl chloride associated with the closed Class I Unit of the B.K.K Landfill. There are four findings of this report:

1. From the available monitoring data, the single year of highest detected exposure to vinyl chloride was 1981. The maximum detected concentration of vinyl chloride at Station A in 1981 was 50 ppb, compared to 0.16 ppb in 1994. The average annual concentration at Station A in 1981 is estimated to have been 7.2 ppb, compared to an estimate of 0.05 ppb in 1994. For individuals who lived near Stations A, B, and MY during the 1980's, past exposures to vinyl chloride were higher than current exposures. The cumulative individual excess lifetime cancer risk suggests that for exposures beginning in 1980, the majority of the cancer risk was incurred by 1983-1984, and that there is little change (on the order of 15 to 22 percent) in the cumulative risk after 1986.

2. The landfill gas collection system, which was expanded over time, and the cap covering the closed Class I Unit have been successful in lowering the frequency and level of vinyl chloride emissions to levels that pose little, if any, risk to nearby residents.

3. Regulatory agencies with responsibility for remediation or control of the B.K.K. Landfill may want to include past exposures of vinyl chloride near Stations A, B, and MY in subsequent health assessments of emissions from the landfill to ensure future risks do not add significantly to the past cumulative risk.

4. In spite of using best available scientific knowledge, there is considerable uncertainty associated with the estimated exposures, the carcinogenic potency value of vinyl chloride, and young-age sensitivity methodology. The estimated excess cancer risks calculated in this addendum report are upperbound estimates, based on the assumptions, methodology, limitations and uncertainties specified. The actual excess cancer risk is likely to be lower than these estimates and may be zero. Because the number of people who actually lived or are living near Stations A, B, and MY is small, it is unlikely that excess cases of cancer will occur. Each exposed individual may have an increased risk of developing cancer, however the added risk is small.

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APPENDIX A

ERRATA

ERRATA

The following errors were contained in the interim report, "Health Risk Assessment of the B.K.K. Landfill, West Covina, California, November 1990," prepared by the Hazardous Waste Toxicology Section, Environmental Epidemiology and Toxicology Branch (EETB), Health Hazard Assessment Division, California Department of Health Services (DHS):

- Page 8, third paragraph: *The B.K.K. Landfill, operated by B.K.K. (Kenneth B. Kazanan) Corporation, is located within the corporate boundary of the City of West Covina, California, 18 miles east of downtown Los Angeles in Los Angeles County (Figure 1)."*

The reference to Kenneth B. Kazanan as the BKK Corporation is not correct. The text should read: *The B.K.K. Landfill, operated by the B.K.K. Corporation, is located within the corporate boundary of the City of West Covina, 18 miles east of downtown Los Angeles in Los Angeles County (Figure 1).*

- Page 80, third paragraph: *In 1985, EPA estimated the population within a one-mile radius of the center of the B.K.K. Landfill to be approximately 40,000 persons (LPA, 1985).*

The reference *(LPA, 1985)* should read *(EPA 1985)*.

CMA 114394

APPENDIX B

"B.K.K. LANDFILL, WEST COVINA, CALIFORNIA FACT SHEET, DECEMBER 1990,"
CALIFORNIA DEPARTMENT OF HEALTH SERVICES
ENVIRONMENTAL EPIDEMIOLOGY AND TOXICOLOGY BRANCH
EMERYVILLE, CALIFORNIA

California Department of Health Services
Environmental Epidemiology
and Toxicology Branch

BKK LANDFILL
West Covina, California

FACT SHEET

DECEMBER 1990

In-Depth Summaries Of Health Studies Near BKK Landfill

This fact sheet contains summaries of two cancer studies and a health risk assessment concerning the BKK landfill, a hazardous waste site in West Covina, California. These studies were undertaken by the California Department of Health Services (DHS) in response to the concerns of residents near the landfill.

Background

In 1963, the BKK landfill, operated by the Ben Kenneth Kazarian Corporation, opened on 130 acres in the San Jose Hills in the City of West Covina in Los Angeles County. Initially, the landfill accepted only municipal waste such as household and commercial refuse and construction debris. In 1971, the City of West Covina approved an expansion of the facility to its present size of 583 acres. In 1972, a 40- acre hazardous waste management unit (a state-designated Class I landfill) opened on the existing site and began receiving hazardous wastes. This unit was expanded by an additional 100 acres in 1975. In November, 1984, the landfill stopped accepting hazardous waste. In March, 1989, the unit was formally closed. The closing process included building a low-permeability soil cap over 180 acres, that is, over the regulated hazardous waste area and some surrounding municipal waste.

In 1969, area residents filed their first odor complaint against the BKK landfill with the South Coast Air Quality Management District. In 1979, 278 odor complaints were reported. Studies have shown that hazardous waste migrated from the landfill into air and water around the site. People living near the landfill have been concerned about possible health effects due to hazardous waste releases in air, surface water runoff, spills and dust from the landfill. Drinking water supplies are not contaminated.

1. The Cancer Studies

What Were The Studies Looking For?

Under contract by the California Department of Health Services (DHS), Dr. Thomas Mack, Dr. Duncan Thomas and Richard Pinder from the University of Southern California, Cancer Surveillance Program, conducted two studies that investigated the distribution of cancer cases in residents living near the BKK landfill between 1972 and 1982.

The aim of the first study, completed in 1985, was to determine whether residents living south and west (mostly downslope) of the BKK landfill had a greater chance of getting cancer than the average resident of Los Angeles County. The second study, completed in 1988, focused on the area to the north and east (mostly downwind during the day) of the landfill. Both studies relied on data collected by the Los Angeles County Cancer Surveillance Program, and examined cancer incidence from 1972 to 1982. Each study analyzed cancer rates for all childhood cancers, for each of 89 different types of cancers, and for all cancers taken together. In both studies, the distribution of those cancers found to have occurred at a higher than expected rate was examined to determine if the landfill might be a possible cause of the increase.

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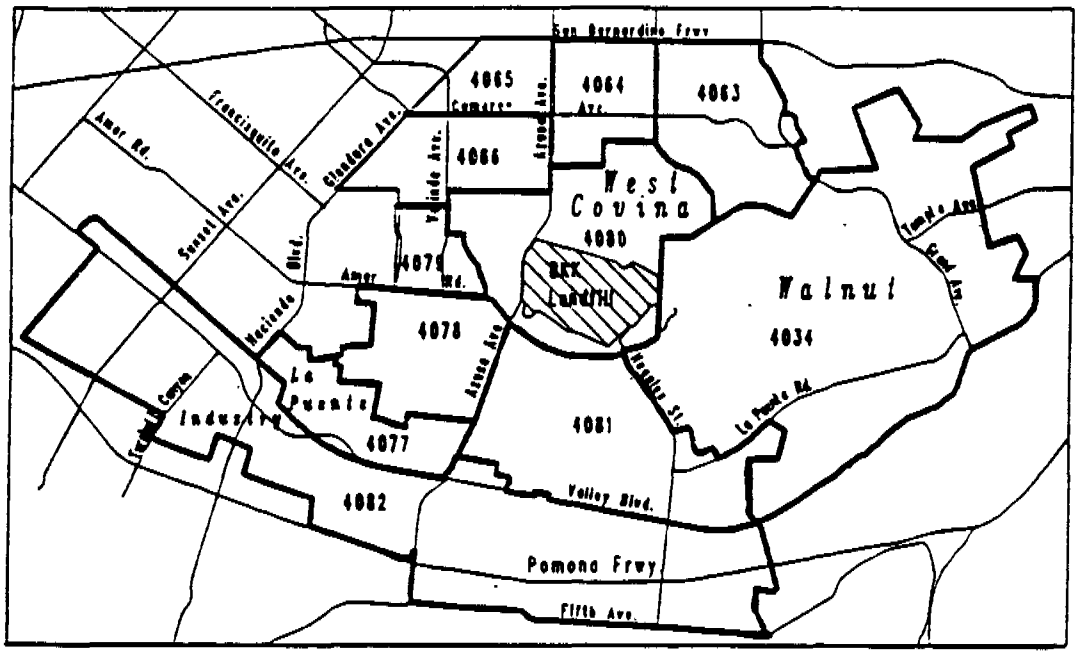
CENSUS TRACTS IN STUDY AREA
BKK LANDFILL, WEST COVINA, CA



California Department
Of Health Services

1 inch = 7,500 feet

4089 Census Tract
Highway

The Cancer Surveillance Program collects information on each Los Angeles County resident who has received a microscopically verified cancer diagnosis or for whom cancer is recorded on a death certificate. These two studies focused on persons who were diagnosed with cancer during the 11-year period between January 1, 1972, and December 31, 1982. Residents of 11 census tracts (1960 definitions) comprised the population living near the BKK landfill (see map above). In total, about 100,000 people lived in the BKK study area in 1977 (midway through the 1972-1982 study period), and about 2,000 cancer cases were identified during the 11-year study period. Table 1 shows for each tract the estimated 1977 population (based on 1970 and 1980 census data) and the number of residents diagnosed with cancer between 1972 and 1982.

What Did The Studies Find?

Among residents of the 11 census tracts, the overall rate of cancer for both adults and children was similar to that for Los Angeles County as a whole. The rate of cancer was not higher for residents living closer to the landfill, nor did the average rate of cancer increase between the earlier and later study time periods (from 1972-1976 to 1978-1982). Overall sex-specific cancer rates are shown in Table 2 for each census tract, along with a comparison to total Los Angeles County rates.

Even though the overall cancer rate in the BKK study area was not higher than the Los Angeles county rate, researchers studied 89 individual types of cancer to determine whether unexpectedly high levels of any one of them appeared in any census tract. A type of cancer was considered in excess if: 1) there were more extra cases than could be accounted for by chance alone, 2) cases occurred at least twice as often as usual in Los Angeles County, and 3) that type of cancer met at least one of four other requirements (more than four excess cases; elevated in at least two census tracts; elevated in both sexes; or the rate of that cancer at least doubled over the study

(continued on Page 4)

Table 1

Total number of cancer cases in 1972-1982 and estimated 1977 population in 11 census tracts around the BKK landfill, West Covina, California.

Census Tract	Direction of Census Tract from BKK Landfill	Est. 1977 Population	Total 1972-82 Cancer Cases
	Tract in Both Study One and Two		
4080	Contains BKK landfill	8,565	142
	Other Tracts in Study One		
4077	Southwest	8,652	209
4078	Southwest	7,370	159
4079	West	4,577	78
4081	South	27,280	380
4082	South and West	9,807	218
	Other Tracts in Study Two		
4034	East	10,533	166
4063	North and East	3,470	111
4064	North	6,720	236
4065	North	5,626	199
4066	Northwest	8,955	277
Total Tract Aggregate		101,555	2,175

Table 2

1972-1982 average annual cancer incidence rates by sex in Los Angeles County and in 11 census tracts around the BKK landfill, West Covina, California.

Census Tract	Sex	1972-1982 Cancer Cases	Average Incidence Rate[1]	Rate Relative To LA CO Rate[2]
Los Angeles County	Male	127,003	375.6	–
	Female	148,563	328.5	–
4080	Male	83	368.1	1.0
	Female	59	187.9	0.6
4077	Male	87	364.7	1.0
	Female	122	362.7	1.1
4078	Male	76	466.1	1.2
	Female	83	364.3	1.1
4079	Male	35	297.5	0.8
	Female	43	263.5	0.8
4081	Male	176	290.7	0.8
	Female	204	245.7	0.7
4082	Male	95	262.5	0.7
	Female	123	255.9	0.8
4034	Male	77	343.7	0.9
	Female	89	284.2	0.9
4063	Male	56	354.2	0.9
	Female	55	290.4	0.9
4064	Male	101	367.7	1.0
	Female	135	384.7	1.2
4065	Male	99	477.8	1.3
	Female	100	327.8	1.0
4066	Male	118	336.9	0.9
	Female	159	345.9	1.1

[1] Average number of cancer cases per 100,000 population per year.

[2] A value of 1.0 means that the cancer rate in the census tract is equal to the rate in L.A. County. A value greater than 1.0 (e.g., 1.2) means that the rate in census tract is greater than rate in L.A. County (e.g., by 1.2 times). A value less than 1.0 (e.g., 0.8) means the rate in the census tract is less than the rate in L.A. County (e.g., by 0.8 times).

period). Ten such types of cancer were identified: hepatoma, meningioma, tongue, nasopharynx, larynx, upper colon, pancreas, bladder, melanoma of trunk or scalp, and endometrium (Table 3).

Whenever small geographic areas are closely studied as was done here, too many — and too few — cases of a few types of cancer are expected to appear in some areas by chance alone. Each cancer type listed in Table 3 was scrutinized to determine if any other evidence of a public health risk existed, for example, whether any cancer showed a consistent pattern of increased risk as might be expected from exposure to the landfill. Researchers concluded that excess cases of each of these 10 types of cancer were most likely not due to the landfill, but to other characteristics of the affected population. First, some types of cancer are known to be related to high income and education. One is endometrial cancer. Risk of endometrial cancer is greater among high-income populations who use estrogen replacement hormones and delay childbearing. When the rates of this cancer in the BKK study area were compared with rates in census tracts of similar socioeconomic status in Los Angeles County, the rates were nearly the same.

Second, the increase in some identified cancers disappeared after 1982. In the case of melanoma, its relatively higher rate in one census tract between 1972 and 1982 has not continued, and no additional cases appeared between 1983 and 1985. This drop in the rate led the authors to conclude that the 1972-82 rate was not due to the site.

Third, and more significantly, no excess cancer cases were identified in the census tract containing the landfill (4080), which covered an area extending approximately one mile from the site. Residents with these 10 types of cancer were more likely to live farther away from the landfill than residents with other types of cancer. If exposures from the site were causing the cancers, there would be a pattern of more cases near the site, not farther from it.

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Table 3

Selected characteristics of 10 specific types of cancers identified for in-depth analysis in two cancer studies of 11 census tracts around the BKK landfill, West Covina, California.

Type of Cancer	Sex	Census Tract	Total Cancer Cases	Annual Rate Relative To LA CO Rate[1]	Number Excess Cases	Rate in 1978-82/1972-76[2]
Hepatoma	F	4082	3	4.9	2.4	2.0
Meningioma	M	4079	1	3.7	0.7	0.0
	M	4081	5	3.5	3.6	0.7
	M	4082	2	2.7	1.3	1.0
Tongue	F	4078	3	10.1	2.7	0.5
	F	4082	3	4.6	2.3	0.5
Nasopharynx	F	4077	2	13.8	1.9	Inf[3]
	M	4078	2	9.1	1.8	Inf[3]
Larynx	M	4082	6	2.0	3.0	1.0
Upper Colon	M	4077	7	3.0	4.7	1.3
	F	4082	9	1.8	4.1	2.0
Pancreas	F	4077	6	2.8	3.8	0.5
Bladder	M	4078	9	2.4	5.3	1.3
Melanoma, Trunk/Scalp	M	4034	6	2.8	3.9	Inf[3]
Endometrium	F	4064	23	2.0	11.7	0.8
	F	4066	24	1.7	9.4	0.8

[1] Value greater than 1.0 (e.g., 1.7) means rate in census tract is greater than rate in L.A. County (e.g., by 1.7 times).

[2] This column is the rate of cancer occurrence in 1978-82 divided by the rate during 1972-76. For hepatoma, for example, the rate in 1978-82 was 2 times the rate in 1972-76.

[3] Inf means infinity, and indicates that there were no cases diagnosed in 1972-1976.

What May The Studies Have Missed?

Although a thorough study has been conducted, all studies have limitations. If there was a relationship between the BKK landfill and cancer, there are a few reasons why the study may have failed to find it. First, too small a proportion of the population in the nearby census tracts may actually have been exposed to the landfill. If, for example, only people on the blocks close to the landfill had exposures from the site, then analyzing cancer rates for the whole census tract may have been too crude. Census tracts, however, were the smallest unit from which the Cancer Surveillance Program could analyze its records. Second, because people moved in and out of the community often, exposed individuals who developed cancer may well have left the area by the time the disease appeared and so were not in the Cancer Surveillance Program's data base. A third reason may be that not enough time has elapsed for cancer to occur: the consensus among scientists is that there is often a 20- to 30-year period between exposure to a cancer-causing chemical and the onset of disease.

Conclusion

These studies compared the rate of cancer occurrence in residents of 11 census tracts adjacent to the BKK landfill to rates in Los Angeles County as a whole. There was no overall increase in the cancer rate among residents living near the BKK landfill. There was no consistent pattern of occurrence to suggest that the BKK landfill is linked to individual types of cancer. The results do not substantiate concerns that there were excess cancers around the BKK landfill between 1972 and 1982.

2. The Health Risk Assessment

Introduction

In response to residents' requests, DHS conducted a health risk assessment of the BKK landfill based on environmental monitoring from 1982 through 1987. It provides an estimate of the health risks to individuals living near the BKK landfill from exposure to chemicals found in the air, groundwater and surface water. The assessment is primarily concerned with estimating the risk of developing cancer from exposure to chemicals found in the air because: 1) monitoring activities indicated that air was the main route of exposure, and 2) it is assumed that no amount of exposure to a cancer-causing chemical is without some risk.

A risk assessment is an ongoing process that requires revision as science and technology provide new information. The 1990 health risk assessment of the BKK landfill updates a 1982 risk assessment and will be revised when information about vinyl chloride, one of the cancer-causing chemicals found in the air around the BKK landfill, is updated.

There were three previous health risk assessments of the BKK landfill: one by public agencies and two by private consultants to the BKK Corporation.

Previous Cancer Risk Assessments

In 1982, the Department of Health Services (DHS), the South Coast Air Quality Management District (SCAQMD), and the California Air Resources Board (CARB) monitored the air in residential neighborhoods around the BKK landfill. The following seven chemicals known or suspected to cause cancer were identified: vinyl chloride, benzene, perchloroethylene, trichloroethene, 1,1-dichloroethylene, 1,2-dichloroethane, and trichloromethane (chloroform).

Public health data show that each person has a one-in-three risk of developing cancer during a 70-year lifetime. This means that 33,000 people in 100,000 will develop cancer sometime during their lifetime. This "one-in-three" risk is referred to as a "background" risk.

The 1982 study estimated that for people living near the landfill, and exposed to all seven chemicals, the risk of developing cancer increased to 33,005 in 100,000.

In 1986, consultants to attorneys for the BKK landfill performed two separate risk assessments. In the first, the increased cancer risk from the same seven chemicals found in 1982 was estimated to be much less: between two and 11 in 1 million people. Results in the second assessment were similar: an estimated increased cancer risk

(continued on Page 6)

from the seven chemicals of two additional cases in 1 million people.

The estimates vary due to differences in the methods and assumptions used by the risk assessors. DHS has used more health-protective methods and assumptions when estimating risk. For example, DHS assumes that 100% of a chemical detected in the environment is absorbed by an individual and that individuals are at home 80% of the time. One consultant to BKK used a method that assumed that 50% of a chemical found in the environment is absorbed by an individual and that individuals are at home less than 80% of the time.

What Did The 1990 Health Risk Assessment Find?

In 1985, the United States Environmental Protection Agency (EPA) hired an engineering firm (CH2MHill) to evaluate chemical exposure to people living close to the BKK landfill. The report, completed in 1988, evaluated chemical contaminants in the air, groundwater and surface water. Chemical analysis of the soil at the landfill was not done.

DHS based the 1990 health risk assessment primarily on environmental measurements reviewed in the 1988 report. The measurements showed contaminants in groundwater (water found beneath the surface), surface water (water from steams, storm drains or run-offs) and air around the BKK landfill.

Groundwater: People living near the BKK landfill are not exposed to the contaminated groundwater because it is not used for drinking water or irrigation.

Surface water: People may have been exposed to contaminated surface water. However, the health risk assessment determined that any exposure would have involved small quantities, occurred only once in a while, and lasted only a short period of time. Consequently, the risk to people who may have been exposed to contaminated surface water is considered to be extremely small.

Air: People living near the BKK landfill may have been exposed to contaminants in the air. In 1981, vinyl chloride was found in the air at the landfill border. From 1982-1987 the amount of vinyl chloride found in residential air monitoring stations around the BKK landfill was at times above the California air quality standard of 10 parts of vinyl chloride per billion parts of air (10 ppb).

Other chemicals found in the air around the landfill are also commonly found in the Los Angeles air basin at detectable levels. However, vinyl chloride is rarely detected in the Los Angeles air basin. Therefore, vinyl chloride most clearly represents the increased risk of developing cancer associated with the landfill. Liquid wastes containing vinyl chloride were buried at the landfill until June 1981.

Vinyl chloride causes cancer in humans. Health effects other than cancer are seen at levels above 10,000 parts per billion, which is much higher than any levels found near the BKK landfill.

It is assumed that no amount of exposure to a cancer-causing chemical is without some risk. Therefore, the 1990 health risk assessment focuses on the risk of developing cancer from breathing the amount of vinyl chloride detected by air monitoring around the landfill from 1982 through 1987. The information used to estimate the risk included the amount of vinyl chloride detected in the air and the unit risk value for vinyl chloride. The unit risk value is a measure of the cancer-causing potential of a chemical. It is the estimated excess risk of developing cancer as a result of constant exposure over 70 years to one microgram of a chemical in one cubic meter of air. Unit risk values are determined by EPA and DHS toxicologists based on experimental animal and human epidemiological information.

Among people who were exposed to vinyl chloride around the landfill between 1982-1987, the health risk assessment estimates that an additional two people in 100,000 may develop cancer. This means that in every 100,000 people, 33,002 of them (rather than 33,000 as in the U.S. average) may get cancer. For the BKK study area, few if any additional cases are expected, too few to detect by a health study. Thus the cancer study and risk assessment produced similar results.

What The Health Risk Assessment May Not Show

As the health risk assessment was being completed, DHS also completed a re-evaluation of the ability of vinyl chloride to cause cancer. The re-evaluation suggests that vinyl chloride is more potent than was previously thought. This re-evaluation was not available for use in the health risk assessment.

(continued on Page 7)

The health risk assessment used the amount of vinyl chloride that was measured near the landfill between 1982 and 1987. Since 1987, vinyl chloride has rarely been detected near the landfill, so residents' exposure has been reduced. The lower exposure would reduce the risk and the estimated number of additional cancers.

Next Steps

DHS plans to conduct a new health risk assessment in 1991-92 based on both the new potency information and recent air measurements. DHS also will continue to watch the rate of cancer around the BKK landfill to see whether it increases. This is because cancer may take a number of years to develop. Residents will be kept up to date as new information becomes available.

DHS is conducting a third study at the request of people living near the landfill. This study is examining births in the area to see whether there has been any increase in such problems as infant deaths and low weight at birth. Residents will receive a summary of that study when it is completed in the first half of 1991.

Regulation of vinyl chloride by governmental agencies includes the following:

The Department of Health Services (DHS) limits the amount of vinyl chloride in drinking water to 0.5 parts per billion (ppb).

The California Occupational Safety and Health program (Cal/OSHA) limits the level of vinyl chloride in the workplace air to 100 ppb averaged over an eight-hour work period, never to exceed 100 ppb.

The California Air Resources Board (CARB) limits the concentration of vinyl chloride in the ambient air to 10 ppb, based on the technology available in 1978.

CARB is also proposing to identify vinyl chloride as a toxic air contaminant. A toxic air contaminant is an air pollutant which may cause an increase in serious illness. Identification of vinyl chloride as a toxic air contaminant allows CARB to develop control strategies that reduce vinyl chloride air levels far below 10 ppb.

If you have any questions or comments about the studies described in this newsletter, please contact:

Elinor Blake
Community Relations Coordinator
California Department of Health Services
Environmental Epidemiology
and Toxicology Branch
5900 Hollis Street, Suite E
Emeryville, CA 94608
(415) 540-3657

For information about BKK landfill cleanup activities, please contact:

Tom Mays
Public Participation Specialist
California Department of Health Services
Toxic Substances Control Program
Region 3
1405 N. San Fernando Road, Suite 300
Burbank, CA 91504
(818) 567-3176

Copies of the full epidemiologic studies of cancer in people living near the BKK landfill, the health risk assessment and other relevant documents about the BKK landfill are available at the following libraries:

La Puente Library
15920 East Central Avenue
La Puente, CA 91744
(818) 968-4613

West Covina Library
1601 West Covina Parkway
West Covina, CA 91790
(818) 962-3541

Walnut Library
21155 La Puente Avenue
Walnut, CA 91789
(714) 595-0757

CMA 114402

APPENDIX C

UPDATED TOXICITY TABLES FROM THE 1990 INTERIM REPORT

CMA 114403

UPDATED TOXICITY TABLES FROM THE 1990 INTERIM REPORT

In the 1990 interim report, OEHHA-HWTS provided a table showing the USEPA weight-of-evidence classification and site of cancer effect for the carcinogens detected in the environmental monitoring data. Since 1990, USEPA has added chemical abstract service (CAS) numbers to metals and updated the sites of carcinogenic effects associated with specific chemicals; "Lung" has been added to the site of carcinogenic effects associated with vinyl chloride (USEPA, 1994b). **APPENDIX C-TABLE 1** updates this information.

APPENDIX C-TABLE 2 updates the unit risk values for carcinogens detected at the landfill. A side-by-side comparison of the 1990 and 1994 values shows the changes (increases/decreases or no change) for each chemical. The 1994 values are taken from Cal/EPA's "Criteria For Carcinogens" (SCWG, 1994). OEHHA's slope factor for vinyl chloride is slightly more potent than USEPA's at this time: 0.27 $(mg/kg\text{-}day)^{-1}$ versus 0.29 $(mg/kg\text{-}day)^{-1}$, respectively. The weight-of-evidence classification for the carcinogens has not changed, although USEPA is in the process of reviewing the weight-of-evidence for trichloroethylene.

CMA 114404

APPENDIX C-TABLE 1[a]

CARCINOGENS DETECTED IN GROUNDWATER, SURFACE WATER, AND AIR
BKK LANDFILL WEST COVINA, CALIFORNIA
Reported in the 1988 Exposure Characterization Report[b]

Carcinogen	CAS#[c]	USEPA[d] Class	Cancer[e] Site
Arsenic (inorganic)	*7440-38-2*	A	lung, skin
Cadmium	*7440-43-9*	B1	lung, prostate, breast
Chromium(VI)[f]	*18540-29-2*	A	lung
Lead	*7439-92-1*	B2	kidney, lung, *stomach*
Nickel(subsulfide)	*12035-72-2*	A	lung, nasal
Benzene	71-43-2	A	hematopoietic system (leukemia)
Bis-2-chloroethyl ether	111-44-4	B2	liver
Carbon Tetrachloride	56-23-5	B2	liver
Chloroform	67-66-3	B2	kidney, liver, *rectum*, *colon*, *bladder*
1,2-Dichloroethane	107-06-2	B2	forestomach, breast, uterus circulatory system, lung, liver
1,1-Dichloroethylene	75-35-4	C	kidney, breast, lung
Methylene chloride (dichloromethane)	75-09-2	B2	liver, breast, lung, leukemia
N-nitrosodimethylamine	62-75-9	B2	*multiple sites*, liver
Perchloroethylene	127-18-4	C	liver
1,1,2,2-Tetrachloroethane	79-34-5	C	liver
1,1,2-Trichloroethane	79-00-5	C	liver, *pheochromocytomas*
Trichloroethylene	79-01-6	B2	lymph, liver
Vinyl chloride	75-01-4	A	liver, *lung*

a: Update of Table 3, "Health Risk Assessment of the BKK Landfill, West Covina, California," Interim Report, November 1990, page 24. Hazardous Waste Toxicology Section, California Department of Health Services, Sacramento, California. Additions are italicized and underlined (i.e., *stomach*).

b: "BKK Landfill Environmental Exposure Characterization Report, West Covina, California," Volume I, August 15, 1988, CH2M Hill, Santa Ana, California.

c: Chemical Abstract Service number.

d: USEPA weight of evidence classification: A, sufficient evidence in humans; B1, sufficient evidence in animals, limited in humans; B2, sufficient evidence in animals, inadequate in humans; C, limited evidence in animals, inadequate in humans. Taken from USEPA's Integrated Risk Information System (IRIS), November 1994, and August 1995. Database access August 1995 shows vinyl chloride, perchloroethylene, and trichloroethylene under review. With the exception of 1,1-dichloroethylene, the above carcinogens are listed as known to the State of California to cause cancer for purposes of the Health and Safety Code Section 25249.5 *et seq.*

e: Additional sites of carcinogenic effects taken from IRIS November 1994 and August 1995, with the exception of vinyl chloride. Lung site for vinyl chloride listed in "Health Effects Assessment Summary Tables: FY - 1994 Annual," March 1994, Environmental Criteria and Assessment Office, U. S. Environmental Protection Agency. 9200.6-303 (94-1) EPA 540/R-94/020; PB94-921199.

f: Inadequate evidence to quantitatively evaluate the carcinogenicity of this compound by the oral route.

CMA 114405

APPENDIX C-TABLE 2[a]

INHALATION UNIT RISK (UR) VALUES FOR CARCINOGENS DETECTED AT BKK LANDFILL, WEST COVINA, CALIFORNIA

Carcinogen	Molecular Weight	1990 UR[b] $(\mu g/m^3)^{-1}$	1994 Cal/EPA UR[c] $(\mu g/m^3)^{-1}$	1994 Cal/EPA UR[d] $(ppb)^{-1}$	USEPA[e] Class
Arsenic (inorganic)	74.92	4.3E-3	3.3E-3	1.0E-2	A
Cadmium	112.40	1.2E-2	4.2E-3	1.9E-2	B1
Chromium(VI)	52	1.5E-1	1.5E-1	3.2E-1	A
Lead	207.2	-	8.0E-5	6.8E-4	B2
Nickel	58.71	4.8E-4	2.6E-4	6.2E-4	A
Benzene	78.11	5.3E-5	2.9E-5	9.3E-5	A
Bis-2-chloroethylether	143.02	3.3E-4	7.1E-4	4.2E-3	B2
Carbon Tetrachloride	153.81	4.2E-5	NC[f]	2.6E-4	B2
Chloroform	119.39	2.3E-5	5.3E-6	2.6E-5	B2
1,2-Dichloroethane	98.96	2.2E-5	NC[f]	8.9E-5	B2
1,1-Dichloroethylene	96.95	5.0E-5	NL[g]	-	C
Methylene Chloride	84.93	1.0E-6	NC[f]	3.5E-6	B2
N-Nitrosodimethylamine	74.10	1.4E-2	4.6E-3	1.4E-2	B2
Perchloroethylene	165.85	5.8E-7	5.9E-6	4.0E-5	C
1,1,2,2-Tetrachloroethane	167.84	5.8E-5	NC[f]	4.0E-4	C
1,1,2-Trichloroethane	133.40	1.6E-5	2.1E-5	1.1E-4	C
Trichloroethylene	131.4	1.3E-6	2.0E-6	1.1E-5	B2
Vinyl Chloride	62.5	2.7E-6	7.8E-5	20E-5	A

a: Update of Table 10, "Health Risk Assessment of the BKK Landfill, West Covina, California," Interim Report, November 1990, page 47. Hazardous Waste Toxicology Section, California Department of Health Services, Sacramento, California.

b: Values listed were derived by the United States Environmental Protection Agency (USEPA) or the California Department of Health Services, Health Hazard Assessment Division, Air Toxics Section (DHS) and were taken from Table 3.15, "Air Toxics Assessment Manual," California Air Pollution Control Officers Association, October 1987 pgs. 3.5-24 to 3.5-28.

c: Taken from Standards and Criteria Work Group. (1994). "California Cancer Potency Factors: Update." California Environmental Protection Agency (Cal/EPA), Office of Environmental Health Hazard Assessment, Sacramento, California. NOTE. 1,1-Dichloroethene is no longer considered a carcinogen in California and has been deleted from the California Cancer Potency Factors. Changes in the unit risk values are due to increased knowledge about the inherent toxicity of the chemicals.

d: $(ppb)^{-1}$ = ([$\mu g/m^3$ x molecular weight] / [$24.45 m^3/\mu mole$]).

e: USEPA weight of evidence classification: A, sufficient evidence in humans; B1, sufficient evidence in animals, limited in humans; B2, sufficient evidence in animals, inadequate in humans; C, limited evidence in animals, inadequate in humans. Taken from USEPA's Integrated Risk Information System (IRIS), November 1994. Database access August 1995 shows vinyl chloride, perchloroethylene, and trichloroethylene under review.

f: NC = No Change.

g: NL = Not Listed by Cal/EPA: no change in unit risk value or classification by USEPA per IRIS, November 1994.

CMA 114406

APPENDIX D

STATISTICAL EVALUATION OF THE
1983-1990 AMBIENT VINYL CHLORIDE MONITORING DATA

CMA 114407

STATISTICAL EVALUATION OF THE 1983-1990 AMBIENT VINYL CHLORIDE MONITORING DATA

When estimating the average concentration of an environmental contaminant over time, a certain proportion of the data is often reported to be below the detection limit. Comparing two groups of data, such as site data and background data, requires summary statistics including means and standard deviations. Calculating means for samples with nondetectable concentrations requires exchanging nondetects for a value. There are three methods of estimating summary statistics for data that include samples with nondetectable concentrations: distributional, robust, and substitution methods (Helsel, 1990). Use of these methods when more than half the data are samples with nondetectable concentrations may result in biased or imprecise estimates (Hornung & Reed, 1990).

Distributional methods use the characteristics of known distributions to estimate summary statistics. A critical assumption when using distributional methods is how well the data can be expected to fit the assumed distribution. Commonly used distributions include the normal, lognormal, and Weibull. Estimates of summary statistics are computed that best match the observed concentrations above the detection limit and the percentage of data below the limit. A frequently used method for fitting points to a distribution is the maximum likelihood estimation (MLE).

Robust methods combine the observed data above the detection limit with below-limit values extrapolated in order to compute the mean and standard deviation. The robustness results primarily from the use of observed data rather than a fitted distribution above the detection limit.

Substitution methods for nondetects include using the detection limit, half the detection limit, or zero. The substitution of zero produces estimates that are biased low, and the substitution of the detection limit results in estimates considered to be above the true mean. USEPA recommends substituting half the detection limit (USEPA, 1989b).

In generating values for samples with nondetectable concentrations in the SCAQMD data, HWTS requested statistical evaluation of the data by DHS-DEODC. The fit of the air monitoring data to normal, lognormal, exponential, and Weibull distributions was evaluated using data from Stations A, B, and MY for the years 1983 through 1989.

To evaluate each distribution, Gibbs technique was used to estimate the value of samples with nondetectable concentrations as follows: first, the value of half the detection limit was substituted for samples with nondetectable concentrations; then, the MLE was used to estimate the parameters for the entire distribution; then, a random number generator was used to generate new values for samples with nondetectable concentrations. The data were generated using the given distribution and the most recent parameter estimate. The process was then iterated until a convergence criterion was met. The Gibbs method differs from other algorithms in that it requires no numerical integration. The integration is replaced by the random number generation.

The lognormal distribution showed the best fit. Arithmetic means of the lognormal distribution for vinyl chloride concentrations at Stations A, B, and MY for the years 1983 through 1989 are given in **APPENDIX D - TABLE 1**. Blanks in the table indicate insufficient data to compute the algorithm. These concentrations are similar to the concentrations reported in TABLE 6 of this addendum report. When substituted into the risk equation, average annual concentrations using the Gibbs technique result in risk estimates per year that are similar to the risk estimates using the simple substitution method for samples with nondetectable concentrations. Since the substitution method can be more easily reproduced and the results are comparable to a more sophisticated method, the substitution method was used for samples with nondetectable concentrations.

APPENDIX D - TABLE 1

AVERAGE ANNUAL CONCENTRATIONS OF VINYL CHLORIDE BASED ON GIBBS TECHNIQUE* FOR SAMPLES WITH NONDETECTABLE CONCENTRATIONS

	1983	1984	1985	1986	1987	1988	1989
Station A	2.84	4.08	3.34	1.77	1.51	1.25	-
Station B	2.98	3.88	2.20	1.24	1.12	1.87	-
Station MY	-	5.38	5.36	2.97	2.45	2.32	0.83

*The Gibbs technique was used to estimate the value of samples with nondetectable concentrations as follows: first, the value of half the detection limit was substituted for samples with nondetectable concentrations; then, the maximum likelihood estimate was used to estimate the parameters for the entire distribution; then, a random number generator was used to generate new values for samples with nondetectable concentrations. The data were generated using the given distribution and the most recent parameter estimate. The process was then iterated until a convergence criterion was met. The Gibbs method differs from other algorithms in that it requires no numerical integration. The integration is replaced by the random number generation.

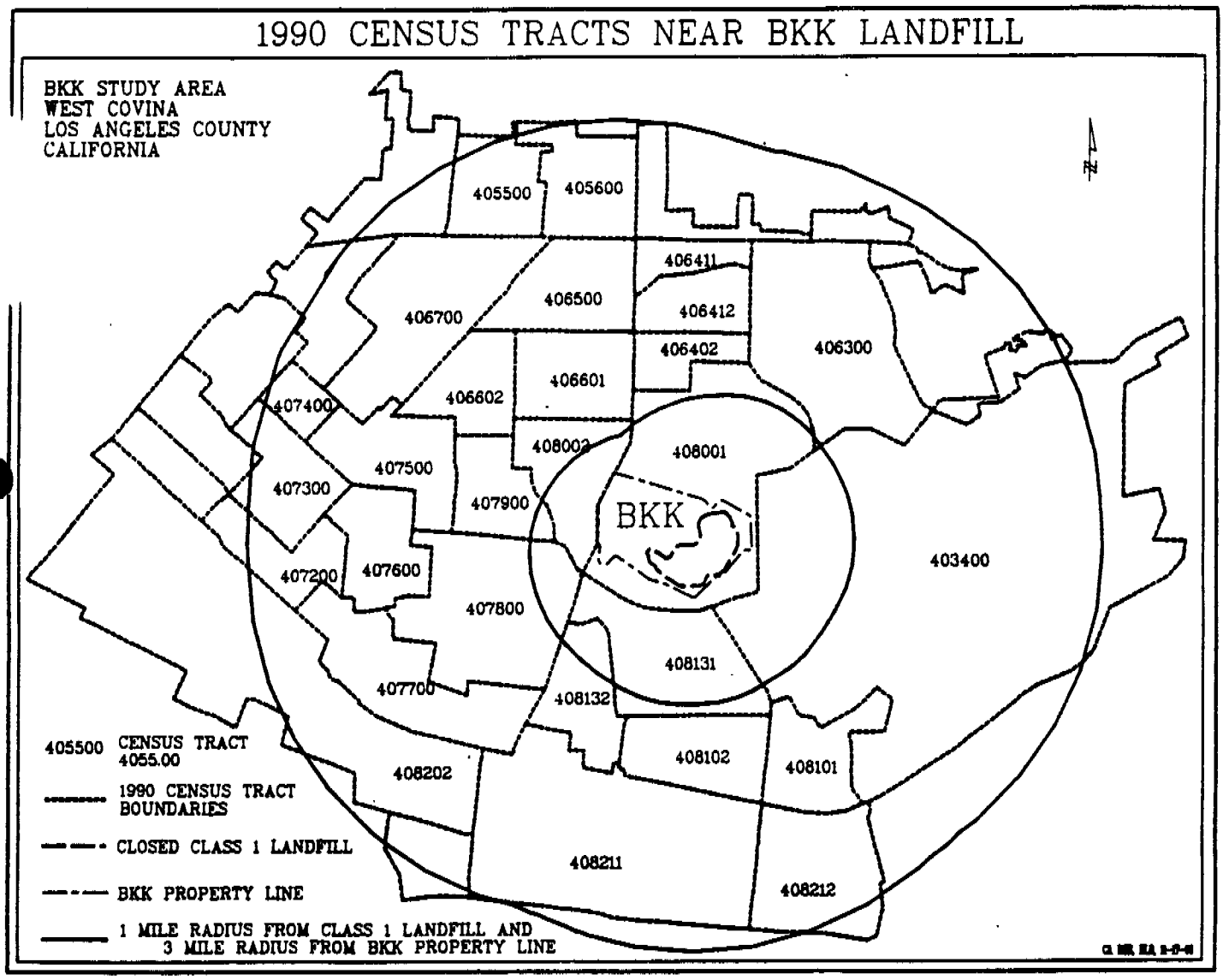
NOTE: Values computed by Joel Swartz, Ph.D., California Department of Health Services-Environmental Epidemiology and Toxicology Section, Emeryville, California, 1992, using South Coast Air Quality Management District's ambient vinyl chloride monitoring data, January 1983 through December 1989.

APPENDIX E

1990 POPULATION CENSUS TRACT DATA FOR THE VICINITY OF THE
B.K.K. LANDFILL, WEST COVINA, CALIFORNIA

CMA 114411

APPENDIX E-FIGURE 1
(Department of Health Services, Division of Environmental and Occupational Disease Control)



Courtesy of : Rachel Broadwin, California Department of Health Services, Division of Environmental and Occupational Disease Control, Environmental Health Investigations Branch, Emeryville, California, April 1994.

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APPENDIX E-TABLE 1[a]

1990 POPULATION CENSUS TRACT DATA FOR THE VICINITY OF THE BKK LANDFILL, WEST COVINA, CALIFORNIA[b]

	Contains BKK	Census Tract to the North of the BKK Landfill													
	4080.01	4080.02	4079	4075	4074	4067	4055	4056	4065	4066.02	4066.01	4064.11	4064.12	4064.02	4063
I. All Groups															
Total	6,871	7,051	5,455	6,851	1,464	8,332	5,954	4,748	5,834	4,315	4,571	2,006	2,209	1,999	4,655
Children (0-4)	582	643	458	706	108	760	481	334	455	369	313	173	121	133	237
Children (5-9)	546	610	485	688	119	646	453	364	417	339	305	143	131	95	336
Children (10-14)	484	524	506	568	115	598	408	347	424	318	314	192	159	131	358
Children (15-19)	545	520	502	565	141	644	424	339	410	304	310	160	143	125	369
Adults (20-64)	4,281	4,353	3,217	3,845	874	4,833	3,588	2,753	3,529	2,570	2,731	1,192	1,374	1,213	2,899
Adults (65+)	433	401	307	479	109	853	602	611	599	415	598	146	281	302	456
Median Age	29.9	29.4	28.7	27.6	30.2	30.5	32.4	34.0	31.5	31.1	36.2	28.9	37.6	38.0	35.7
II. Females															
Total	3,473	3,489	2,751	3,449	719	4,314	2,988	2,441	3,002	2,208	2,341	1,020	1,129	1,030	2,333
Females (0-14)	786	815	722	970	159	951	646	504	634	523	468	262	222	174	443
Females (15-44)	1,859	1,853	1,341	1,666	351	2,069	1,386	1,086	1,474	1,024	961	486	441	418	1,115
Females (45-64)	602	599	512	524	155	755	613	502	532	436	577	188	313	268	541
Females (65+)	226	222	176	267	54	539	343	349	362	223	335	88	153	170	234

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APPENDIX E-TABLE 1[a] (continued)

1990 POPULATION CENSUS TRACT DATA FOR THE VICINITY OF THE BKK LANDFILL, WEST COVINA, CALIFORNIA[b]

	Census Tracts to the South of the BKK Landfill												
	4034	4081 31	4081 32	4081 02	4081 01	4082 11	4082 12	4078	4077	4082.02	4072	4073	4076
I All Groups													
Total	28,792	13,346	9,281	8,082	7,792	5,519	4,032	7,438	11 642	2,308	6,865	7,438	7,441
Children (0-4)	2,471	1,258	952	786	627	428	262	665	1 196	207	570	596	724
Children (5-9)	2 660	1,073	931	737	609	481	268	673	1 090	192	608	641	671
Children (10-14)	2,766	1,062	873	711	614	368	346	686	908	182	657	688	655
Children (15-19)	2 464	1,178	992	891	677	455	364	740	1 047	204	675	724	687
Adults (20-64)	17,327	8,213	5,087	4,525	4,921	3,225	2,501	4 289	6,761	1,317	3 872	4,353	4,251
Adults (65+)	1,104	542	446	432	344	562	291	385	640	206	483	436	453
Median Age	31 3	28 1	24 4	25 4	29 2	30 5	32 7	27 5	26 0	28 1	27 3	28 4	26 6
II Females													
Total	14,501	6,809	4,628	3,992	3,970	2,751	2,057	3,780	5 681	1,068	3,384	3,715	3,634
Females (0-14)	3,907	1,723	1,387	1,096	908	591	436	1,019	1,534	283	839	945	1,030
Females (15-44)	7,627	3,747	2,243	2,009	2,134	1,355	974	1,903	2,864	506	1,652	1,761	1,796
Females (45-64)	2,336	1,011	741	624	729	464	457	649	883	153	639	770	576
Females (65+)	631	328	257	283	199	339	188	209	400	126	254	239	232

a Update of Table 4, "Health Risk Assessment of the BKK Landfill, West Covina, California," Interim Report, November 1990, page 34, Hazardous Waste Toxicology Section, California Department of Health Services, Sacramento, California Taken from "BKK Landfill Environmental Exposure Characterization Report, West Covina, California, "Volume I, August 15 1988, CH2M Hill, Santa Ana, California

b "United States Department of Commerce, 1990 Census of Population and Housing, population and Housing Characteristics for Census Tracts and Block Numbering Areas, Los Angeles-Long Beach CA PMSA Section 1 of 7 " August 1993 Economic and Statistics Administration, Bureau of the Census, Washington D C 1990 CPH-3-215B

CMA 114414

APPENDIX F

AIR DISPERSION MODELING OF THE
1983-1990 AMBIENT VINYL CHLORIDE MONITORING DATA

AIR DISPERSION MODELING OF THE 1983-1990 AMBIENT VINYL CHLORIDE MONITORING DATA

Ogden Environmental and Energy Services Co., Inc. (Ogden) was contracted by DHS-DEODC and OEHHA-HWTS in February 1991 to select an air dispersion model and develop a data base which would be used to model estimates of historical vinyl chloride concentrations in ambient air around the landfill. The modeled estimates were to be used in the pending DHS-DEODC reproductive outcomes study and in the present addendum health risk assessment. ARB provided technical consultation for the project and assisted staff from DEODC in refining the emission rates. In November 1992, Ogden submitted its report, "Air Quality Modeling for the B.K.K. Landfill Air Toxic Exposure Study" (Ogden, 1992).

The modeling was conducted using the Fugitive Dust Model (FDM), a Gaussian air dispersion model developed by USEPA and similar to USEPA's Industrial Source Complex (ISC) model. The ISC model is an approved model for regulatory purposes. The FDM model was selected over the ISC model because at the time the study was initiated, the FDM model was considered to estimate air concentrations close to large area sources, like the B.K.K. Landfill, better than the ISC model.

The version of ISCST available at the time of the project represented an area source by a single line source segment. Consequently, estimated concentrations for receptors close to a source ("near-field") could be under- or over-predicted. Near-field concentrations have been associated with highest detected values of vinyl chloride in monitoring around the landfill. The inability of ISCST to capture near-field concentrations was considered to be a serious limitation for this project.

Although intended primarily for the calculation of particulate concentrations, FDM is equally suited for any pollutant from non-buoyant sources. FDM uses the standard Gaussian plume formulation for computing downwind concentrations from an emission point, line, or area source. The area source algorithm was used for the B.K.K. Landfill. The algorithm provides a uniform distribution of emissions from the area source, represented as a series of line sources (rectangles) oriented perpendicular to the wind. For the purposes of this project, the area source for the model was considered to be the 170-acre closed Class I Unit.

Neither ISCST nor FDM can account for terrain influences on the direction of plume travel. Studies conducted in the 1980s around the landfill document a nighttime wind flow from the landfill down the hill to the residential community adjacent to the landfill. All of the available Gaussian complex terrain models are designed to simulate impacts on terrain elevated above the source; none are intended to simulate impacts on nearby receptors below the emission point, as exists at the B.K.K. Landfill.

Meteorological data required for the model were obtained from three sources. The primary source was SCAQMD's meteorological station located at Station A. Wind speed and wind direction data were collected hourly between 1983 and 1985 at Station A. Data missing from Station A were replaced with corresponding data from SCAQMD's meteorological station at Walnut. Additional meteorological data used in the modeling were obtained for the same time period using hourly data collected by the National Climatic Data Center at Ontario Airport in Ontario. All three years of meteorological data were combined to represent average annual meteorological conditions at the B.K.K. Landfill during the modeled time periods.

To predict annual average concentrations for the years after 1985, the three years of hourly meteorological data were run through the stability array (STAR) program. STAR is a statistical meteorological program which produces a composite wind direction frequency distribution, stratified by wind speed and stability class. Wind speed and wind direction determine both the location of pollutant impacts and their magnitude. Station A wind data were considered to be the most representative available historical information on the transport of pollutants from the landfill because vinyl chloride was monitored almost daily at Station A from June 1981 through March 1990. Walnut Station is located approximately

three kilometers south-southeast of the landfill and is influenced by different terrain features. Walnut data were not deemed as representative as the primary data source but were considered acceptable to fill in gaps missing in Station A data.

Stability class is a measure of the dispersive capacity of the atmosphere. It is not measured directly but derived from other measurements. USEPA developed a data processing package called RAMMET which uses concurrent sky cover measurements and wind speed to derive stability class. Hourly stability categories were developed using the RAMMET program and sky cover data from Ontario Airport.

Mixing height determines the depth through which pollutants can disperse in the atmosphere. Modeling for ground-level sources such as the B.K.K. Landfill are mostly insensitive to mixing height. Hourly mixing heights were calculated using the RAMMET program and average, seasonal mixing heights interpolated from values published for the contiguous United States.

Annual vinyl chloride emission rates were estimated from continuous 24-hour average vinyl chloride concentrations measured at SCAQMD's three residential monitoring stations south of the landfill, Stations A, B, and MY. Average annual concentrations were then determined for each monitoring station, with the nondetected samples set at one-half the detection limit (1983 through 1987, 2 ppb; January 1988 through September 1989, 1.4 ppb; October 1989 through March 1990, 2 ppb).

To calculate emission rates, the FDM model was run with a nominal emission rate for each year at each monitoring station when monitoring data were available. All of the vinyl chloride was assumed to have originated homogeneously from a rectangle approximating the Class I unit. The emission rates were then scaled so that the modeled concentrations equaled the measured concentrations. The highest average annual emission rate from the three stations for each year was selected as representative of vinyl chloride emissions for that year. For all years except 1988, the highest emission rate was obtained from Station A. For 1988, the highest emission rate was obtained from Station MY.

ARB provided technical consultation to the project. ARB recommended running tests to estimate the difference that would be predicted using different meteorological data sets (SCAQMD's Station A and Walnut Station) and different models (USEPA's regulatory models, ISCST and SCREEN; and FDM).

To test the effects of using Station A meteorological data versus Walnut data, FDM was run twice: first using hourly Station A data for the time period October through December 1984, then using the wind data from Walnut for the same time period. In both tests, the model was run with a unit emission rate, and the landfill emission rate was then back-calculated from the measured concentrations at Station A, B, and MY. The results showed that significant differences in emission rates were obtained, with smaller emission rates obtained using Station A data. The conclusion for this project was that the site-specific data from Station A would more accurately reflect the micrometeorology around the B.K.K. Landfill.

Comparison of the FDM and ISCST models was made using the estimated emission rates for January-March 1983. The results showed similar isopleth contours and a similar point of maximum impact, located on the landfill boundary. However, the maximum concentration predicted by FDM was almost three times higher than that predicted by ISCST. The conclusion for this project was that FDM would not underpredict near-field receptors, such as Stations A, B, and MY.

All air dispersion models are designed to be health protective. SCREEN is a worst-case model which, rather than using actual meteorological data, calculates hourly concentrations from a matrix of wind speeds and stability conditions, and reports the maximum. Comparison of the FDM and SCREEN models was made using emission rates for Station A for the time period January-March 1983. Results showed that SCREEN predicts concentrations three- to ten-fold greater than FDM. The conclusion for this project was that the order of health protective conservatism built into the three models is: SCREEN > FDM > ISCST, as desired.

APPENDIX F-FIGURE 1 is an isopleth of vinyl chloride at 2.1, and 0.05 ppb modeled concentrations around the B.K.K. Landfill in 1989 generated by FDM. The purpose of the isopleth is to show the general direction of the plume. Confidence in the extent of contamination depicted in the isopleth is low for the following reasons:

- All of the vinyl chloride was assumed to have originated homogeneously from a rectangle approximating the Class I Unit. This assumption may overestimate concentrations at residential locations on the eastern side of the landfill and underestimate concentrations at residential locations on the western side of the landfill since vinyl chloride may have been released from other areas of the landfill. Similarly, the assumption does not estimate potential localized "hot spot" areas of vinyl chloride emissions;

- Vinyl chloride monitoring data and the meteorological data were solely from the southern boundary of the landfill. Less confidence can thus be placed on modeled estimates at other areas around and away from the landfill;

- Emission rates were developed from average annual vinyl chloride concentrations calculated with nondetected measurements set at one-half the detection limit. Between 37 and 100 percent of the measurements in any given year were below the detection limit. This assumption may produce overly conservative concentration estimates. In 1989, 99 percent of the data from Station A was non-detects;

- The emission rate estimated for each year was the highest emission rate back-calculated from the data at the three stations. Emission rates calculated using Station A data were on the average 139 percent higher than emission rates calculated using Station B data. This assumption will overestimate concentrations near stations which were not used to estimate a given year's emission rate, and may result in higher concentration estimates in other areas as well.

In addition to data and methodology limitations given above, there are uncertainties and limitations inherent to all Gaussian air dispersion modeling, the extent of which, regarding the B.K.K. Landfill, were not apparent until after working with the model:

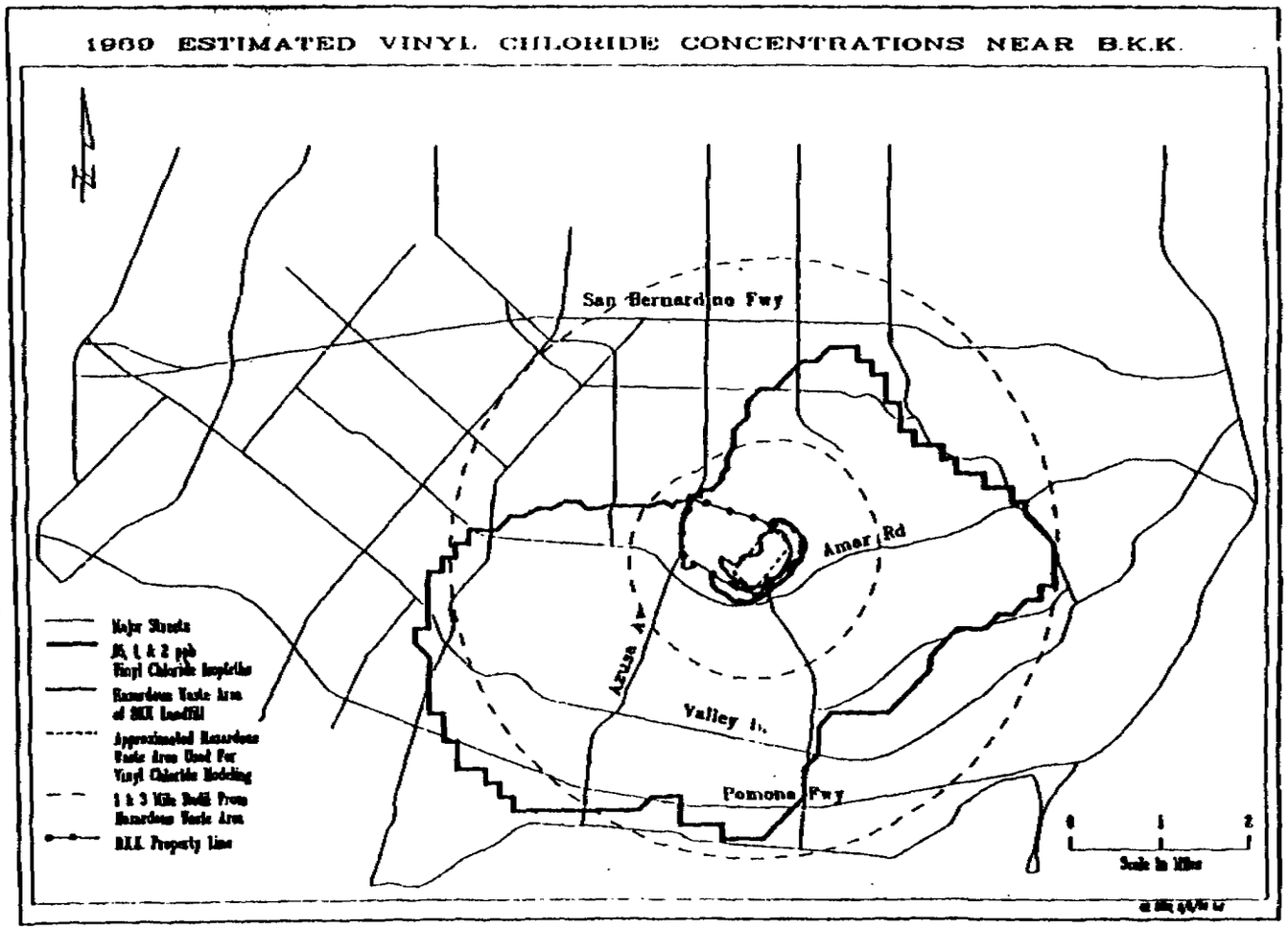
- Gaussian models cannot calculate concentrations accurately when wind speeds fall below one meter per second (m/s). As recommended by ARB, all wind speeds which fell below one m/s were adjusted to one m/s. At the B.K.K. Landfill, wind speeds below one m/s occurred 60 percent of the time. Setting a lower wind speed boundary at one m/s would tend to overestimate the back-calculated emission rates. The effect is somewhat offset by the fact that emission rates were back-calculated from measured data near the landfill.

- Gaussian models cannot factor in complex, topographical terrain, as exists around the B.K.K. Landfill; that is, models cannot account for an emission source which is at a higher elevation than the receptors.

CMA 114418

APPENDIX F-FIGURE 1
(Department of Health Services, Division of Environmental and Occupational Disease Control)

Note: Confidence in the extent of modeled vinyl chloride concentrations is low because of the assumptions used in, and limitations and uncertainties of, the Fugitive Dust Model. In the figure, the 2 ppb vinyl chloride isopleth is on the landfill, the 1 ppb vinyl chloride isopleth extends to Amar Road, and the 0.05 ppb vinyl chloride isopleth extends to the three mile radius southwest and east of the landfill. The purpose of the isopleth is to show the general direction of the estimated modeled plume.



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APPENDIX G

UPDATED RISK ESTIMATE OF THE 1984 INDOOR AIR
MONITORING PROGRAM EVALUATED IN THE 1990 INTERIM REPORT

UPDATED RISK ESTIMATE OF THE 1984 INDOOR AIR MONITORING PROGRAM EVALUATED IN THE 1990 INTERIM REPORT

This Appendix revises the risk estimate for the USEPA-DHS 1984 indoor air monitoring program using updated unit risk values.

On July 17, 1984, 19 homes along the southern and southeastern border of the B.K.K. Landfill were evacuated when methane, from lateral subsurface landfill gas migration, was detected at concentrations above the lower explosive limit (50,000,000 ppb). The Southern California Gas Company detected the methane gas during a routine (every four years) survey of residential gas distribution piping system. Methane gas migrating from a landfill can serve as a carrier gas for nonmethane hydrocarbons, such as vinyl chloride, which are deposited or generated at landfills. On July 18, 1984, a team from SCAQMD, USEPA, and DHS collected grab air samples in 2-liter bulb containers from inside the evacuated homes. The samples were analyzed for methane, nonmethane hydrocarbons, vinyl chloride and selected volatile organic compounds. Ten of the evacuated homes were designated "Priority I" based on the concentrations of vinyl chloride detected. The highest detection of vinyl chloride in the 2-liter bulb containers was 990 ppb. (Five days later, analysis inside the same home showed 160 ppb vinyl chloride, and on August 1, 1984, vinyl chloride was not detected at or above the detection limit of 2 ppb.)

In October 1984, the ten Priority I homes and one "control" home were resampled. Samples were collected in Tedlar bags over 24-hours. The purpose of the indoor air monitoring program was to determine the safety of reoccupying the homes. There was a discrepancy regarding the concentrations of benzene detected in the Priority I homes. CH2M Hill reported laboratory contamination. However, no documentation or explanation was provided, and the benzene data were included in the appendices of the exposure characterization (CH2M Hill, 1988).

To estimate the duration of exposure for the risk characterization in the interim report, HWTS reviewed the records of the Southern California Gas Company. According to the records reviewed, elevated "field gas" (combustible gas originating from natural decomposition of organic material and not the company's metered gas) was detected at one residential location near Station B in May 1984. The detection was determined to be not hazardous. Beginning July 3, 1984, other residential locations in the vicinity of Station B were surveyed. Detections of field gas were determined by the gas company to pose a hazard to people or property. The gas company continued to monitor the area, leading to the evacuation on July 17, 1984.

HWTS estimated that the duration of exposure to landfill gas contaminants in indoor air of the evacuated homes was no more than 1-year. This assumption may underestimate or overestimate the exposure. An article published in 1987 described the gas migration as "fresh" and "new," and stated that "it may not have been going on long enough for steady state conditions to have been achieved" (Wood and Porter, 1987).

The limited scope of the indoor air program (grab samples in July and 24-hour samples in October) precludes its usefulness in the long-term assessment of this addendum report.

APPENDIX G-TABLE 1 updates the risk estimate associated with the five carcinogens detected in the evacuated homes in October 1984. The individual excess lifetime cancer risk associated with an assumed one-year exposure to the concentrations detected in the homes is estimated to be approximately 2×10^{-5}. Vinylidene chloride (1,1-dichloroethene) is no longer considered a carcinogen in California and has been deleted from the list of California Cancer Potency Factors. Consequently, it is not included in the revised risk estimate.

APPENDIX G-TABLE 1[a]

INDIVIDUAL EXCESS LIFETIME CANCER RISK[b]
USEPA-DHS 1984 EXTENDED MONITORING PROGRAM
(INDOOR AIR, EVACUATED HOMES)
BKK LANDFILL, WEST COVINA, CALIFORNIA

Carcinogen	Median[c] ppb	Median[c] $\mu g/m^3$	Unit Risk[d] $(\mu g/m^3)^{-1}$ 1990	Unit Risk[d] $(\mu g/m^3)^{-1}$ 1994	Cancer Risk 1990	Cancer Risk 1994	USEPA[e] Class
Vinyl Chloride	4	10.2	2.7 E-6	7.8 E-5	3.1 E-7	9.1 E-6	A
Perchloroethylene	2.5	17 0	5.8 E-7	5.9 E-6	1.1 E-7	1.1 E-6	C
Trichloroethene	1.5	8.1	1.3 E-6	2.0 E-6	1.2 E-7	1.9 E-7	B2
1.2-Dichloroethane	3	12.1	2.2 E-5	2.2 E-5	3.0 E-6	3.0 E-6	B2
Benzene	7	22.4	5.3 E-5	2.9 E-5	1.4 E-5	7 4 E-6	A
Total Excess Lifetime Cancer Risk Including Benzene					1.8 E-5	2.1 E-5	
Total Excess Lifetime Cancer Risk Excluding Benzene					3.5 E-6	1.3 E-5	

a: Update of Table 16, "Health Risk Assessment of the BKK Landfill, West Covina, California, Interim Report, November 1990," pg. 68, Hazardous Waste Toxicology Section, California Department of Health Services, Sacramento, California.

b: Calculated by ECR = C x YR x EF x UR x 1/LT where

- ECR = Excess lifetime cancer risk
- C = Concentration in $\mu g/m^3$
- YR = Years of residence associated with exposure (1)
- EF = Exposure factor, i.e. percent of time at home (80%)
- UR = Unit risk value
- LT = Human individual lifetime in years (70)

c: Median values taken from Table 6-13, "BKK Landfill Environmental Exposure Characterization Report, West Covina, California," CH2M Hill, 1988, Volume I, p. 6-45.

d: 1990 values were derived by the United States Environmental Protection Agency (USEPA) or the California Department of Health Services, Health Hazard Assessment Division, Air Toxics Section and were taken from Table 3.15, "Air Toxics Assessment Manual," California Air Pollution Control Officers Association, October 1987 pgs 3.5-24 to 3.5-28. 1994 values were taken from the Standards and Criteria Work Group, "California Cancer Potency Factors: Update 1994", California Environmental Protection Agency (Cal/EPA, Office of Environmental Health Hazard Assessment), Sacramento, California. NOTE: 1,1-Dichloroethene is no longer considered a carcinogen in California and has been deleted from the California Cancer Potency Factors. Consequently, it is not included in the table update. Changes in the unit risk values are due to increased knowledge about the inherent toxicity of the chemicals.

e: USEPA weight of evidence classification: A, sufficient evidence in humans; B1, sufficient evidence in animals, limited in humans; B2, sufficient evidence in animals, inadequate in humans; C, limited evidence in animals, inadequate in humans. Taken from USEPA's Integrated Risk Information System (IRIS), November 1994.

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APPENDIX H

DEPARTMENT OF HEALTH SERVICES-CANCER SURVEILLANCE SECTION
LETTER REGARDING REVIEW OF RECENT TUMOR INCIDENCE DATA

TE OF CALIFORNIA—HEALTH AND WELFARE AGENCY
PETE WILSON, Governor

PARTMENT OF HEALTH SERVICES
744 P STREET
BOX 942732
RAMENTO, CA 94234-7320
(916) 540-2711

November 16, 1995

RECEIVED

NOV 20 1995

Hazardous Waste Toxicology Section

Robert L. Holtzer M.D.
Hazardous Waste Toxicology Section
Office of Environmental Health Hazards Assessment
601 North 7th Street/MS 241
P.O.Box 942732
Sacramento, CA 94234-7320

Dear Dr. Holtzer,

In response to your request, the Cancer Surveillance Section of the Department of Health Services, in collaboration with the Cancer Surveillance Program of Los Angeles, has examined the incidence of invasive cancer in the six census tracts (see enclosed map) around the BKK landfill in West Covina. We have compared the observed cancer incidence in the area with the incidence expected on average if the residents had the same cancer rates as Los Angeles County as a whole.

As you know, California has had a statewide cancer reporting system since 1988. The California Cancer Registry (CCR) has information on every cancer case diagnosed in a California resident after January 1, 1988, with the exception of squamous and basal cell carcinomas of the skin, which are very common and usually treated in doctors' offices. The information gathered includes addresses at the time of diagnosis, which are allocated to the appropriate census tracts. Data for Los Angeles County are collected by the Cancer Surveillance Program of Los Angeles, University of Southern California.

These data enabled us to obtain the observed number of cancers, that is the number of invasive cancers of all anatomical sites combined that were actually diagnosed among residents of the area of the six census tracts during the six-year period 1988-93. We estimated the expected number by applying the average annual incidence rates of invasive cancer for Los Angeles County, specific for five-year age group, race, and sex, to the corresponding 1990 population data for the census tracts. Assuming the population either to be stable or to have increased linearly between 1988 and 1993, multiplying the expected numbers for 1990 by six, produces estimates of the cases expected during the six years, 1988-93. The table below shows the numbers.

THE INCIDENCE OF INVASIVE CANCER IN THE BKK LANDFILL AREA
ALL RACES, ALL ANATOMICAL SITES COMBINED
LOS ANGELES COUNTY, 1988-93

Census Tract	Expected No. 1990	Expected No. 1988-93	Observed No. 1988-93
4034	56.3	338	333
4078	17.0	102	114
4079	12.6	76	85
4080.01	18.7	112	114
4080.02	16.8	101	81
4081.31	23.7	142	128
Total	**145.1**	**871**	**855**

As the the table shows, the observed number of cases in the entire area, 855, was actually lower than the number expected, on average, during this time period, 871. Neither this difference nor the differences between the observed and expected numbers for the individual census tracts are statistically significant: the differences are within the bounds of what can be expected to occur, in either direction, by chance alone.

No cases of hemangiosarcoma of the liver were reported among residents of these six census tracts during the years 1988-93. Of the 27 hemangiosarcomas diagnosed in California during this time period, seven occurred in Los Angeles County, a proportion roughly equivalent to the population proportion in Los Angeles. Only one case occurred within five miles (neither downwind nor downwater) of the BKK landfill. This was in an elderly person who moved into the area in 1989, and was diagnosed a few years later. Thus there is no indication of an increase in hemangiosarcoma of the liver in the area around the BKK landfill durin this time period.

In summary, for the six-year period, 1988-93, there is no indication that the incidence of invasive cancer among residents of the area around the landfill has been different than cancer incidence among residents of the entire Los Angeles region.

If there are any questions, please call me at (510) 540-2711.

Sincerely,

Eva R. Glazer, M.D., M.P.H.
Cancer Surveillance Section
2151 Berkeley Way, Annex 2
Berkeley, CA 94704

cc: John L. Young Jr., Dr.P.H., CTR
Cancer Surveillance Section
P.O. Box 942732 MS 592
Sacramento, CA 95814

Wendy Cozen D.O., M.P.H.
USC Cancer Surveillance Program
1540 Alcazar St., CHP-204
Los Angeles, CA 90033

Phil Jacobs
Toxics Epidemiology Program
Los Angeles County Department of Health Services
510 S. Vermont, Room 215
Los Angeles, CA 90020

Richard Kreutzer, M.D.
Environmental Health Investigations Branch
5900 Hollis Street, Suite E
Emeryville, CA 94608

APPENDIX I

RELEASE OF THE DRAFT ADDENDUM REPORT

CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

NOTICE TO INTERESTED PARTIES
DECEMBER 15, 1995

NOTICE OF AVAILABILITY OF DRAFT ADDENDUM HEALTH RISK ASSESSMENT OF AMBIENT FUGITIVE VINYL CHLORIDE EMISSIONS FROM THE CLASS I UNIT OF THE B.K.K. LANDFILL, WEST COVINA, CALIFORNIA

and

NOTICE OF PUBLIC WORKSHOP

The Office of Environmental Health Hazard Assessment (OEHHA) released the "Draft Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions From the Class I Unit of the B.K.K. Landfill, West Covina, California" for a 60-day public comment period beginning December 15, 1995.

The 1995 draft addendum health risk assessment updates the 1990 interim "Health Risk Assessment of the B.K.K. Landfill, West Covina, California."

The draft addendum health risk assessment will be the subject of a public workshop to be held on Thursday, January 11, 1996, from 7:30 p.m. to 10:00 p.m., at the City of West Covina Council Chambers, located on the First Floor of City Hall, 1444 West Garvey Avenue, West Covina, California. Comments may be presented during the workshop, or submitted in writing to the address below. Written comments must be received by OEHHA on or before February 13, 1996.

At the end of the comment period, the final report will be prepared and released. The addendum report plus the interim report will complete the health risk assessment of the B.K.K. Landfill promised to the residents by the State of California.

Please direct any questions regarding the workshop or the draft addendum health risk assessment to Lillian Kelly at the address and telephone number indicated below. Copies of the draft addendum health risk assessment may be obtained by submitting a written request.

Lillian Kelly, M.P.H.
Office of Environmental Health Hazard Assessment
Hazardous Waste Toxicology Section
601 North Seventh Street, MS 241
P. O. Box 942732
Sacramento, California 94234-7320
PHONE No. (916) 324-2829
FAX No. (916) 322-9705

Copies of the draft addendum and interim health risk assessments and other health studies regarding the B.K.K. Landfill are available to be read on the premises at the following libraries:

West Covina Library
1601 West Covina Parkway
West Covina, California 91790
(818) 962-3541

Walnut Library
21155 La Puente Avenue
Walnut, California 91789
(909) 595-0757

La Puente Library
15920 East Central Avenue
La Puente, California 91744
(818 968-4613

APPENDIX J

OEHHA'S RESPONSE TO COMMENTS ON THE DRAFT ADDENDUM REPORT

OEHHA's Response to Comments From Regulatory Agencies

United States Environmental Protection Agency - Region IX

OEHHA'S RESPONSE TO COMMENTS FROM USEPA

Response to Comments From Gerald F.S. Hiatt, Ph.D., Senior Risk Assessment Advisor

1. Cal/EPA has participated with USEPA in the evaluation and presentation of USEPA's assessment of increased sensitivity of infants and children to inhaled vinyl chloride. USEPA's October 20, 1995 policy statement says that "the Agency will develop a separate assessment of risks to infants and children." To date, quantitative assessments of young-age sensitivity to inhaled vinyl chloride have not been used extensively in assessments such as the addendum report. OEHHA's unit risk for vinyl chloride is derived from the Drew *et al.* studies in female mice, and includes consideration of increased sensitivity of young animals to vinyl chloride induced carcinogenicity. In selecting this value OEHHA noted: "The animal studies demonstrate a relationship between tumor formation and the sex and age of the animal at first exposure. Fetuses, newborns, younger animals, and females exhibited the highest carcinogenic sensitivity (Drew *et al.*, 1983). In the epidemiological studies of vinyl chloride workers, who were predominantly male, the average age at first exposure was 29.7 years. Thus, to protect all members of the general population, it is more appropriate to base risk assessment calculations on the animal inhalation studies, which because of their use of more sensitive categories, the young and females, reflect a wider range of population sensitivity."

   Utilizing the USEPA's approach to evaluate the theoretical upperbound excess lifetime risk for exposure to vinyl chloride at a young age suggests a risk estimate at Station A that is approximately 14-fold higher than the estimate obtained in the addendum report. This qualitative estimate has been added to the Risk Characterization, Limitations and Uncertainties.

2. USEPA's guidance states that "Sampling data from Superfund sites have shown that data sets with fewer than 10 samples per exposure area provide poor estimates of the mean concentration (i.e., there is a large difference between the sample mean and the 95 percent UCL), while data sets with 10 to 20 samples per exposure area provide somewhat better estimates of the mean, and data sets with 20 to 30 samples provide fairly consistent estimates of the mean (i.e., the 95 percent UCL is close to the sample mean). Remember that, in general, the UCL approaches the true mean as more samples are included in the calculation (USEPA, 1992a)." TABLE 7 of the addendum report indicates that the minimum number of samples collected by SCAQMD in one year is 191. Thus, we believe that the average annual concentrations used in the addendum report are justified.

3. Discussion of the underestimation of exposure at Station MY for the years 1980 through 1983 has been expanded in Risk Characterization, Assumptions Regarding Ambient Vinyl Chloride Monitoring Data. For the years 1985 through 1988, concentrations of vinyl chloride at Station MY were 1.6- to 1.9-fold higher than concentrations at Station A, and Station MY had between 25 to 33 percent fewer samples with nondetectable concentrations.

Response to Comments From Carmen D. Santos-Prior, Project Manager, B.K.K. Landfill

1. The sentence has been corrected.

2. Information about the subsurface soil gas investigation USEPA is requiring the B.K.K. Corporation to conduct has been added.

3. The scope of the risk assessment required of the B.K.K. Corporation under the USEPA Order has been expanded.

4. Please see Exposure Assessment, Other Hazardous Chemicals Detected in Ambient Air.

5. Information about the Response Action Plan for Vinyl Chloride has been added to the Hazard Identification section, Vinyl Chloride Emissions From the Class I Unit.

6. Correspondence from SCAQMD (SCAQMD, 1994a) has been added as the reference for SCAQMD's acceptance of B.K.K. Corporation's contract laboratory protocol for vinyl chloride.

ENCLOSURE 1

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION IX
75 Hawthorne Street (H-9-3)
San Francisco, CA 94105

MEMORANDUM February 13, 1996

Subject: **BKK - Cal/EPA Draft Risk Assessment**

From: **Gerald F.S. Hiatt, Ph.D.**
Senior Risk Assessment Advisor Gerald FS Hiatt

To: **Carmen Santos**
Project Manager - BKK Site

Per your request, I have reviewed the Draft Addendum Health Risk Assessment of Ambient Vinyl Chloride Emissions from the Class I Unit of the B.K.K. Landfill, West Covina, California, prepared by the Office of Environmental Health Hazard Assessment of Cal/EPA and dated December 1995.

In general, the risk assessment is well written and represents a thorough analysis of the relatively extensive data collected by the various airborne vinyl chloride monitoring efforts over a number of years at the BKK site. I was especially impressed by the various approaches the authors used to incorporate non-detects from the earlier years of vinyl chloride monitoring (when limits of detection were in the 2 to 10 ppb range) and the various different ways that risks are apportioned over the entire period of time covered by the draft risk assessment.

There are two specific issues on which the Cal/EPA approach used in this risk assessment differs from the procedures used by U.S. EPA and these are worthy of comment, as is one of the vinyl chloride exposure assumptions used by Cal/EPA:

1. Vinyl Chloride Risks in Children: Under U.S. EPA risk assessment policy and guidelines, the risk assessment should quantitatively address the sensitivity of young children to the carcinogenic action of inhaled vinyl chloride for less-than-lifetime exposure scenarios. It is U.S. EPA policy to explicitly assess risks to children whenever possible:

> *"It is the policy of the U.S. Environmental Protection Agency (EPA) to consider the risks to infants and children consistently and explicitly as a part of risk assessments ... the Agency will develop a separate assessment of risks to infants and children ..."* (Browner: New Policy on Evaluating Health Risks to Children (memo), October 20, 1995).

Consistent with this policy, and with reports in the scientific literature documenting the enhanced sensitivity of neonates to the carcinogenic action of inhaled vinyl chloride, the U.S. EPA Region 9 Office and Office of Research & Development derived a procedure for assessing enhanced risks to infants and children from inhaled vinyl chloride. This procedure has been adopted as risk assessment guidance within Region 9, has been presented and discussed at a number of national EPA and independent conferences on risk assessment, has been presented in a number of scientific publications (including peer-reviewed journals) and has been incorporated quantitatively into risk assessments at three hazardous waste sites in Region 9 (including the on-going ambient air study at BKK).

This issue is especially important in light of the fact, as detailed in the draft report (pp. xii, 63), that most of the exposure (and hence risk) during the entire period covered by the risk assessment accrued during the early 1980's - vinyl chloride levels detected by the monitoring program decreased significantly after this period. Because of the childhood sensitivity, this disproportionate accrual of risk will be further enhanced for anyone who was an infant or young child in the community during that period.

2. Exposure Concentration Term: The draft risk assessment uses the mean (arithmetic average) of the monitoring data as the vinyl chloride concentration term in the exposure equations (first paragraph, p. 54). U.S. EPA risk assessment guidance for the Office of Solid Waste and Emergency Response (OSWER) is explicit on this point - the concentration term in exposure equations should be the 95% upper confidence limit on the arithmetic mean (95% UCL):

> "... *the concentration term ... in the intake [exposure] equation is an estimate of the arithmetic average concentration for a contaminant based on a set of site sampling results. Because of the uncertainty associated with estimating the true average concentration at a site, the 95 per cent upper confidence limit (UCL) of the arithmetic mean should be used for this variable.*" (Supplemental Guidance to RAGS: Calculating the Concentration Term. OSWER Publ 9285.7-081, May 1992.)

Please note that the 95% UCL is used as "an estimate of the arithmetic average", not as an "upperbound" estimate as implied by the draft BKK risk assessment (p. 54).

3. MY Area - 1983 Vinyl Chloride Concentration: For the draft risk assessment "[t]he 1983 average annual concentration at

Station MY is assumed to be 2.8 ppb, that is at least equal to Station A's 1983 average annual concentration ..." (p. 52) - this results in an assumed exposure during 1983 which is almost one-half of that during 1984 (5.4 ppb). This assumption probably underestimates actual vinyl chloride levels in the MY area by a significant amount. As is apparent from the graph on page 31, for all of the years monitored prior to 1989, vinyl chloride levels in the Station MY area were significantly greater than, not equal to, those at Station A. Also, the overall trend in the monitored areas is for vinyl chloride levels to spike in 1983 and then generally decrease over the rest of the monitoring period (p. 31).

These two trends indicate that the assumption of equivalent exposures in the MY and A areas probably understates the actual exposure levels for the former area. It may not be possible to incorporate this consideration into the risk assessment in a quantitative fashion, but a more thorough discussion would highlight the uncertainty this assumption brings to the risk calculations.

If you have any questions or need any clarification on these comments, I can be reached by voice at (415) 744-2319 or by fax at (415) 744-1916.

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CMA 114436

OEHHA's Draft Addendum Health Risk Assessment
BKK Landfill, West Covina, California

U.S. Environmental Protection Agency
Additional Comments

February 12, 1996

The U.S. Environmental Protection Agency has the following additional and general comments on the OEHHA ***Draft Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions from the Class I Unit of the B.K.K. Landfill, West Covina, California*** dated December 1995.

Additional Comments

1) **Objective, Methodology, and Scope of the Draft Addendum Health Risk Assessment, Page 3, Second Paragraph:** The closed Class I Unit is not under a RCRA permit as stated. BKK is seeking a post closure permit which is now being drafted by Cal-EPA DTSC. The Class I unit is currently regulated under interim status.

2) **Objective, Methodology, and Scope of the Draft Addendum Health Risk Assessment, Page 4, Second Paragraph:** The report states that "At this time, there is no evidence that subsurface landfill gas migration is a problem for homes in close proximity to the landfill". EPA recommends that this sentence be deleted because BKK still needs to conduct a subsurface soil gas investigation to determine if a potential could exist for migration of subsurface soil gas to occur despite the gas collection system. BKK will conduct this investigation under the RCRA Section 3008(h) Corrective Action Order on Consent, U.S. EPA Docket No. RCRA-09-89-0019 (Order). EPA is currently reviewing BKK's subsurface soil gas investigation work plan.

3) **Previous Health Risk Assessments of the B.K.K. Landfill, Page 5, Last Paragraph:** The report states that "The current health risk assessment required by USEPA will be based on a one-year ambient air sampling program of 19 volatile organic compounds, including vinyl chloride at onsite and offsite locations (B.K.K. Corporation et al., 1994)." As to the scope of the health risk assessment that BKK is required to conduct under the EPA Order, the cited statement provides a limited description of the scope for this risk assessment.

U.S. Environmental Protection Agency
Additional Comments (Continued)
February 12, 1996

The risk assessment required of BKK under the Order will address evaluations of all environmental media: groundwater, soils, air and if possible, surface water. The air medium will be evaluated for the ambient air pathway and the subsurface soil gas pathway in order to achieve a complete evaluation of the air medium. Please expand on the scope of the risk assessment required under the EPA Order.

4) **Hazard Identification, Vinyl Chloride in the Class I Unit, Page 9, Paragraph 1:** EPA agrees that vinyl chloride is one of the chemicals of concern and a very important one. However, data from previous ambient air sampling studies were not sufficient to demonstrate the potential contribution of the other chemicals to the risk calculations. EPA recommends that these limitations of previous sampling programs be discussed in this context.

5) **Hazard Identification, Vinyl Chloride Emissions from the Class I Unit, Page 10-12:** This Section does not make mention of the BKK Response Action Plan for Vinyl Chloride (RAP) dated July 21, 1994 which EPA approved on August 4, 1994 (EPA letter to BKK corporation, August 4, 1994, Re: BKK's Response Action Plan for Vinyl Chloride - EPA Approval and Errata Sheet). BKK submitted and implemented the RAP under the Order. Under the RAP, BKK took actions (e.g., increase maintenance of the Class I landfill cap by sealing cracks; pumping of liquids from gas collection wells to increase gas collection capacity) for the purpose of reducing vinyl chloride emissions whenever ambient air sampling results showed vinyl chloride at concentrations equal to or above 0.5 ppbv.

6) **Hazard Identification, Vinyl Chloride Emissions from the Class I Unit, Page 12:** The report states at the bottom of the paragraph that "USEPA has stated that the analytical procedure appears to be acceptable to USEPA and SCAQMD for the purposes of ambient air monitoring at the B.K.K. Landfill (USEPA, 1994e)". EPA recommends that OEHHA cite SCAQMD correspondence directly regarding SCAQMD's opinions about the BKK vinyl chloride analytical procedure with 0.005 ppbv detection limit instead of EPA correspondence.

OEHHA's Response to Comments From Interested Parties

Jean Arneson

B.K.K. Corporation

Chemical Manufacturers Association

William F. Polich, Ph.D.

OEHHA'S RESPONSE TO COMMENTS FROM JEAN ARNESON

1. ARB's records (ARB 1982a) indicate that vinyl chloride was deposited prior to 1981. There could very well have been exposures for residents near Station A from the period 1977 to 1981 since residential development near Station A was completed in 1977. However, it is not possible to quantitatively estimate that exposure. The report has been modified to reflect that 1981 was the single year of highest detected exposure to vinyl chloride.

2. More information has been added to Appendix G concerning the 1984 indoor air sampling for vinyl chloride.

3. Although other hazardous chemicals were emitted from the B.K.K. Landfill, it is not possible with the available data to state that emissions of other hazardous chemicals from the B.K.K. Landfill were significant. Because these other chemicals typical pollutants found in the ambient air of the South Coast air basin, determining the significance of any specific source is not possible with the available data.

February 9, 1996

David M. Siegel, Ph.D. Chief
Hazardous Waste Toxicology Section
California Environmental Protection Agency
601 N. 7th St. MS-241
P.O.Box 942732
Sacramento, Calif. 94234-7320

Dear Dr. Siegal,

I have some concerns about the Draft Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions from the Class I Unit of the BKK Landfill, West Covina, California.

The assumption is made that because no monitoring data is available for the years 1978-1980, that 1981 was the single year of highest exposure to vinyl chloride. Since vinyl chloride dumping stopped in June 1981, isn't it logical to assume that more vinyl chloride was dumped in the years prior to 1981 and there could very well have been years of higher exposure. Therefore if this is to be a truly conservative estimate, shouldn't the report state that the actual excess cancer risk might be higher for any residents living near StationsA, B, and MY since 1978. Especially if the report is to be a guide to ensure future risks do not add significant cumulative risk, we need to consider all those people impacted since vinyl chloride was dumped at BKK..

Also Appendix G-Table 1a has a footnote(c) that median values of 4ppb. were taken from Table 6-13 "BKK Landfill Environmental Exposure Characterization Report, West Covina, Calif." CH2MHill, 1988, Volume 1, page6-45. The Table is of the Extended Monitoring Program from August to October. However on page 1-8 of the same CH2MHill Report it states that on July18 levels inside two of the evacuated homes ranged from 5 to 990 ppb. It makes me wonder the value of including the chart without mentioning the levels in the homes prior to the chart date.

Also at the end of paragraph 5 on page x of the executive summary I think it should say,"However it is expected that signifigant emissions from other hazardous chemicals have occurred.

Thank you for considering my comments.

Jean Arneson

Jean Arneson
1116 Donna Beth Ave.
West Covina, Calif. 91791 (818) 919-3304

OEHHA'S RESPONSE TO COMMENTS FROM B.K.K. CORPORATION

Response to Comments From Julia Bussey, Director, Corporate Regulatory Affairs

1 There are no data to show that dilution in the concentration of vinyl chloride occurs with distance from the site, although it is reasonable to assume. B.K.K. Corporation's 1995 average annual concentration has been added in the Risk Characterization, Limitations and Uncertainties.

2. The lower average annual value in 1995 is noted in the Risk Characterization, Assumptions Regarding Ambient Vinyl Chloride Monitoring Data and in the Limitations and Uncertainties.

   The typical landfill gas generation curve has distinct, timed phases. The production of methane gas follows lag-log-steady state-decline curve. The steady state phase begins approximately three years after initial acceptance of waste and continues up to 40 years (Farquar and Rovers, 1973). ARB's report, *Suggested Control Measures for Landfill Gas Emissions, August 1990*, notes that "The gas generation rate depends on several site specific factors including: the amount of moisture present, composition of the waste, temperature, age of the refuse, pH, and quantity and quality of nutrients. Most of California has a dry climate which tends to reduce the amount of moisture in landfills. This slows the gas generation rate, but it also tends to extend the period of time during which a landfill actively generates gas." SCAQMD's review of the TeraMeth Industries, Inc. Landfill Gas to Methanol Project, notes "The BKK Landfill is currently expected to reach its design capacity by 1995. At that time existing operational activities are expected to cease, although landfill gas emissions could continue for an estimated 20 to 40 years into the future" (Letter from Jack Broadbent, Director of Planning, SCAQMD to Mr. Jeffrey Collier, Director of Planning, City of West Covina, dated October 4, 1994).

3. OEHHA has attempted to discuss fully the strengths and limitations of the 1981-1982 data in the addendum report.

Response to Comments From Robert Scofield, D.Env. and Shari Libicki, Ph.D., ENVIRON

1. Comments based on Community Meeting, first paragraph: The second and third paragraphs of the Introduction explain the purpose of the addendum report.

2. Comments based on Community Meeting, second paragraph: The report is organized to provide risk information in three ways, with increasing technical language: the first is the Fact Sheet, the second is the Executive Summary, and the third is the report with Appendices. We believe Finding #4 adequately communicates the added risk.

3. Comments based on Community Meeting, third paragraph: Occupational exposure concentrations are presented in the Toxicity Assessment, History of the Assessment of Vinyl Chloride Toxicity. Risk estimates using unit risk values derived from the epidemiological (occupational) studies are presented in TABLE 10. Appendix H updates tumor incidence information.

4. Comments based on Community Meeting, fourth paragraph: Please see Exposure Assessment, Other Hazardous Chemicals Detected in Ambient Air.

5. Specific Comments on the Report and Fact Sheet, Document Title: While it is technically correct that emission rates *per se* are not actually measured, it has been clearly established that vinyl chloride emissions are associated with the closed Class I Unit of the B.K.K. Landfill.

6. Specific Comments, Page 3, paragraph 1: The B.K.K. Landfill assessment is based on detections of vinyl chloride, a known human carcinogen, in ambient air in a residential area. This chemical is known to be emitted from the landfill. Reliable information on potential past exposures to specific chemicals is generally lacking for other landfill, Superfund or RCRA sites. The ambient vinyl chloride monitoring data collected by SCAQMD and others allows for the quantitative estimation of risk at this site.

7. Specific Comments, Page 44, paragraph 5: Clarification has been added to the Exposure Assessment, Odor Complaints Data.

8. Specific Comments, Page 53, paragraph 2: This comment has been added to the fourth paragraph regarding the 1995-2009 data.

9. Specific Comments, Page 58, TABLE 10: See response to ENVIRON #3.

10. Specific Comments, Page 62, paragraph 1: The phrase has been deleted.

11. Specific Comments, Page 63, Finding 2 and 3: There is no intention to imply that any additional environmental control may or may not be required. We recognize that this would be a risk management decision by the appropriate regulatory agency.

12. Specific Comments, Page 63, Finding 4: We believe that the addendum report adequately states that the risks are associated with detections of vinyl chloride at the three monitoring stations, and that detections of vinyl chloride in other neighborhoods were not as frequent or as high. The discussion on odors complaints data has been clarified.

BKK CORPORATION

2550 237th Street
Torrance, California 90505
(310) 539-7150
Fax (310) 539-3833

February 13, 1996

Ms. Lillian Kelly, M.P.H.
Office of Environmental Health Hazard Assessment
Hazardous Waste Toxicology Section
601 N. 7th Street, MS 241
PO Box 942732
Sacramento, CA 94234-7320

Subject: Comments on Draft Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions from the Class I Unit of the BKK Landfill, West Covina, California

Dear Ms. Kelly:

This letter is to convey our comments on OEHHA's risk assessment. Comments prepared by ENVIRON on behalf of BKK are attached to this letter.

First, please let me express our appreciation for OEHHA's work on the completion of the risk assessment. We believe that OEHHA attempted to objectively and fairly portray the risks at the site. This is very difficult because of the competing concerns and sensitivity regarding the site. While we do not agree with all of the assumptions that were made, we do appreciate the time and effort which were evident in OEHHA's choices.

As noted in the letter from ENVIRON, we are pleased that Dr. Holtzer added some perspective to the public meeting and hope that his comments and information will be included in OEHHA's revisions to the risk assessment.

In addition to ENVIRON's comments, BKK has one technical comment. BKK believes that the risk is overestimated based on the future estimated ambient air concentration assumed in the risk assessment. BKK has three bases for this comment:

1. The current annual average is 0.12 ppbv at the Nogales End station. OEHHA used the estimate of 0.25 ppbv at Myra Court. The estimate is twice as high as the currently measured concentration. In addition, the location of Nogales End is on the BKK property line whereas the Myra Court location is offsite more than one hundred feet from the BKK property line. The experience of BKK in drawing its Proposition 65 isopleth is that air concentrations decrease radically with distance. Data from the ambient air site sampling program suggest that the concentrations drop by one-half every 200 feet from the source. Therefore, the actual concentration at Myra Court must be lower than the Nogales End station.

2. The risk assessment bases the 30-year reasonable maximum exposure and risk in part on 15 years of future exposure to vinyl chloride at 0.25 ppbv (from 1996 to 2009). The exposure due to vinyl chloride is acknowledged in the risk assessment to be derived from the Class I landfill. The Class I landfill's emissions are due to

the degradation of wastes into methane gas which then carries vinyl chloride into the ambient air. It is well established that landfill gas production from degradation reduces over time in a predictable landfill gas decline curve. This is found in many texts by both the EPA and other scientists who study landfills.[1,2] BKK's gas production has thus far followed the predicted landfill gas generation curve as modeled using a first order decay rate of 7% per year of gas emissions. This means that methane gas production and corresponding emissions have been decreasing and will continue to decrease over time. Therefore, a future risk scenario based on a single estimate which does not decrease is an unreasonable overestimation of the risk because it does not take into account the real decreases in future emissions.

3. As noted by Dr. John R. Frolnes in his comments issued in May 1995, the ambient air data from 1981 to 1982 is unreliable. We understand that you have the data and wish to use it. However, both the CH2MHill risk evaluators working for USEPA on an exposure assessment for the BKK Landfill and Dr. Frolnes have considered the use of the data in a quantitative risk assessment inappropriate for many technical reasons. This data represents the majority of the risk estimate for the 30 year reasonable maximum exposure. We would encourage you to reevaluate the scientific merits of use of this data. It contributes to an unreasonable overestimation of risk and increases the scientific uncertainty of the risk estimate.

Thank you again for completing the risk assessment. Our technical staff is available to answer any questions you might have and to provide additional technical information to you, if you would like. Please call me at (310) 539-7150 if you would like more information.

Sincerely,

Julia Bussey

Julia Bussey, Director
Corporate Regulatory Affairs

cc: Steve Samaniego, LEA, City of West Covina
Carmen Santos, US EPA Region 9
Steve Lavinger, DTSC Region 3
Dennis Delaney, SCAQMD
Rick Vergets, RWQCB

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1 EPA-600/8-80-055a LANDFILL AIR EMISSIONS ESTIMATION MODEL, Control Technology Center, December 1980

2 Energy Technology Review No. 64 LANDFILL METHANE RECOVERY Schumacher, Noyes Data Corporation, 1982.

ENVIRON

February 13, 1996

Ms. Lillian Kelly, M.P.H.
Office of Environmental Health Hazard Assessment
Hazardous Waste Toxicology Section, 601 N. 7th Street, MS 241
P.O. Box 942732
Sacramento, CA 94234-7320

Re: Comments on December 1995 Draft Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions from the Class I Unit of the BKK Landfill, West Covina, California

Dear Ms. Kelly:

On behalf of the BKK Corporation, ENVIRON is providing comments on the December 1995 Draft Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions from the Class I Unit of the BKK Landfill, West Covina, California. These comments are based on our review of the report and observations we made during the community meeting held in West Covina on January 11, 1996. We appreciate the opportunity to provide these comments.

We think the OEHHA staff should be commended for doing a very good job of responding to difficult questions from the community at the January 11 meeting. It was our perception that many of the answers directly responded to specific concerns expressed by people who live in the vicinity of the landfill provided by the OEHHA staff. We believe the effectiveness of the OEHHA risk assessment report as a risk communication tool would be enhanced by adding some of the information provided by the OEHHA staff during the community meeting to the report. In addition, we believe the effectiveness of the report as a risk communication tool would be enhanced by clarifying a few points addressed in the report. These comments are discussed in more detail below.

Comments Based on Community Meeting

At least one person attending the meeting noted differences in methods (e.g., exposure assumptions) between the most recent OEHHA report and past risk assessments (e.g., the last OEHHA risk assessment). The reason for these changes were not apparent to him and clearly had diminished his confidence in the conclusions presented in the OEHHA report. Dr. Siegel and Ms. Kelly responded to his question; and we believe the report would be strengthened by an expanded discussion of their response that default

CMA 114446

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assumptions and other aspects of risk assessment methodology vary between regulatory programs and change over time. In addition, BKK has been required to perform several different risk assessments under various regulatory programs (e.g., Proposition 65, AB 2588, RCRA 3008(h)). Some of these other risk assessments are mentioned in the OEHHA risk assessment report, but the relationship between these other risk assessments and the OEHHA report is not clear. It may be worth clarifying in the report that the OEHHA report is being performed in addition to the other risk assessments that BKK must perform to satisfy their regulatory obligations.

As was clear from some of the questions asked at the community meeting, many people want to know if the site poses a real health risk, as opposed to a hypothetical upper-bound risk based on RME and other assumptions. In other words, there is a risk communication need in addition to needing to know how the risks calculated under the varying assumptions of various regulatory programs compare against risk levels that trigger action (e.g., notification, remediation) under specific regulations.

At the community meeting, Dr. Holtzer, from OEHHA, provided some responses that directly addressed many of the community's expressed concerns. Specifically, Dr. Holtzer compared the concentrations measured in the workplaces that were studied in the epidemiology studies of workers exposed to vinyl chloride to the concentrations in the vicinity of the BKK landfill. As mentioned below, we believe it would be useful to present the risk results that are currently provided in the report using epidemiology-based CSFs along with some of the information Dr. Holtzer provided.

Someone from the audience at the community also noted that the most recent risk assessment report only addressed vinyl chloride, while the previous report had addressed several other volatile organic chemicals. Dr. Siegel noted that the other chemicals had been shown to pose de minimis risks. Adding this point to the report would enhance the credibility of the State's analysis.

Specific Comments on the Report and Fact Sheet

Document Title: This title refers to "Ambient Fugitive Vinyl Chloride Emissions". The report should refer to "Ambient Vinyl Chloride Concentrations", as emissions per se, are never mentioned.

Page 3, Paragraph 1 This paragraph cites EPA Region IX as noting that past exposures may be important at selected sites but does not explain what feature of selected sites warrants consideration of past exposures. It does not explain what is unique about past exposures at the BKK landfill that warrants any different treatment than would be given to past exposures at any other landfill, Superfund Site, or RCRA site.

Page 44, Paragraph 5: The discussion of odor complaints notes that the detection of odors suggests the potential for exposure. The discussion does not explain that a large

number of odor complaints came from areas (e.g., to the north of the landfill) where monitoring has consistently failed to detect vinyl chloride. The odor complaints discussion does not attempt to describe the correlation between the location of the odor complaints noted on Figures 16 and 17 and the chemical monitoring results from the same areas. By omitting such an analysis, the report misses an opportunity to communicate that the detection of odor in some areas near the landfill does not necessarily correspond to exposure to vinyl chloride. By restating and confirming the common concern that odor indicates chemical exposure without discussing the specific lack of correlation between the two in the neighborhoods near the BKK landfill the discussion of odor complaints now in the OEHHA report is in fact misleading. Clarification of this point is would help prevent unwarranted concerns.

Page 53. Paragraph 2: Current monitoring data show decreasing concentrations over 1994 data. This should be mentioned.

Page 58. Table 10 The report's value as a risk communication vehicle would be further enhanced by giving greater emphasis to some of the epidemiology information already in the State's report. In particular, the risks calculated using cancer slope factors based on epidemiological data could be given a more prominent presentation. Perhaps, the risks calculated on the basis of the human-based CSF could be more explicitly described as being based on human data, rather than merely being a "Different Unit Risk Assumption" (e.g., in Table 10). We believe that adding a separate section of the report, with its own heading, in which all the epidemiological information is presented would enhance the risk communication aspect of the report. This section could include the points Dr. Holtzer made along with the quantitative estimates of risk based on the human-based CSF that is already presented in the report.

Page 62. Paragraph 1: Text states that the 1993-1994 Proposition 65 data have not been fully validated by a regulatory agency; neither has the agency data. Either both, or neither should be mentioned.

Page 63. Finding 2 and 3 In Finding 2, OEHHA notes that the gas collection system and cap put in place over the closed Class I unit, "have been successful in lowering the frequency and level of vinyl chloride emissions to levels that pose little, if any, risk to nearby residents".

In spite of this finding, Finding 3 is presented and implies that additional environmental control may be required to "ensure future risks do not add significantly to the past cumulative risk". Finding 3 is also presented in spite of the results presented in Table 9 showing that future risks add little to no additional cumulative risk. The only potential significant contribution to future contribution was estimated in the OEHHA report for the MY station. As discussed in the accompanying letter from Julia Bussey and based on 1995 data, projected future concentration of 0.25 ppb at Myra Court is approximately 50%, or more, higher than we would expect.

Page 63, Finding 4 While the report clearly states that the results presented are applicable to people who live in the vicinity of Stations A, B, and MY, these three monitoring stations only represent the exposures of a small fraction of the people who live in the vicinity of the landfill. We believe the risk communication value of the report would be greatly enhanced by a more direct statement regarding the fact that vinyl chloride levels at other locations are substantially lower than at A, B, and MY. A discussion of the lower levels detected at other monitoring stations and the fact that VC was never detected at some stations needs to be added elsewhere to the report to support this statement. We believe a brief summary discussion of monitoring results at other stations is particularly important to address the questions and concerns of the people who live distant from the A,B, and MY stations. These people may get the mistaken impression that the risks at these three stations apply to their location as well. This clarification is particularly important in light of the impression created that odor detection equates to vinyl chloride exposure.

We appreciate the opportunity to have provided these comments and would be happy to discuss them with you.

Sincerely,

Robert Scofield, D.Env.
Principal

Shari Libicki, Ph.D.
Principal

RS:lf
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OEHHA'S RESPONSE TO COMMENTS FROM CHEMICAL MANUFACTURERS ASSOCIATION

Chemical Manufacturers Association submitted three articles for consideration. The "EPA Facts About Vinyl Chloride, June 1992" provides a brief summary of information about vinyl chloride. The other two articles describe an alternate method of deriving a cancer potency factor for vinyl chloride ("Predicting Cancer Risk from Vinyl Chloride Exposure with a Physiologically-Based Pharmacokinetic Model," Reitz *et al.*, 1995, and "Considering Pharmacokinetic and Mechanistic Information in Cancer Risk Assessments for Environmental Contaminants: Examples with Vinyl Chloride and Trichloroethylene", Clewell *et al.*, 1995).

The physiologically-based pharmacokinetic (PBPK) model described by Reitz *et al.* predicts for humans that continuous exposure to 0.869 ppb of vinyl chloride would increase lifetime cancer risk by one in a million. The carcinogenic potency of vinyl chloride estimated using this PBPK model is approximately 137 times less than Cal/EPA's unit risk value used in the addendum report. Assuming the PBPK value for vinyl chloride suggests an estimate of risk at Station A that is approximately 140-fold lower than the estimate obtained in the addendum report. This qualitative estimate has been added to the Risk Characterization, Limitations and Uncertainties.

CHEMICAL MANUFACTURERS ASSOCIATION

February 9, 1996

David M. Siegel, Ph.D., Chief
California Environmental Protection Agency
Office of Environmental Health Hazard Assessment
601 North 7th Street, MS-241
P.O. Box 942732
Sacramento, CA 94234

RECEIVED

FEB 13 1996

Hazardous Waste Toxicology Section

Dear Dr. Siegel:

The CMA Vinyl Chloride Health Committee submits the following reports to aid the California Office of Environmental Health Hazard Assessment in its cancer risk assessment for vinyl chloride using pharmacokinetic and mechanistic:

(1) Common Chemicals Found at Superfund Sites, Office of Emergency and Remedial Response, U.S. EPA, 1994;

(2) Predicting Cancer Risk from Vinyl Chloride Exposure with a Physiologically - Based Pharmacokinetic Model, Reitz et al, Toxicology and Applied Pharmacology (In Press); and,

(3) Considering Pharmacokinetic and Mechanistic Information In Cancer Risk Assessments for Environmental Contaminants: Examples with Vinyl Chloride and Trichloroethylene, Cleweil et al, Chemosphere, 1995.

The Committee looks forward to receiving the final "Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions From the Class I Unit of the BBK Landfill, West Covina, California," when completed and released.

Thank you for your consideration of the enclosed information in preparing a final risk assessment addendum for vinyl chloride.

Please call me at 703-741-5637 if you have any questions or need additional information. My fax number is 703/741-6091.

Sincerely,

Has Shah

Hasmukh C. Shah, Ph.D.
Manager, Vinyl Chloride Panel

cc: Lillian Kelley (with enclosures)
Enclosure

CMA 114452

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9203.1-17
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August 1994

Common Chemicals

Found at Superfund Sites

EPA

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EPA Facts About *Vinyl Chloride*

June 1992

What is vinyl chloride?

Vinyl chloride is a colorless gas with a mild, sweet odor. It is a man-made chemical that does not occur naturally. Most of the vinyl chloride produced in the United States is used to make polyvinyl chloride (PVC). This material is used to manufacture a variety of plastic and vinyl products including pipes, wire and cable coatings, packaging materials, furniture and automobile upholstery, wall coverings, housewares, and automotive parts. Much smaller amounts of vinyl chloride are used as a cooling gas and in the manufacture of other compounds.

Emissions from vinyl chloride and PVC manufacturers are responsible for the majority of vinyl chloride released to the environment.

How might exposure to vinyl chloride occur?

Vinyl chloride has been found in approximately 41S of the 1,300 hazardous waste sites on the *National Priorities List (NPL)*. Vinyl chloride is mainly released into the air and discharged in wastewater from the plastics industry. Most of the vinyl chloride that enters the air gradually breaks down into less harmful substances. Levels of vinyl chloride found in the environment are usually more than a thousand times below levels found in occupational settings. Elevated outdoor levels are usually expressed in terms of parts of vinyl chloride present in a billion parts of air or water (ppb). The term "parts per billion" is a way of expressing the concentration of a contaminant in a liquid or air. One part per billion is equal to one inch in a distance of about sixteen thousand miles (or a penny in ten million dollars), a very small amount. Outdoor levels of vinyl chloride result from the discharge of exhaust gases from factories that manufacture or process vinyl chloride, or evaporation from areas where chemical wastes are stored. The highest outdoor levels have been measured in air near vinyl chloride factories or over chemical waste storage areas.

Vinyl chloride that enters drinking water comes from factories that release vinyl chloride wastes into rivers and lakes, and from *leaching* into groundwater in areas where chemical wastes are stored. Small amounts of vinyl chloride can enter drinking water from contact with polyvinyl chloride pipes. In the past, higher than expected amounts were present in foods packaged in plastic that contained vinyl chloride. Currently, the U.S. Food and Drug Administration (FDA) regulates the amount of vinyl chloride allowed in food packaging in order to limit the intake of vinyl chloride.

How can vinyl chloride affect human health?

Short-term exposures to very high levels of vinyl chloride in air can cause dizziness, lack of muscle coordination, headaches, unconsciousness, or death. Long-term exposure to lower amounts in factories which produce or use vinyl chloride has caused "vinyl chloride disease". This disease is characterized by severe damage to the liver, effects on the lungs, poor circulation in the fingers, changes in the bones of the fingers, thickening of the skin, and changes in the blood. An increased risk of developing cancer of the liver and possibly several other tissues has been linked with breathing air in factories containing vinyl chloride.

Some health effects observed in humans have also been seen in laboratory animals. Effects on the nervous system of animals have occurred following short-term exposure to very high levels of vinyl chloride in air. Animals exposed to high levels for a short period of time, as well as to low levels for a long period, developed liver damage. Kidney effects have also occurred following exposure to high levels. Animals developed cancer in several tissues after eating food or breathing air that contained vinyl chloride.

How can vinyl chloride enter the body?

The most likely way that vinyl chloride can enter the body is by inhalation. This *exposure route* is of concern for persons employed in vinyl chloride manufacturing or processing, for people living in communities where vinyl chloride plants are located, and for individuals living near hazardous waste disposal sites. Vinyl chloride can also enter the body through ingestion. Absorption of vinyl chloride through the skin is not likely to be an important exposure route.

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Is there a medical test to identify vinyl chloride exposure?

Vinyl chloride can be measured in urine and body tissues, but these tests cannot be used to determine the level of vinyl chloride exposure. Measuring the amount of the major *breakdown product* of vinyl chloride in the urine may give some indication of recent exposure; however, the quantity of this breakdown product may vary for different people. Neither of these tests is routinely available at a doctor's office. Laboratory tests commonly used by doctors to evaluate liver damage and liver function are usually not helpful in determining whether liver damage has resulted from vinyl chloride exposure.

What levels of exposure have resulted in harmful health effects?

Vinyl chloride is regarded worldwide as a chemical that causes cancer in humans, but exposure levels necessary to cause cancer are not known. The U.S. Environmental Protection Agency (EPA), therefore, uses animal data to estimate risk in humans. According to this data, it is estimated that breathing air containing 1 ppm vinyl chloride for 70 years may place as many as [illegible] persons in a population of 100,000 at risk of developing cancer. Eating food containing 1 ppm vinyl chloride every day for 70 years may place as many as 6,440 persons in a population of 100,000 at risk of developing cancer. Similarly, drinking water containing 1 ppm vinyl chloride every day for 70 years may place as many as 9,570 persons in a population of 100,000 at risk of developing cancer.

What recommendations has the federal government made to protect human health?

EPA requires that community drinking water systems that regularly serve the same 25 persons for at least 8 months of the year must limit vinyl chloride in the drinking water to 2 ppb. Recently, the FDA changed its regulations regarding the vinyl chloride content of various plastics used in food packaging and to carry water used in food processing in order to limit the intake of vinyl chloride in food to levels considered to be safe. Limits range from 5 to 50 ppm depending on the nature of the plastic and its use.

What are the methods of treatment and disposal of vinyl chloride?

The recommended method of disposal of vinyl chloride is incineration, following mixing with another combustible fuel. Complete combustion must be ensured to prevent the formation of phosgene, a poisonous gas. An acid scrubber is also required to remove hydrochloric acid produced during incineration.

EPA has classified vinyl chloride as a "hazardous component of solid waste" in order to control the handling of this chemical. All releases greater than one pound must be reported to the National Response Center.

GLOSSARY

Breakdown Product: Most contaminants are combinations of specific substances. Contaminants are degraded, or separated, into these individual substances through chemical or physical means.

Exposure Route: The way in which people come into contact with a substance. The main routes are ingestion, inhalation, and absorption through the skin.

Leach: The process by which substances are released from the soil by dissolving in fluids, usually rain and surface water, and are carried down through the soil.

National Priorities List (NPL): EPA's list of uncontrolled or abandoned hazardous waste sites identified for possible long-term clean-up under the Superfund Program.

For more information about Vinyl Chloride, please contact EPA at the following address:

U.S. Environmental Protection Agency
ATTN: Superfund Hotline
401 M Street, S.W.
Washington, D.C. 20460
1-800-424-9346 or 1-800-535-0202

The information contained in this fact sheet was extracted from the Toxicological Profile for Vinyl Chloride, Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, in collaboration with the U.S. Environmental Protection Agency, August, 1989. *This fact sheet focuses on the impacts of hazardous wastes on human health; however, EPA does evaluate their impacts on the environment, including plants and animals.*

CMA 114455

Predicting Cancer Risk from Vinyl Chloride Exposure with a Physiologically-Based Pharmacokinetic Model

November 19, 1995

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Melvin E. Andersen[3]
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ABSTRACT

Predicting Cancer Risk from Vinyl Chloride Exposure with a Physiologically-Based Pharmacokinetic Model. Reitz, R.H., Gargas, M.L., Andersen, M.E., Provan, W.M., and Green, T.L. (1996) *Toxicol. Appl. Pharmacol.*, **000**, 000-000.

A Physiologically-Based Pharmacokinetic (PBPK) model capable of describing the metabolism of vinyl chloride (VC) in rats, mice, and humans has been developed and validated by comparison with experimental data from experiments not used in model development. This PBPK model has been used to predict measures of delivered dose (reactive VC metabolites produced in the livers of the affected species) hypothesized to be involved in the induction of liver angiosarcoma in rats, mice, and human populations exposed to VC. Measures of delivered dose in rats were fit to an empirical dose response model (the linearized multi stage model of Crump et al.) and used to make predictions of liver angiosarcoma incidence in mice and human populations exposed to VC. This procedure gave a good prediction of angiosarcoma incidence in mice. Predictions of angiosarcoma incidence in humans were more than two orders of magnitude lower than risk estimations which did not utilize pharmacokinetic data (HEAST, 1995), but were still almost an order of magnitude higher than actually observed in exposed human populations.

INTRODUCTION

Vinyl chloride (1-chloroethylene, VC) is a colorless, explosive gas. VC is only slightly soluble in water but dissolves readily in fats and organic solvents. VC is most commonly used as a precursor for the production of polyvinylchloride (PVC) plastics, and the highest potential for human exposures exists at the sites where PVC's are manufactured. Because this material has relatively low acute toxicity, occupational exposure standards for VC (OEL) were typically 500 ppm (ECETOC, 1988) until 1970 when Viola discovered that rats exposed to VC vapor developed an increased incidence of tumors (Viola, 1970; Viola et al., 1971). Viola's results were confirmed by Maltoni et al. in 1974, and Maltoni also reported that a rare form of liver cancer (angiosarcoma) was induced in rats by VC (Maltoni et al., 1974).

In that same year Creech & Johnson (1974) reported that a search of the medical files of employees exposed to VC at a Goodrich plant in the USA revealed three cases of death from the same rare type of liver cancer (angiosarcoma). Since that time, VC has been the subject of numerous animal studies and epidemiological surveys, and it is clear that VC induces angiosarcomas of the liver in both animals and humans (see ECETOC, 1988 for a review). Other types of tumors (non-liver) have been associated with VC exposure in animals, but the epidemiological data

have not linked exposure to VC to induction of other types of tumors in humans (ECETOC, 1988).

VC is metabolically activated to a reactive species (probably chloroethylene oxide) which is capable of binding to DNA and causing genotoxicity in vivo (ECETOC, 1988). This activation is catalyzed by Cytochrome P450 enzymes, and there is good evidence that the metabolism of VC is saturable in vivo and in vitro (Kappus et al., 1976; Gehring et al., 1978; Guengerich & Watanabe, 1979). A large body of data relating the tumorigenic response in the livers of animals and humans to biochemical events taking place in the various species is available. The purpose of this paper is to discuss methods for using this database to prepare estimates of cancer risk for human populations exposed to VC.

VC is worthy of consideration for another reason. In most cases where estimations of the human cancer risk have been based on animal studies, it is not possible to know whether the projections of risk are realistic or not (epidemiological data are not precise enough to either confirm or deny the risk projections). In the case a rather large body of epidemiological data indicates that significant increases in human cancer have occurred as a result of past practices which resulted in high human exposures to VC. This provides a unique opportunity to test the ability of current risk assessment practices to provide reliable estimates of human cancer risk from animal data.

Objectives:

Physiologically-based pharmacokinetic (PBPK) models of chemical disposition have been developed for a variety of chemicals, including the chlorinated ethylenes (NAS, 1987). These models are particularly well suited for risk extrapolations because they are based on specific physiological and biochemical properties of the different species and dose routes as well as physical chemical information about the solubilities and vapor pressures of the different compounds (Andersen et al., 1987). Our objectives in this project were:

1. To develop a PBPK model capable of predicting the metabolism of VC in both rodents and humans.
2. To validate this model with existing data sets for rodents and humans.
3. To develop a quantitative risk assessment procedure based on the predictions of the validated PBPK model for VC.
4. To compare the PBPK based risk assessment procedure with the existing EPA risk assessment procedure (HEAST, 1995)
5. And finally, to compare the results from this PBPK based risk assessment with the actual incidence of liver angiosarcomas in human populations exposed to VC in the workplace. (Simonato et al., 1991)

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METHODS

Construction of the PBPK Model:

The PBPK model for VC was based on a PBPK model developed by Ramsey & Andersen (1984) to describe the kinetics of inhaled styrene in rats and humans. In this model a series of simultaneous differential equations describing the distribution, elimination, and metabolism of chemical was incorporated into a computer program using an integrated software package containing routines for numerical integration, optimization, sensitivity analysis, and graphical display. This software package (SimuSolv[5]) is commercially available from Mitchell & Gauthier Associates, 200 Baker Ave, Concord MA 01742-0013, USA.

The VC model contains four tissue groups (fat, muscle, rapidly perfused tissues, and liver) and assumes that all metabolism takes place in the liver where the rate of metabolism is described by the Michaelis-Menten equation. Detailed descriptions of this type of model are given elsewhere (Ramsey and Andersen, 1984; Andersen et al., 1987). An annotated copy of the source code for this model is available from the corresponding author (Reitz)."

Physiological parameters in the model (blood flows, ventilation rates, organ sizes) appropriate for rats, mice, and humans were identical to those used by Andersen et al., (1987) in a multispecies PBPK model for methylene chloride with two changes: (1) the size of the liver compartment for rodents was based on historical data for control animals from the Toxicology Laboratory of the Dow Chemical Company and (2) the allometric constants for alveolar ventilation and cardiac flow in rats used by Andersen et al. (1987) were increased from 15 to 18 in order to provide a more consistent description of the gas uptake data sets.

Blood/air partition coefficients for rat, mouse, and humans and tissue/air partition coefficients for rat liver, rat muscle, and rat fat were determined using the vial equilibration method of Sato and Nakajima (1979) as modified by Gargas et al. (1989). Tissue/blood partition coefficients for rats were obtained by dividing the tissue/air partition coefficients by the blood/air partition coefficient. No direct measurements were available for the tissue/air partition coefficients in the rapidly perfused group of tissues in this model, so this partition coefficient was set equal to the partition coefficient for liver, a technique that has proven successful in the development of PBPK models for other halogenated, volatile materials (Andersen et al., 1987; Reitz et al., 1988; Reitz et al., 1990a,b). Tissue/blood partition coefficients for mice and humans were estimated by dividing the tissue/air partition coefficients for rats by the blood/air partition coefficients for mice or humans respectively. All of the partition coefficients used in the PBPK model for VC are listed in Table 1.

Metabolic Constants for Rats: Metabolic parameters for male and female Sprague Dawley rats (body weights 200-400 grams) were obtained by computer optimization of gas uptake data sets (four experiments for each sex) according to procedures previously described by Gargas et al. (1986). Basically, given the physiology of the animals and the solubilities of VC in rat blood and tissues, the metabolic rate constants VMaxC and Km were varied until a satisfactory description of data gathered in several independent gas uptake experiments was obtained for both male and female rats (Figures 1a, 1b).

Metabolic Constants for Mice and Humans: Vinyl chloride belongs to a large class of low molecular compounds (including -benzene, styrene, CCl_4, $CHCL_3$, CH_2Cl_2, CH_3Cl, CH_3CCl_3, 1-2 dichloropropane, ethylene dichloride, ethylene dibromide, vinyl bromide, acrylonitrile, vinyl carbamate, ethyl carbamate, and trichloroethylene) which are readily metabolized by Cytochrome P-450, IIE1 (P450 2E1). Guengerich et al. (1991) observed that metabolism of these substrates by microsomal preparations from liver (a) showed the same relative activity for different preparations of microsomes with all the substrates, (b) was sensitive to inhibition by known inhibitors of P450 2E1 metabolism such as diethyldithiocarbamate and disulfiram with all the substrates, and (c) showed inhibition by specific antibodies to P450 2E1 raised in rabbits, but was not sensitive to addition of antibodies specific for other forms of P450 such as P450 IIIA or $P450_{MP}$. Guengerich also reported that purified preparations of P450 2E1 were also active on all these substrates.

Based on these and other studies, oxidation of this broad group of substrates by P450 2E1 has been identified as the primary means of biotransformation in both animals and man (Nakajima et al., 1990; Guengerich et al., 1991; Raucy et al., 1993). Consequently, the extensive in vivo and in vitro studies carried out with CH_2Cl_2 (Andersen et al., 1987; 1991) and $CHCl_3$ (Corley et al., 1990; Reitz et al., 1990a) provide a reliable basis for estimating in vivo metabolic rates for other members of this class (including VC) in humans. The process by which these data were used to estimate in vivo metabolic rate constants for humans and mice for VC is outlined below:

1. In vivo maximum rates of metabolism (VMax's) in rats are obtained by experimentation (VC) or from the literature (CH_2Cl_2 $CHCl_3$). These values are listed in Table 2.
2. The weight of the liver in animals used in the in vivo studies is calculated for each chemical and each species from the percent liver and the body weight (Table 2).
3. The VMax for each species is divided by the weight of liver to give an in vivo rate per gram for each chemical. These VMax/gs are normalized to the rat for each chemical, and the "Ratio to Rat" is listed in the last row of Table 2 for each chemical.
4. For chloroform and methylene chloride, the in vivo "Ratio to Rat" is nearly constant (2.570, 2.707) suggesting that after normalization, the "Ratio to Rat" does not depend upon the chemical being studied (at least within this limited series of chemicals all metabolized by P450 2E1).

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5 In vivo studies by Andersen et al (1991) with CH_2Cl_2 provide a basis for calculating the "Ratio to Rat" for humans of 0.208: Table 2. (Comparable in vivo studies for $CHCl_3$ in humans were not available.)

6 Finally, the experimentally determined in vivo VMax for VC in rats and the "Ratio to Rat" for mice and humans were used to calculate in vivo VMax's for VC by multiplying VMax/g (rat) by the "Ratio to Rat" and weight of liver in each species.

For example the in vivo VMax for VC in humans is calculated as: (0.968/5.69) x 0.208 x 2198 = 77.7 mg/hr (Table 2).

RESULTS

Model Validation

Validation in Rats

As noted in the Methods section, metabolic rate constants were obtained from in vivo gas uptake experiments previously at Wright Patterson AFB (Gargas et al., 1990). To verify that the model using these metabolic rate constants was broadly descriptive of metabolism in the rats, independent experiments performed by Watanabe et al. (1976) were evaluated. Watanabe and coworkers exposed rats to a series of concentrations of radiolabeled VC for six hours, and then collected radioactive excreta from these animals for up to 72 hrs. These studies allowed the estimation of the total amounts of VC metabolized by the rats (Watanabe et al., 1976; Gehring et al. (1978). The amounts of radioactive metabolites observed by Watanabe et al. were compared to the predictions of the PBPK model for rats. Other than changing the body weights and exposure concentrations to reflect the different experimental conditions, no changes were made in the model developed from gas uptake experiments. The results are shown in Figure 2.

The model gave an excellent simulation of Watanabe et al.'s data. Over a range of concentrations from 1.4 ppm up to 4,600 ppm, the PBPK model accurately predicted the levels of radioactive metabolites produced and successfully identified the region where saturation of VC metabolism occurs (200-500 ppm: Figure 2).

Validation in Mice

Metabolic rate constants for $B_6C_3F_1$ mice were estimated by extrapolation from in vivo results in $B_6C_3F_1$ mice obtained with model substrates as outlined in the Methods section. In order to test whether these estimated rate constants accurately reflected the in vivo metabolism of VC in mice, an independent set of gas uptake experiments conducted in male and female $B_6C_3F_1$ mice was used for validation.

For this validation, the basic PBPK model for rats was adapted to mice by (a) incorporating the known physiological differences between rats and mice (see

Andersen et al., 1987), (b) changing the blood/air partition coefficient for VC to that measured in the laboratory with samples of $B_6C_3F_1$ mouse blood (Gargas et al., 1989), and (c) setting the allometric constant describing the maximum rate of metabolic oxidation of VC to the value estimated from in vivo studies with other volatile, low molecular weight, halogenated hydrocarbons (Table 2). Other than these changes, the structure of the model was not altered.

Simulated and actual data from four experiments with male $B_6C_3F_1$ mice (initial concentrations of 345, 570, 1065, and 3190 ppm) and four experiments with female $B_6C_3F_1$ mice (initial concentrations of 280, 550, 975, and 2950 ppm) are shown in Figures 3a and 3b. As can be seen from inspection of these figures, the model gives a reasonable (but not perfect) simulation of the gas uptake data.

The highest concentrations of VC (~3000 ppm where metabolism is probably saturated) were well described by the PBPK based on the metabolic rate constants estimated from model substrates. These data depend primarily upon the value chosen for VMax in the model and suggest that the extrapolation procedure employed for estimating the maximum in vivo rate of metabolism in $B_6C_3F_1$ mice was successful.

The low concentrations (~300 ppm, where metabolism is presumably first order) are also well described by the model. For rapidly metabolized substances, the uptake is largely flow-limited (i.e. depends upon how rapidly the material is delivered to the liver rather than the specific values of VMax and Km). Correspondence of model simulations with experimental data at low concentrations suggests that the physiological parameters for mice (flow rates and partition coefficients) used in the PBPK models for $B_6C_3F_1$ mice are appropriate.

However, for the experiments involving intermediate concentrations of VC (550-1065 ppm) the model predicts slightly more metabolism (uptake from the chamber) than was experimentally observed. These results were obtained in the region where metabolism of VC changes from flow-limited conditions to zero order (enzyme saturation), and the simulation of these concentrations is sensitive to the value chosen for Km in the Michaelis-Menten equation.

To explore the possibility that a different value of Km might give a better simulation of the experimental results, a computer optimization was conducted with SimuSolv®. The results of this optimization (in which the computer varied both VMaxC and Km) are shown in Figure 3c. In this optimization, it was found that a Km of 0.28 gave a much better simulation of the gas uptake data than the Km obtained from the rat experiments (Km = 0.04 in rats). However, the optimum value for VMaxC remained relatively constant (the optimum value of VMaxC was 8.13; quite close to the extrapolated value of 9.04).

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Given that the same enzyme (P450–2E1) is responsible for oxidation of VC in both species, it may seem surprising that the apparent Km's differ by this amount. However, based on studies of deuterium isotope effects on CH_2Cl_2 metabolism, Andersen et al. (1994) have suggested that the apparent Km's for oxidation of P450–2E1 substrates likely reflect a more complicated series of events than simple "binding" of a substrate to an enzyme's active site. Andersen speculated that the apparent Km's for P450 2E1 substrates may be "... a measure of reactivity of activated oxygen species with available C–H bonds". If this hypothesis proves to be correct, then the Km for VC oxidation in humans would be expected to be more like the Km of rats than the Km of mice, since the mouse liver has an usually high capacity for oxidation of these substrates (Andersen et al., 1987; Corley et al., 1990).

The preceding validation exercise indicates that the procedure used to estimate in vivo metabolic rate constants for VC should give accurate representations of human VC metabolism at either high or low concentrations of VC. The extrapolation procedure would be less precise in identifying the region where transition from first order to zero order kinetics with VC occurs when extrapolating to species other than the rat.

Validation in Humans

We also attempted to locate independently gathered human data for validation of the human PBPK model for VC. Validation of human models for other solvents (CH_2Cl_2, 1,1,1-trichloroethane, styrene) has been previously reported (Andersen et al., 1991; Reitz et al., 1988; Ramsey and Andersen, 1984), providing support for the techniques for estimating partition coefficients and physiological constants in humans. Consequently, the primary emphasis was on evaluation of the metabolic rate constants for VC estimated by the techniques outlined in the Methods section. No attempt was made to "curve fit" experimental data by adjustment of model parameters in validating the human PBPK model.

Ideally, we hoped to find measurements of the rate of production of VC-specific metabolites in human subjects (e.g. excretion of VC-specific metabolites in the urine of humans exposed to VC similar to data gathered in humans exposed to trichloroethylene: Müller et al., 1974) Unfortunately, we were unable to locate this type of dataset for VC. However, we did locate a dataset in which exhaled air concentrations of VC were reported for human volunteers following VC exposure and this dataset was evaluated with the human PBPK model.

In these studies, Baretta et al. (1969) exposed groups of human volunteers to 59, 261, or 492 ppm VC for 7.5 hrs in a carefully controlled laboratory setting. Workers entered the chamber and were exposed to VC at the indicated concentrations for approximately 3.5 hrs. Then they left the chamber to have lunch in an area free of

VC for approximately 0.5 hrs. following which they returned to the chamber for another 4 hrs (7.5 hrs exposure to VC). This exposure pattern was simulated by the PBPK model during the validation exercises.

Samples of exhaled breath were collected from these volunteers (4-7 subjects in each exposure) at different times post-exposure for up to 20 hrs. The sampling procedure involved giving each person several glass tubes (20 mm diameter, approximately 23 cm in length) capped with screw cap septa. Each worker was asked to inhale through his nose and exhale by mouth into the glass tube four times. After the fourth breath, the workers quickly capped the glass tubes with impermeable septa and returned the tubes for analysis. A comparison of observed and simulated results for these exposures is presented in Figure 4. The model for VC gave an good simulation of expired air data for all three concentrations over the period from 1 hr post exposure to 20 hr post exposure. It is noteworthy that these experiments included exposures at concentrations up 500 ppm, a region where metabolic saturation occurs in rats (Figure 2).

A sensitivity analysis was conducted to determine whether this fit was dependent upon selection of the proper values for the metabolic rate parameters (Vmax and Km) or whether other model parameters were more influential in the prediction of expired air concentrations of VC (CEX) as described elsewhere (Reitz et al., 1990b). Predicted values of CEX one hour and ten hours post exposure were compared to the same values predicted with "baseline" values for all model parameters with VC concentrations of 59 ppm and 492 ppm (the lowest and highest VC concentrations studied by Baretta et al., 1969).

The sensitivity analyses revealed that model parameters associated with flow rates (fraction cardiac output directed to fat compartment, alveolar ventilation and cardiac output) and partitioning within the body (blood/air, fat/air, and, at the 1 hr post exposure period, muscle/air) had 3-30 fold more influence on the predicted values of CEX than the metabolic rate parameters Vmax and Km. In fact, doubling or halving the values of Vmax or Km did not appreciably change the fit to the data collected by Baretta et al. (simulations not shown).

Buchter et al. (1978) also studied the pharmacokinetics of VC in human subjects. Human volunteers inhaled VC vapor from (and exhaled back to) a closed system containing 10 ppm and the removal of VC from this system for periods up to 30 minutes was evaluated. In other studies, volunteers inhaled a constant concentration of VC (~2.5 ppm) for 15-30 minutes. Data reported by Buchter et al. were also well simulated by the PBPK model (simulations not shown), but a sensitivity analysis revealed that simulations of these data were even less sensitive to the values chosen for Vmax and Km than the data of Baretta et al., (1969).

It is reassuring that the limited human data for VC are consistent with the PBPK model developed here. However, it must be conceded that the validation procedures described above neither support nor refute our procedure for estimating VC metabolic rate constants in humans. Unfortunately, since VC is a known human carcinogen, it is considered unlikely that definitive studies of VC metabolism capable of rigorously establishing metabolic rate constants for the human PBPK model will be conducted in the foreseeable future.

Risk Estimation

The carcinogenicity of VC in animals has been studied extensively (in fact it may be the most extensively studied of any of the animal carcinogens known). Exposure paradigms include single exposures, high exposures for short periods (days or weeks), exposures early in the natural lifespan versus late in the natural lifespan, etc. Given the wealth of data available for preparation of risk estimations, we were forced to select a subset of the data for illustrative purposes. We have chosen to focus on studies in which VC was administered for a substantial fraction of the animal's lifetime (12 months exposure in each of the cases evaulated) with follow-up until the animals' death wherever possible. The reader is referred to the publications of Maltoni, Drew, and Lee for further details of these and other studies.

We are aware that the pattern of exposure (i.e., whether a given exposure occurs early or late in life) may influence the carcinogenic potency of VC, but we have not attempted to consider this factor in our analyses. Similarly, we are aware that the life expectancy of mice (but not rats) exposed to VC in the first 12 months of their life is significantly shorter than the life span of control mice (c.f., Drew et al., 1983 who reported a mean survival time of 780 days for control female $B_6C_3F_1$ mice compared to 301 days in the group exposed to 50 ppm VC for 6 hr/day). However, we have not attempted to apply any "less than lifetime" correction factors to the cancer potencies obtained from the mouse studies.

Deriving Rat Potency Estimates

Maltoni conducted a series of inhalation bioassays of VC in male and female Sprague Dawley rats at concentrations ranging from 1 ppm to 30,000 ppm (Maltoni, 1974). These animals were exposed to VC for 4 hr/day, 5 days/week, with exposures beginning in young adult animals and continuing until the animals reached one year of age. After the first year of exposure, animals were held until they died and then examined for the presence of tumors. Survival of the animals was compromised at the highest concentrations, so these results were not employed in derivation of a rat potency for VC. Results from exposures conducted at 0, 1, 5, 10, 25, 50, 100, 150, 200, 250, 500, 2500, and 6000 ppm were selected as the basis for fitting a dose response curve for induction of liver angiosarcoma by VC metabolites. Tumor incidence data used in constructing this curve are listed in Table 3.

The dose surrogate (measure of dose delivered to the target organ) chosen for risk analysis was the average daily amount of metabolite produced per day per liter of liver tissue. This type of dose surrogate is appropriate for risk analysis when the metabolite is highly reactive (as the chloroethylene oxide formed from VC would be) and either reacts with DNA or water in the target organ with a very short half-life (i.e. would not be expected to persist long enough to circulate to other organs in the body). Rationale for the selection of dose surrogates from PBPK models have been discussed extensively elsewhere (Andersen et al., 1987) and the reader is referred to this publication for further details.

The PBPK model for VC in rats was then used to calculate the amount of metabolites produced during a typical day of exposure (4 hr exposure to the selected VC concentration). Maltoni exposed rats to VC for 5 days/week and for 1 year, so the lifetime average daily doses (LADD) were calculated by multiplying the values obtained from the computer by 5/7 (to correct for less than daily exposure) and 1/2 (to correct for less than lifetime exposure). Results from male and female rats were combined and empirically fitted to a metabolic dose/tumorigenic response curve with the computer program GLOBAL83 (Howe and Crump, 1982; Howe, 1983). The predicted (maximum likelihood estimate) and observed results are depicted graphically in Figure 5.

The risk estimation based on the PBPK model (curved, heavy line in Fig 5) describes the tumor incidences observed by Maltoni over a broad range of doses, showing the ability of the PBPK model to compensate for the effects of metabolic saturation in the activation of VC. For illustrative purposes, a hypothetical risk estimation based on administered dose (ppm VC) at the two highest concentrations (2500, 6000 ppm) is also shown with a dotted line in Figure 5. This represents the type of risk estimation that EPA might have conducted if the VC data were from a "typical" bioassay (i.e. the only tumor incidences reported were for MTD and MTD/2), and it is noteworthy that such a procedure would have significantly UNDERPREDICTED the tumor incidences seen in rats by Maltoni at lower concentrations (e.g. 10–100 ppm). This line does not correspond to the actual EPA risk assessment for VC (HEAST, 1995)

For the purposes of illustration in this paper, the dose-response model relating tumor incidence in rats and levels of VC metabolites in liver was obtained from the GLOBAL83 computer program. Other mathematical models relating tumor incidence to doses of carcinogenic species have been developed and could certainly have been employed in addition to or instead of the multi-stage model. However, since the extrapolation range evaluated was relatively small, it was not considered necessary to explore these other models (they all give basically the same results when used for regions where experimental data is available as is the case here).

Extrapolation from the fitted dose/response curve in rats indicates that a LADD of 0.177 mg equivalents of VC metabolites/day/liter of liver is associated with a lifetime increase of 1×10^{-4} in the cancer incidence of rats (MLE estimate). This Risk Specific Dose (RSD) may now be used to estimate the excess risk of cancer in mice and humans exposed to VC under the assumption that equal average concentrations of VC metabolites in the liver of these species produce equal lifetime risks of cancer.

This assumption is precisely the same as that used by most U.S. regulatory agencies when performing risk assessments based on administered LADD (e.g. doses in mg/kg/day) except that an interspecies scaling factor related to body surface area is also employed by those agencies. Andersen et al. (1987) suggested that since the PBPK model already contains provisions for considering metabolic and physiological differences between species, the body surface area factor should be eliminated from risk assessments based on PBPK models. As will be seen later, comparisons of predicted and observed incidences of angiosarcoma in humans exposed to VC are consistent with the proposal of Andersen et al (1987).

Estimation of Risk to Mice from Rat Data

Maltoni also reported the effects of exposure to VC on the incidence of angiosarcomas in Swiss albino mice exposed to VC 4 hr/day, 5 days/week for 30 weeks with the experiment terminated at 81 weeks (Maltoni's experiment BT 4 summarized in ECETOC, 1988). This experiment contained exposure groups of 0, 50, 250, 500, 2500, 6000, and 10000 ppm VC with approximately 30 male or female animals/group (approximately 60 total mice/group) except for the control group which contained 150 male and female mice.

VC was seen to increase the incidence of angiosarcoma of the liver (and angiosarcoma at other sites) in exposed mice in a dose-related fashion (Table 4). As with the rats, the tumor response at very high concentrations of VC reached a plateau and then declined. It is presumed that the decrease in tumor incidence is related to the fact that the animals in the highest dose groups showed very poor survival.

Lifetime average daily dose surrogate measures (LADD) were calculated for the mice and these doses and the tumor incidences were subjected to processing by GLOBAL83 to determine the parameters for the LMS, with the MLE and extra risk options selected. When this was done, the RSD (MLE estimate of dose associated with a lifetime increase in risk of 1×10^{-4} to mice) was found to be 0.0797 mg equivalents of metabolite per liter of liver per day, in fairly good agreement with the RSD previously calculated for rats (0.177 mg equivalents/liter liver/day).

Two other studies of the effect of VC exposure on development of liver angiosarcoma have been reported. Lee et al. (1978) exposed CD-1 mice to VC for 6

hr/day, 5 days/week for 12 months, at which time the experiment was terminated (no holding period after exposure). Lee reported combined incidences of hepatic angiosarcoma of 0%, 4.8%, 36.5%, and 44.9% after exposure to 0, 50, 250, and 1000 ppm of VC respectively. When subjected to GLOBAL83 calculations, the RSD associated with a lifetime increase in risk of 1×10^{-4} to mice (based on Lee et al.'s studies) was found to be 0.120 mg equivalents of metabolite per liter of liver per day, intermediate in potency between the RSD previously calculated for rats (0.177 mg equivalents/liter liver/day) and the RSD based on Maltoni's experiments in Swiss albino mice (0.0797 mg equivalents of metabolite per liter of liver per day). Thus these two mouse studies and the rat study gave quite consistent estimates of the RSD for VC metabolites.

However, when a bioassay of VC in $B_6C_3F_1$ and Swiss CD-1 female mice (and F344 rats and Syrian Golden Hamsters) conducted by the National Toxicology Program (NTP; Drew et al., 1983) was evaluated, quite different results were seen. In these studies, only one exposure concentration was studied (50 ppm, 6 hr/day, 5 days/week), but exposures were begun at different points in the animals life spans and were conducted for different durations (6 months, 12 months, etc.). One of the exposed groups of mice was subjected to an exposure paradigm similar to that employed by Maltoni et al. (1974) in that exposure began when the animals were 9 weeks old, they were exposed for almost 12 months. Drew et al. reported that none of the treated animals survived more than 1 year, and the cause of death in the treated animals was frequently considered to be "... due to the development of neoplasms...".

In this group of female $B_6C_3F_1$ mice VC increased the incidence of angiosarcoma in the NTP study from 5.8% in controls (4/69 animals) to 76.7% (69/90 animals). In contrast, at this exposure concentration and paradigm, Maltoni et al. (1974) and Lee (1978) observed a 2-5% incidence of angiosarcomas in treated mice with no angiosarcomas seen in control animals. Thus the NTP has reported a considerably higher incidence of angiosarcomas in both control and treated animals than either Lee or Maltoni and this difference is not due to differences in survival, since the treated mice studied by Lee and Maltoni all survived longer than in the NTP study.

The RSD for VC in $B_6C_3F_1$ mice (calculated in the same manner as the RSD's for Maltoni's and Lee's studies) was 0.0032 mg equivalents of VC metabolites/day/liter of liver. This RSD is significantly lower (25-55 fold, implying more risk associated with a fixed concentration of VC) than the RSD's calculated from the Lee and Maltoni studies.

The discrepancy in the carcinogenic potency of VC appears to be laboratory rather than strain specific, because NTP also studied another strain of mouse (CD-1 mice) and again observed a much higher incidence of angiosarcomas in the liver (63.8% in treated versus 1.4% in controls) than seen by either Maltoni or Lee. It is noteworthy

that the incidence of liver angiosarcomas reported by NTP (Drew et al., 1983) in rats exposed to 100 ppm VC (20%) was also significantly higher than reported by Maltoni (1-2% incidence).

Estimation of Human Risk

Risk estimates for humans occupationally exposed to VC were prepared by the following procedure:

1. The validated PBPK model for humans was used to construct a table of lifetime average daily doses (LADD) expressed in the same terms as used in the rat model: mg VC metabolites formed/day/liter of liver tissue for conditions thought likely to have been present in the workplace in past years (i.e. TWA's of 50 - 2,000 ppm and employment for 10 - 20 years).

   In performing these calculations, mg equivalents of metabolites were adjusted for the fraction of the day that workers were exposed (8/24), the days/week that the workers were at their jobs (5/7), and the fraction of a lifetime that exposure took place (years/70).

2. Once these estimates of dose had been prepared, the GLOBAL83 program was used to estimate the likelihood that tumors would be produced, based on the potency number derived from rats (RSD = 0.177).

The results of these estimations are presented in Table 5. The predictions range from about two hundred cases per 100,000 (for workers employed 10 years at a plant where the TWA was 50 ppm) to almost 4,000 cancers per 100,000 in workers employed for 20 years in a plant where TWA's were 2,000 ppm.

The predictions of human risk may be compared with results reported by Simonato et al. (1991) based on the world-wide vinyl chloride tumor registry. Simonato's results are based on the evaluation of 12,706 individuals selected from a population of 14,351 subjects from 19 factories where VC was used industrially. The completeness of followup in this study was stated by the authors to be 97.7%, and the average length of followup was 17 years (with 36% of the population followed for >25 years). The total number of person years at risk in this study was 222,746.

Simonato et al. (1991) reported a clear association between both duration of employment and ranked level of exposure. They estimated the absolute risk of angiosarcoma in exposed population with >25 years since first employment to be between 0.2 cases/100,000 for exposures less than 2,000 ppm.years to 280 cases/100,000 for individuals with more than 10,000 ppm.years. Relative risks in highly exposed populations were as high as 45.4:1. Absolute risks observed in the VC cohort with >25 years since first employment are listed in Table 5 for comparison with risks predicted by the linearized multistage model (LMS) using maximum likelihood estimates (MLE) from the PBPK model.

In each case, the estimates from the procedure employing the PBPK model are substantially higher than actually observed in humans. For example, based on the PBPK prediction, workers exposed to 200 ppm TWA VC for 20 years (4,000 ppm years) would be expected to develop 1,465 cases of angiosarcoma/100,000. However, the incidence of angiosarcomas observed in the group with >25 years since first employment and 2,000-5,999 estimated ppm.years was reported by Simonato et al. to be 42.2, which is about 35 fold lower than the PBPK prediction. At higher levels of human exposure (e.g. 6,000-9,999 ppm.years, > 10,000 ppm.years) the PBPK predictions are higher than the observed rates by a factor of ten or so (Table 5).

Potency factors derived from the studies of Drew,et al. (1983) were also used to predict human risk (data not shown). When these potency factors were used, the discrepancy between predicted risk and observed incidence was much greater. Predictions based on the Drew studies were almost three orders of magnitude (1,000 fold) higher than the actual (observed) incidences of liver angiosarcoma in exposed workers, so these studies are clearly less consistent with human experience than the studies conducted by Maltoni and Lee.

It is noteworthy that we did not employ the "body surface area factor" employed by the U. S. Environmental Protection Agency for cancer risk extrapolation between rats and humans in our risk estimations. If the surface area factor had been employed, the predicted risks would have increased by a factor of approximately 5-6 fold for rat to human extrapolations (or 12-13 fold for mouse to human). Inclusion of the surface area factor in the PBPK based risk estimation for VC would clearly have made the risk estimations we produced less consistent with the reported incidences of angiosarcomas in the exposed workers.

DISCUSSION

A multispecies PBPK model capable of quantitatively describing the metabolic activation of inhaled VC was developed from pharmacokinetic principles and then validated with independent data sets for rats, mice, and humans. Only minor modifications of the model described by Ramsey and Andersen (1984) for inhaled styrene were necessary to accomplish this.

VC is metabolized in mammals by the cytochrome P450 enzymes (likely by the 2E1 subclass) to produce reactive, short-lived intermediates (chloroethylene oxide). These reactive intermediates alkylate DNA and are genotoxic (mutagenic) to both bacterial and mammalian cells. The reactive metabolites of VC are generally assumed to be responsible for the induction of angiosarcomas and other tumors in mammals exposed to VC. Since these intermediates are too short-lived (reactive) to circulate in the blood stream very far from the organ of their formation, the carcinogenic effects of VC on a particular organ system is assumed to be related to

the rates of metabolic activation occurring in that organ. We estimated the rates of induction of liver cancer in various species under different exposure regimens by using the PBPK model to predict the rates of metabolism in the liver of the treated animals.

Tumor Induction in Rats

The results presented in Figure 5 indicate that accurate predictions of the risk of developing angiosarcoma must consider the dose dependency of VC metabolism. When risk assessments are based on the concentration of VC inhaled by the animals (instead of the amount of reactive metabolite formed by the animals) the predictions deviate widely from incidences observed by Maltoni and others.

For example, if a risk assessment were prepared from a cancer study which contained only high doses of VC (i.e. doses where metabolic activation was saturated), extrapolation to low doses would significantly underestimate the incidence of tumors in rats (shown by the dotted line in Figure 5). On the other hand, if risk estimations were based doses of VC below the level of metabolic saturation, linear extrapolation to high doses would greatly overestimate the incidence of tumors in rats exposed to high concentrations of VC. As Gehring et al. (1979) pointed out, basing risk estimation on VC metabolites instead of VC itself produces a consistent estimate of carcinogenic potency across the entire dose range (Figure 5).

Tumor Induction in Mice

In addition to increasing the reliability of high dose/low dose extrapolations, PBPK models provide an scientific basis for extrapolations between different species, considering physiological as well as metabolic differences. Since VC has been extensively studied in the mouse as well as the rat, this provided an opportunity to test the ability of the PBPK model to accomplish interspecies extrapolations.

Mice are generally known to contain higher levels of the cytochrome P450 enzymes than either rats or humans and this is reflected by the higher rates of in vivo metabolism seen in mice versus rats for the substrates listed in Table 2 (CH_2Cl_2, $CHCl_3$, VC). Based on this knowledge, mice would be expected to be more sensitive to the tumorigenic effects of exposure to a given concentration of VC than rats, and this has been widely verified (Maltoni et al., 1974; Lee et al., 1978; Drew et al., 1983; ECETOC, 1988). The RSD's (1×10^{-4} lifetime risk) calculated for the different species/strains of mice were:

Maltoni (rat potency)	0.177
Maltoni (Swiss mouse potency)	0.0797
Lee (CD-1 mice)	0.120
Drew ($B_6C_3F_1$ mice)	0.0032

It is noteworthy that the duration of the mouse experiments differed from those of the rat: Maltoni's experiments in rats were "entire lifespan" (generally > 100 weeks), while Maltoni's experiments in mice were terminated at 81 weeks of age. In the case of mouse studies conducted by Lee et al. and Drew et al., all animals had either been sacrified or had died by 52 weeks of age (Lee et al., 1978; Drew et al., 1983). Thus it might be argued that if the mouse experiments had been of longer duration, higher cancer potencies for mice might have been calculated (or that application of a "less than lifetime" correction factor was warranted, reducing the similiarity of the RSD values in rats and mice).

However, since the authors of at least one study (Drew et al.) attributed the early mortality to "... induction of neoplasms <in the treated animals> ...", it is not clear that application of a factor intended to correct for loss of animals from non-neoplastic causes before they had a chance to develop cancer would be appropriate. In any case, the incidence of angiosarcoma was very high in the Drew et al. and Lee et al. studies (77% and 45% respectively), so holding the animals for another year could not have increased the tumor incidences by more than a factor of two. The mouse studies of Maltoni, by contrast, were nearly lifetime studies (81/104) so that only a small "less than lifetime" correction factor would have been required (~2 fold).

Thus, with the exception of the RSD estimated from the Drew et al. (1983) data set, the estimated cancer potencies in rats and mice are remarkably close. This suggests that when the physiological and biochemical differences in rats and mice are properly considered, these species have similar sensitivities to the carcinogenic action of VC metabolites and provides support for the hypothesis that reliable estimates of human liver cancer can be produced by this technique.

The reason for the discrepancy in potency factors derived from the Drew et al. study is not clear, since even within the same species (mouse) and experimental paradigm (12 months exposure, tumors evaluated at or before 12 months; Lee et al., 1978, and Drew et al., 1983) dramatically different potencies were obtained. One possibility is that diagnostic criteria in this bioassay may have differed from those employed by other investigators, since angiosarcomas of the liver (a rare tumor) was not reported in any of the control mice from other groups but were reported in 2-5% of the control animals at NTP. This possibility could be evaluated by an expert committee of veterinary pathologists with access to slides from the archives of the different organizations.

It is also possible that the relatively high background incidence of liver tumors in the $B_6C_3F_1$ mouse has made it abnormally sensitive to the influence of liver carcinogens such as VC. This suggests that rodent strains with high background tumor incidences may not be good models to use when estimating human risk (if humans have much lower background incidences). In any case, as will be discussed later, the results from Maltoni's and Lee's groups appear to be much more consistent with the data from humans exposed to VC than the results of Drew et al. (1983).

Comparison of Potency Factors (PBPK and Conventional)

Maltoni's studies on VC carcinogenicity in rodents probably are the most extensive animal carcinogenicity data set in the world and were chosen as the most appropriate basis for estimations of human risk. Using the potency factor previously calculated for rats, it is possible to calculate the "unit risk" for humans continuously exposed to VC. The fitted dose response curve indicates that lifetime exposure to 1.77×10^{-3} mg equivalents of VC metabolites/day/liter of liver tissue is associated with an increase in liver cancer risk of 1×10^{-6} (maximum likelihood estimate: MLE). The 95% lower confidence limit on dose for this risk would be 1.40×10^{-3} mg equivalents of metabolite per day per liter of liver tissue.

It may be calculated with the PBPK model for humans that continuous exposure to 0.869 ppb of VC would be predicted to increase lifetime cancer risk by one in a million (MLE), or that continuous exposure to 1 µg VC per cubic meter would increase lifetime cancer risk by 4.51×10^{-7} (MLE). The corresponding 95% upper confidence limits (UCL) obtained from GLOBAL83 are 0.687 ppb (for one in a million risk) or 5.70×10^{-7} increase in lifetime excess risk for continuous exposure to 1 µg VC/cubic meter.

The numbers calculated with the PBPK model may be contrasted with the value reported in HEAST (1995). In each case the calculations represent the UCL for excess lifetime risk associated with continuous inhalation of 1 $\mu g/m^3$ of VC:

HEAST Value ..... 8.4×10^{-5} risk
PBPK Based Value ..... 5.7×10^{-7} risk

Thus the value calculated from the PBPK based approach described here suggests that the potency factor currently listed in HEAST should be reduced approximately 147 fold. In performing a risk estimation such as this one, there are many points where use of different assumptions/data (e.g., type of tumor modeled, selection of most sensitive species/bioassay as sole source of data, application of "less than lifetime" correction factors, etc.) can impact the cancer potency factor. Nevertheless, since PBPK modeling predicts roughly an order of magnitude less VC metabolism in humans than rodents at equivalent atmospheric concentrations and does not

include a surface area "correction" factor predicting that humans are always more sensitive than rodents by another order of magnitude. changes in these two factors appear to account for most of the differences (two orders of magnitude) in the two risk estimations.

The HEAST value is taken from a current issue of the EPA publication, but the entry for VC bears the note that the recommended values "... do not incorporate considerable information that is now available." The Office of Health and Environmental Assessment goes on to state that "One unpublished physiologically-based pharmacokinetic model prediction results in a 100-fold increased risk (emphasis added)." If the EPA were to increase the value in HEAST by 100 fold, then the procedures outlined here would differ from those in HEAST by 14,700 fold (more than four orders of magnitude).

Comparison with Human Epidemiology

It was noted above that considerable variation exists in the potency factors available for estimating the incidence of angiosarcomas in human populations exposed to VC. One of the most important questions, therefore, is: "Which of the alternative potency factors gives the most accurate description of the actual human experience?"

To answer this question, we have consulted the epidemiological literature generated on VC during the past 30–40 years. All of these studies, share, to some extent, the common problems of not having complete follow-ups for the total lifetimes of the individuals, the possibility of missing a tumor when another cause of death is present, imprecise measures of exposure, etc. After surveying the avilable literature, we chose to compare our predicted risks to data gathered by Simonato et al. (1991). This epidemiology study was chosen because we believe that it represents one of the most robust analyses available, both with regard to the number of individuals followed and the quality and length of follow up procedures employed.

A weakness in this database is the absence of a precise measure of the magnitude of VC exposures in the workplace. However, Simonato et al. (1991) have attempted to characterize the magnitude of occupational exposure by subdividing workers into groups based on their ppm.years of exposure (calculated as years on the job times TWA ppm levels estimated to be present in the occupational setting during hours of work = ppm.years). This grouping permited simulation of worker exposures with the PBPK model. The reader is reviewed to Simonato's manuscript for further details of the exposure estimations.

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Simonato et al. had 24 cases of liver cancer in their cohort. The overall incidence of liver cancer was statistically different than expected, and the odds ratios as high as 45:1 were observed in some of the groups with the longest duration of exposure and highest exposure concentrations (Table 9 in Simonato et al., 1991). For the purpose of this comparison, we selected a subgroup of workers with more than 25 years since first exposure, subdivided by Simonato et al. into four exposure categories: (1) < 2,000 ppm.years, (2) 2,000-5,999 ppm.years, (3) 6,000-9,999 ppm.years, and (4) >10,000 ppm years. Although this exposure information is obviously imprecise, it allowed the calculation of a roughly equivalent exposure paradigm in Table 5 so that we could predict the approximate tumor incidence in the groups studied by Simonato to compare with the results he reported.

For example, in the subgroup estimated to have the lowest exposures by Simonato (0-2,000 ppm.years), the "reported" incidence of angiosarcoma was 6.2 per 100,000. In contrast, the risk assessment procedure described in this manuscript gave a maximum likelihood estimate (MLE) of between 188 and 736 cases per 100,000 for ppm.years between 500 and 2,000 (Table 5). Thus the PBPK model predicted almost two orders of magnitude more cancer cases than actually occurred.

Similarly, individuals with 2,000-5,999 ppm years had a "reported" incidence of 42.2 cases per 100,000, while the PBPK based procedure estimated the incidence in this group to be from 700 to 1,500 cases per 100,000. A similar disparity existed for the two most highly exposed groups from Simonato et al. (153-280 cases per 100,000 versus 1,500 to 4,000 cases per 100,000 predicted by the PBPK based extrapolation. It is noteworthy that in the higher exposure group, the degree of overprediction by the PBPK procedure seems to decrease (from almost two orders of magnitude overprediction to approximately one order of magnitude: Table 5). It also appears that the degree of overprediction (excess conservatism) is greatest at the lowest rates of VC exposure (below 6,000 ppm.years in Table 5).

It should be noted that a large fraction of the cohort from Simonato is still alive, so it is possible that more tumors may be added to the 24 already reported. Nevertheless, in view of the long follow up time in the subgroup selected for comparison, it is considered extremely unlikely that the incidence will double even when all the workers are followed to the end of their natural lives.

Consequently, it appears that risk assessments based on estimates of the amounts of reactive metabolites of VC delivered to the liver of the target species (calculated with a PBPK model) still significantly overestimate the potential of VC metabolites to induce liver cancer in humans. Since the PBPK has been well validated in several species, we do not believe that this is because the PBPK model has overpredicted the formation of VC metabolites in human liver. Rather, it appears that the livers of humans are less sensitive to the carcinogenic effect of reactive VC metabolites the livers of the commonly used inbred laboratory rodents.

The reasons for this lower sensitivity of human livers to reactive metabolites are not clear, but it has been noted that longer lived species such as humans have higher levels of DNA repair enzymes than rodents. Thus production of a genotoxic lesion in humans may not have the same adverse consequences as in the relatively DNA repair-deficient rodents. A variety of other explanations are also possible, and clearly further research will be required before it is possible to choose between the different possibilities.

In summary, the procedures we have described here (based on a quantitative description of the metabolism of VC in different species and a well characterized oncogenic response to VC in different species) suggest that current estimates of the carcinogenicity of VC based on rodent studies significantly overestimate its oncogenic potential in humans.

Furthermore, there has been considerable discussion as to whether it is appropriate to include a "surface area factor" when using a PBPK model to extrapolate the results of rodent cancer studies to humans. We believe that the results of these studies suggest that inclusion of such a factor in a PBPK based risk assessment cannot be justified on either pharmacokinetic or pharmacodynamic grounds.

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CMA 114478

FIGURE LEGENDS

Figure 1: Predicted (solid line) and observed (open symbols) concentrations of vinyl chloride in a 9.1 liter recirculating exposure chamber containing 3 male rats (Figure 1a) or 3 female rats (Figure 1b).

Figure 2: Predicted (solid line) and observed (open symbols) amounts of radioactive metabolites derived from exposure of male rats to the indicated concentration of ^{14}C-vinyl chloride gas for 6 hours. Data taken from Watanabe et al. (1976).

Figure 3: Predicted (solid line) and observed (open symbols) concentrations of vinyl chloride in a 9.1 liter recirculating exposure chamber containing 14 male mice (Figure 3a) or 14 female mice (Figure 3b) with values of VMaxC and Km calculated from in vitro studies (VMaxC = 9.04, Km = 0.04). Figure 3c shows the same data for 14 male mice after computer optimization of VMaxC and Km (VMaxC = 8.13, Km = 0.25).

Figure 4: Predicted (solid line) and observed (open symbols) concentrations of vinyl chloride exhaled by human subjects following 7.5 hours of exposure to vinyl chloride concentrations of 59, 261, or 492 ppm. Data are taken from Baretta et al., (1969).

Figure 5: Predicted (solid line) and observed (open symbols) incidences of liver angiosarcoma in rats following exposure to various concentrations of vinyl chloride for 4 hr/day, 5 days/week, for 12 months (animals held until death for observation of tumor incidence). The dotted line represents the type of risk extrapolation that might have been prepared by EPA if the only data available for VC had been from two high VC concentrations but does not correspond to the actual EPA risk assessment (HEAST, 1993). Data are taken from Maltoni et al., (1974b).

CMA 114479

FOOTNOTES

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1 RHR Consulting Services. Midland. MI.

2 McLaren/Hart. ChemRisk Division. Cleveland. OH.

3 ICF Kaiser. K. S. Crump Division. Research Triangle Park. NC.

4 Zeneca Central Toxicology Laboratory, Macclesfield. ENGLAND

5 SimuSolv is a registered trademark of the Dow Chemical Co.

6 To receive a copy of the source code, please send a self-addressed stamped envelope. If a copy on magnetic media is desired, please include a formatted 3.5" DOS diskette with your request.

CMA 114480

Table 1

Parameters used in the physiologically based pharmacokinetic model for vinyl chloride for Humans, Rats, and Mice. Alveolar ventilation and cardiac output are calculated from the allometric constants by multiplying the constant by the body weight (kg) of the animal raised to the 0.74 power. VMax is calculated from the allometric constant VMaxC by multiplying the constant by body weight raised to the 0.70 power.

	HUMAN	RAT	MOUSE
WEIGHTS (% of Body Weight)			
Liver	3.14%	2.53%	5.86%
Rapidly Perf.	3.71%	5.0%	5.0%
Slowly Perf.	62.1%	76.47%	76.14%
Fat	22.1%	7.0%	4.0%
FLOWS (Allometric Constants)			
Alveolar Ventilation	15	18	28
Cardiac Output	15	18	28
	% of Cardiac Output		
Liver	24.0%	24.0%	24.0%
Rapidly Perfused	52.0%	52.0%	52.0%
Slowly Perfused	19.0%	19.0%	19.0%
Fat	5.0%	5.0%	5.0%
PARTITION COEFFICIENTS			
Blood/Air	1.16	1.68	2.41
Liver/Air	1.60	1.60	1.60
Rapidly Perfused/Air	1.60	1.60	1.60
Slowly Perfused/Air	2.10	2.10	2.10
Fat/Air	20.0	20.0	20.0
METABOLIC CONSTANTS			
Vmaxc (Allometric)	3.97	2.75	8.13
Km (mg/liter)	0.04	0.04	0.28

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Table 2

In vivo metabolic rate constants for the PB-PK model for vinyl chloride were estimated from data reported by Andersen et al. (1987 rats & mice; 1991 humans) for methylene chloride and Corley et al. (1990) for chloroform. Published in vivo maximum rates (VMax) of oxidative metabolism (catalyzed by P450 enzymes) and historic organ weight data from subchronic studies at Dow Chemical Co. were used to calculate the VMax/kg or liver (VMax/VL). Then the characteristic interspecies ratios of VMax/VL for oxidative metabolism of these typical halogenated hydrocarbons were used to estimate the in vivo VMax's for VC in mice and humans.

	Rat	Mouse	Human
Methylene Chloride:			
Body Wt (kg)	0.233	0.0275[a]	83.0
Percent Liver	2.53%	5.86%	3.14%
Liver Wt (g)	5.895	1.612	2606
VMax (mg/hr)	1.500	1.054	138
Ratio to Rat	1.000	2.570	0.208
Chloroform:			
Body Wt (kg)	0.230	0.0285	-
Percent Liver	2.53%	5.86%	-
Liver Wt (g)	5.819	1.670	-
VMax (mg/hr)	2.431	1.889	-
Ratio to Rat	1.000	2.707	-
Vinyl Chloride:			
Body Wt (kg)	0.225	0.0285	70.0
Percent Liver	2.53%	5.86%	3.14%
Liver Wt (g)	5.69	1.67	2198
Ratio to Rat (Av[illegible])	1.000	2.639	0.208
VMax (mg/hr)	0.968	0.749[b]	77.7[b]
VMaxC (allometric)	2.75	9.04	3.97

[a] Andersen et al. (1987) listed 34.5 g as the body weight for mice in their Table 1 since this was the body weight for the mice in the NTP bioassay of methylene chloride and their PB-PK model was used for risk assessment. However, the average body weight of the mice used in the gas uptake studies was actually 27.5 grams and this body weight is used to calculated the VMax/VL ratio.

[b] Calculated by multiplying the average mouse or human RATIO of VMax/VL's to rat (methylene chloride and chloroform), the in vivo VMax/VL value for VC in rats, and the VL for mice or humans.

Table 3

Incidence of angiosarcomas of the liver observed in Sprague Dawley rats. Data are from Experiments BT1, BT2, BT9, & BT15 conducted by Caseare Maltoni (1974a,b). Data are given as # angiosarcomas / # animals examined for males, females, and combined males and females. Control animals from several experiments are combined in the 0 ppm group. Animals were exposed 4 hr / day, 5 days / week for 52 weeks and then held until they died (typically at least another year). Tumors were scored at the time of death.

	Males	Females	Males + Females
Exposure Concentration (PPM)			
0	0/173	0/239	0/412
1	0/58	0/60	0/118[1]
5	0/59	0/60	0/119[1]
10	0/59	1/60	1/119
25	1/60	4/60	5/120
50	2/174	13/180	15/354
100	0/60	1/60	1/120[1]
150	1/60	5/60	6/120
200	7/60	5/60	12/120
250	1/29	2/30	3/59
500	0/30	6/30	6/60
2500	6/30	7/30	13/60
6000	3/29	10/30	13/59

[1] Eliminated from GLOBAL83 analysis because of mathematical limitations of the PC version of the computer fitting program (only 10 dose / response groups allowed).

Table 4

Incidence of angiosarcomas or the liver observed in Swiss albino mice. Data are from Experiment BT 4 conducted by Caseare Maltoni (1974a,b) and summarized in ECETOC, 1988. Data are given as # angiosarcomas / # animals examined for males, females, and combined males and females. Animals were exposed 4 hr/day, 5 days/week for 30 weeks and then held until tthey reached 81 weeks of age. To calculate the dose surrogates for mice, the PB-PK model was configured according to Table 1 and a 4 hr exposure with 20 hours exposure free was simulated by the model. The simulated values of the dose surrogate were converted to lifetime average daily doses by multiplying by 5/7 (days/week) and 30/104 (fraction of lifetime exposed). Potency values were estimated with GLOBAL83 as described in the Methods section.

	Males	Females	Males + Females	PB-PK LADD
Exposure Concentration (PPM)				
0	0/80	0/70	0/150	0
50	1/30	0/30	1/60	38.4
250	9/30	9/30	18/60	173.1
500	6/30	8/30	14/60	265.2
2,500	6/29	10/30	16/59	331.0
6,000	2/30	11/30	13/60[a]	—
10,000	1/26	9/30	10/56[a]	—

[a] Eliminated from dose response regression because of the likelihood of poor survival at this dose.

Table 5

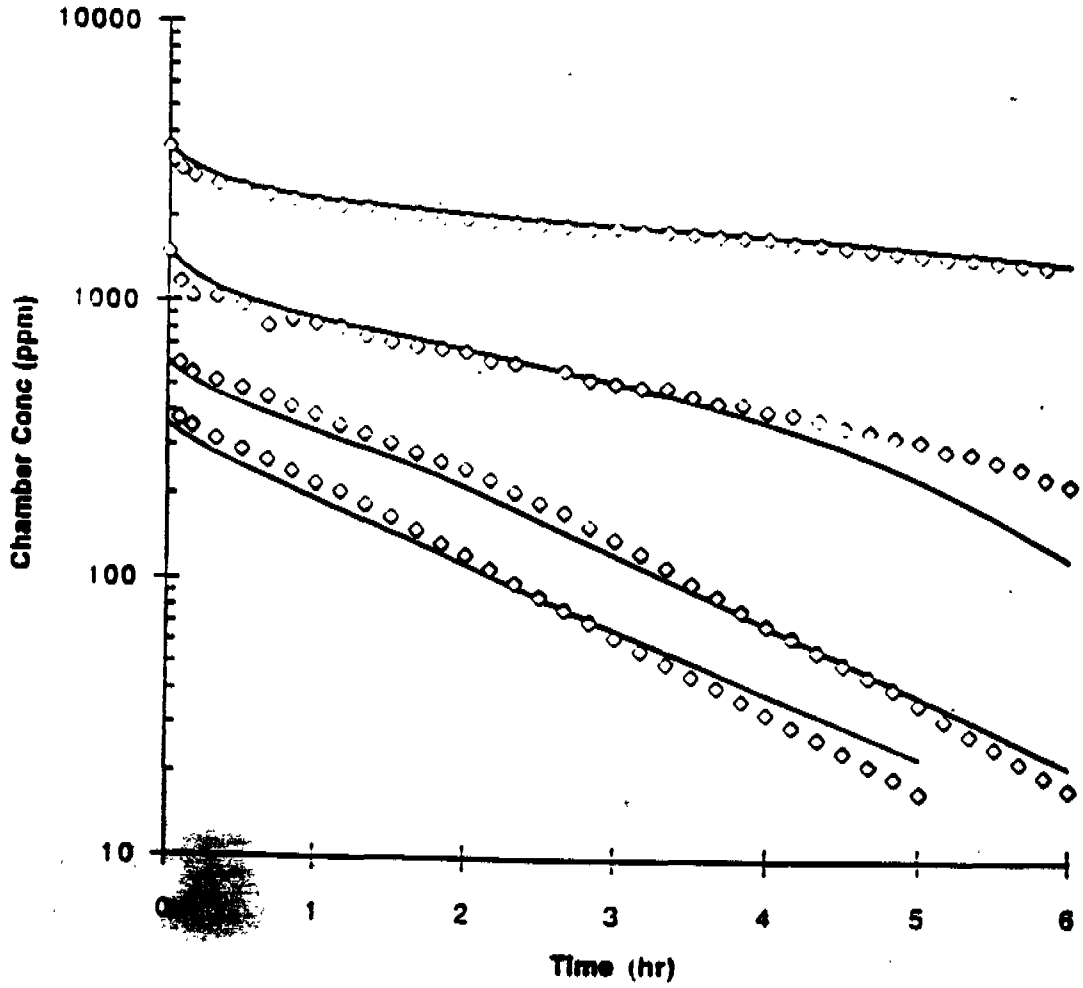
Lifetime Average Delivered Doses (LADD's) of VC metabolites in humans exposed to VC for 5 days/week, 50 weeks/year, for the indicated numbers of years. The LADD's are calculated from the PB-PK model for humans constructed as outlined in Methods, correcting for the fraction of a year exposed (50/52) and the fraction of a lifetime exposed (10, 20, or 30/70). Estimated lifetime risks (incidences/100,000) predicted for these exposures based on the rat potency factor are obtained from the GLOBAL83 computer program (specifying the Maximum Likelihood Estimate, NOT the 95% upper confidence limit). Observed cases per 100,000 at different levels of exposure (cumulative ppm.years) for the group having more than 25 years since first employment are obtained from Table 10 of Simonato et al., (1991).

PPM	Years Exposed	PPM Years	PB-PK LADD	PB-PK Prediction per 100,000	Observed Cases per 100,000
Ten Years Exposure					
50	10	500	3.33	188	—
100	10	1,000	6.63	374	(6.2)[a]
200	10	2,000	13.06	736	—
—	—	4,000	—	—	42.2
500	10	5,000	26.68	1,497	—
—	—	8,000	—	—	152.3
1000	10	10,000	31.28	1,753	—
—	—	>10,000	—	—	(280.0)[b]
2000	10	20,000	36.03	2,532	—
Twenty Years Exposure					
50	20	1,000	6.66	376	(6.2)[a]
100	20	2,000	13.26	747	—
200	20	4,000	26.11	1,465	42.2
—	—	8,000	—	—	152.3
500	20	10,000	53.35	2,971	
—	—	15,000	—	—	(280.0)[b]
1000	20	20,000	62.57	3,476	
2000	20	40,000	72.07	3,993	

[a] Group listed as having <2,000 cumulative ppm years by Simonato et al., entered at 1,000 ppm.years for comparison.

[b] Group listed as having >10,000 cumulative ppm.years by Simonato et al., entered between 10,000 and 20,000 ppm.years for comparison.

Fig 1a



CMA 114486

Fig 1b

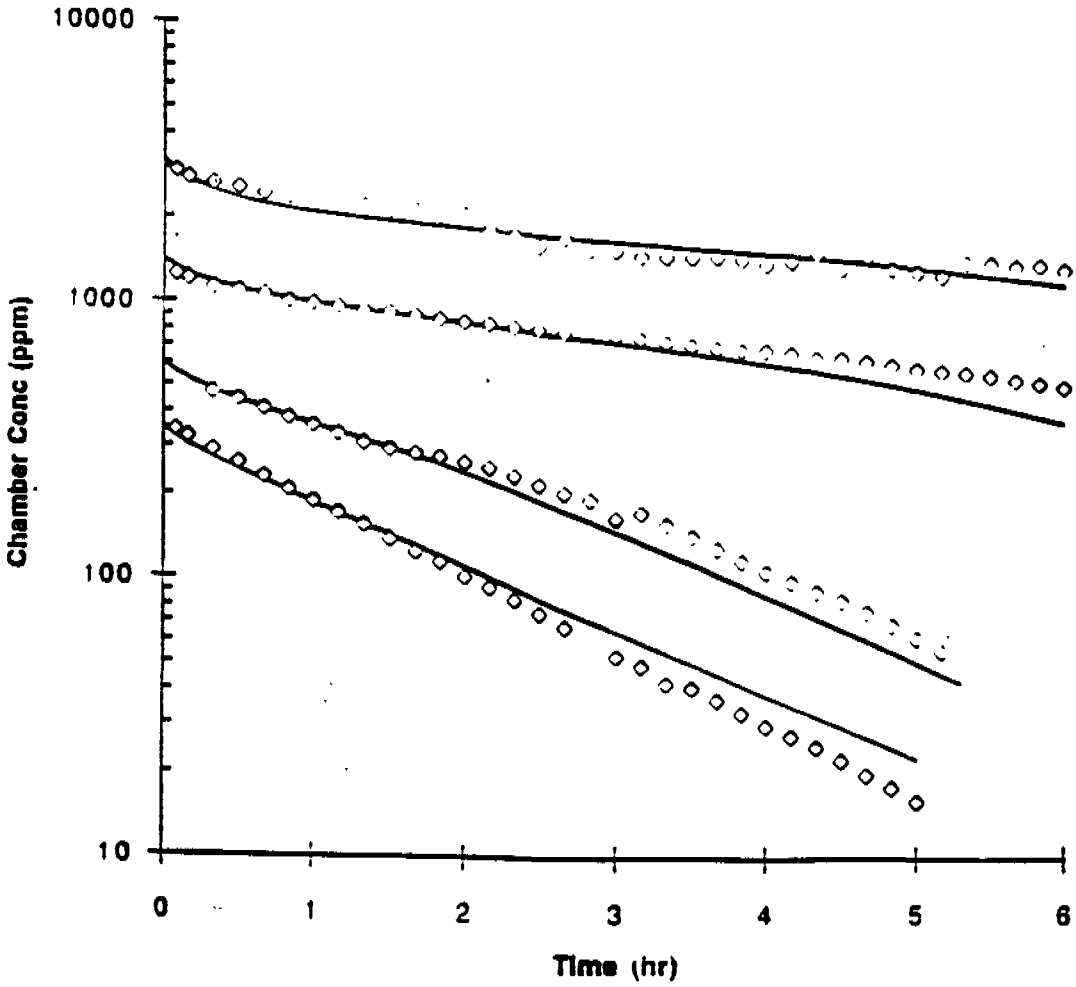


Fig 2

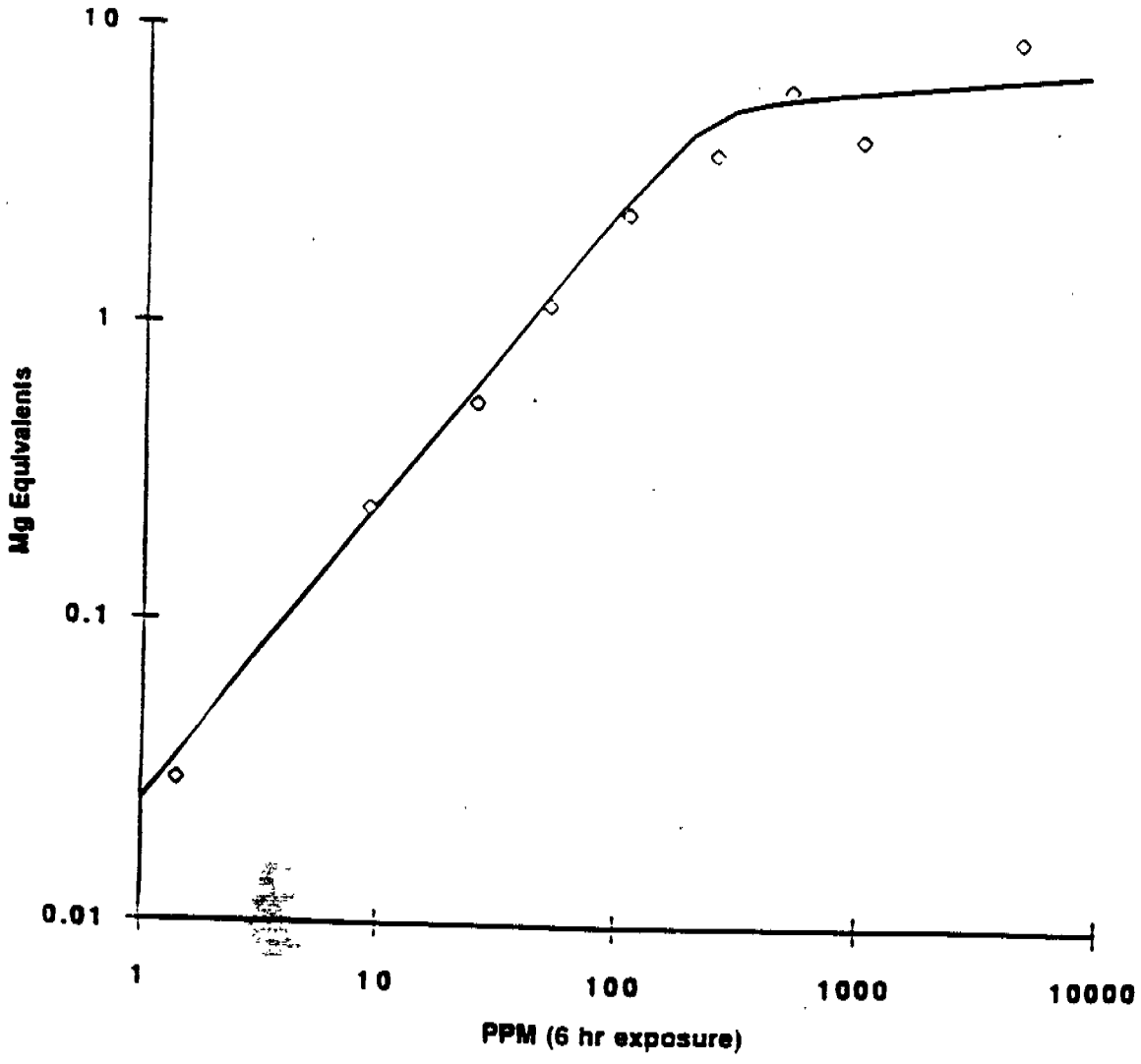


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Fig 3a

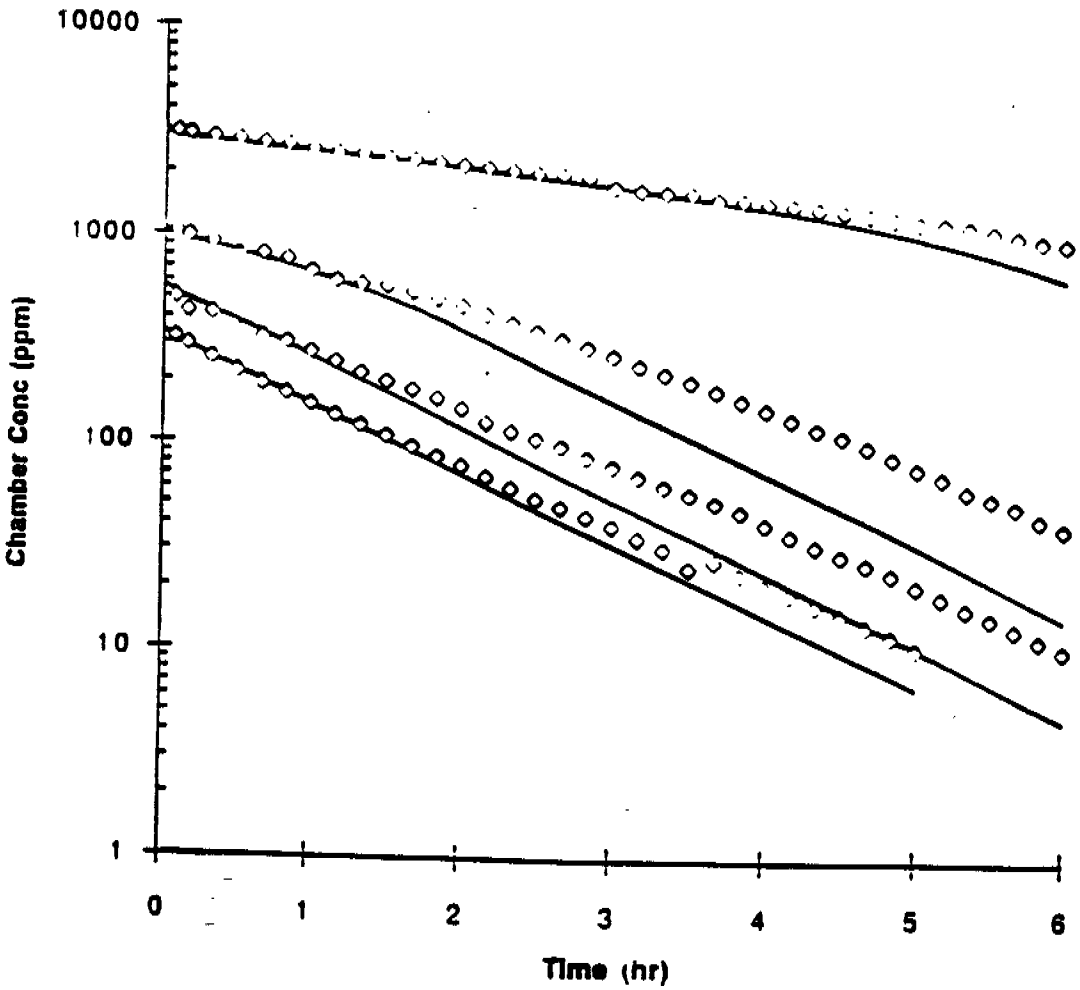
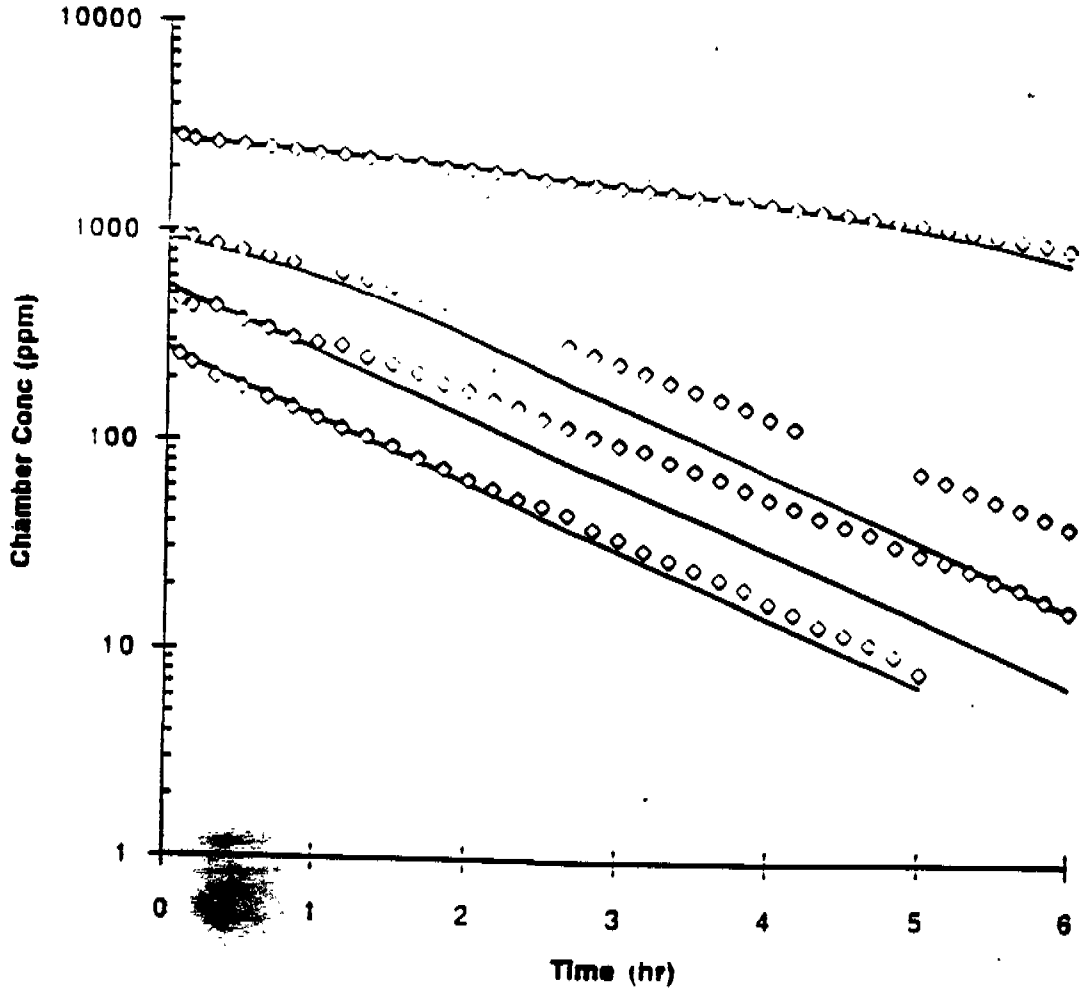
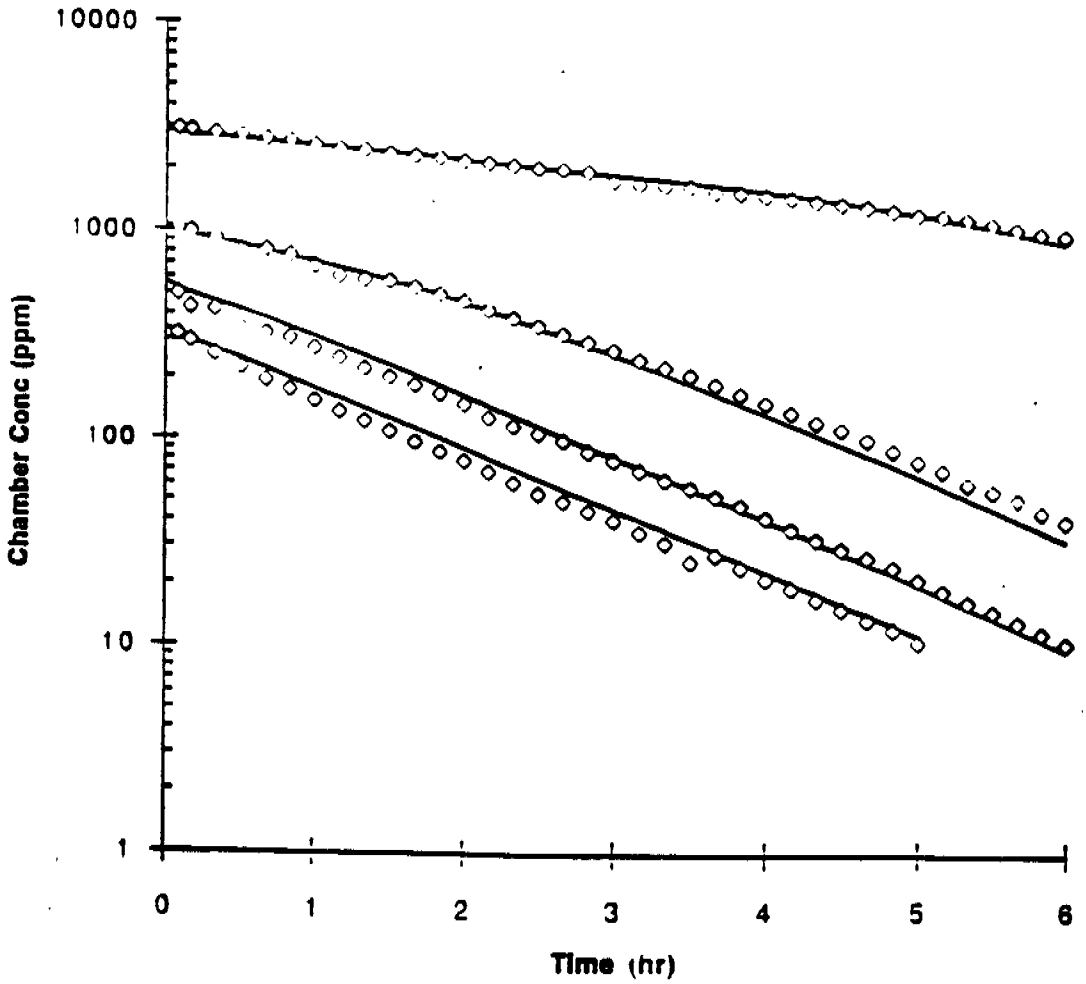


Fig 3b



CMA 114490

Fig 5c



CMA 114491

Fig 4

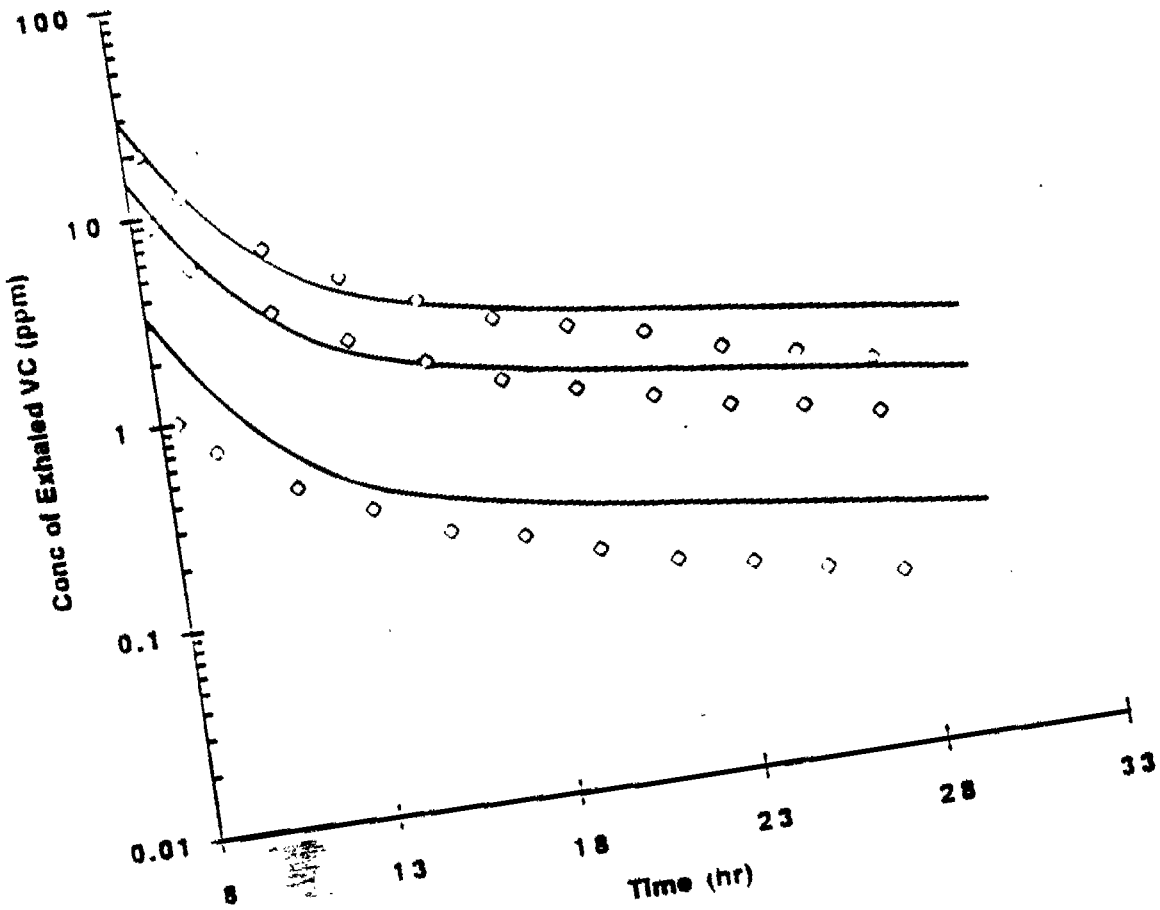


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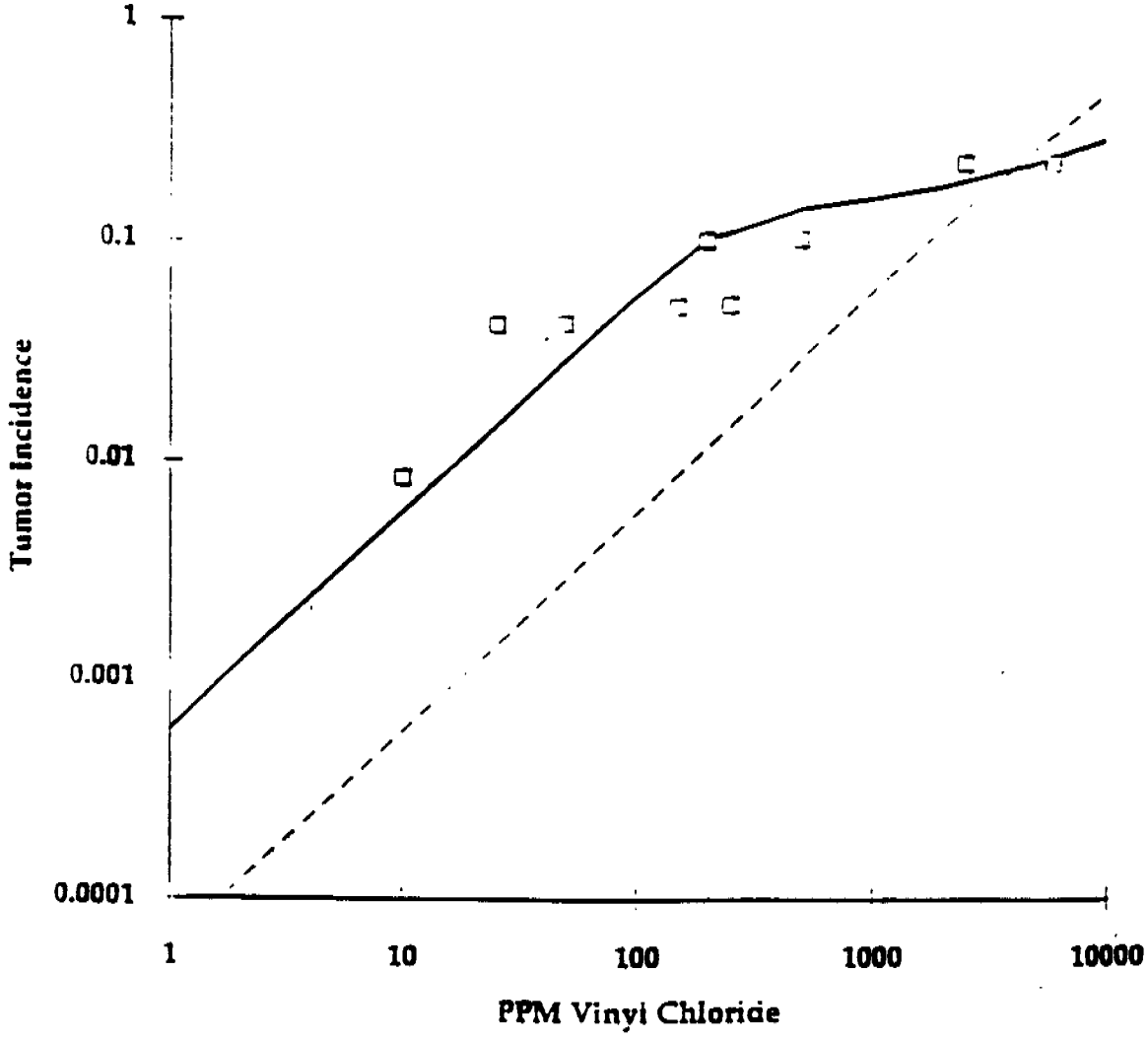
Fig 5



CMA 114493

CONSIDERING PHARMACOKINETIC AND MECHANISTIC INFORMATION IN CANCER RISK ASSESSMENTS FOR ENVIRONMENTAL CONTAMINANTS: EXAMPLES WITH VINYL CHLORIDE AND TRICHLOROETHYLENE

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ABSTRACT

Risk assessments for vinyl chloride (VC) and trichloroethylene (TCE) are presented as examples of approaches for incorporating chemical-specific pharmacokinetic and mechanistic information into a more scientifically plausible cancer risk assessment. For VC, the evidence regarding mode of action includes direct reaction of a metabolite with DNA, resulting in DNA adducts and misstranscription, and cross-species target-tissue correspondence of a rare tumor type. Risk estimates for human exposure to VC predicted with a physiologically-based pharmacokinetic (PBPK) model and the linearized multistage (LMS) model were lower than those currently used in environmental decision-making by a factor of 30 to 50, and were more consistent with human epidemiological data. For TCE, there is evidence of increased cell proliferation due to receptor interaction or cytotoxicity in every instance in which tumors are observed, and the tumors typically represent an increase in the incidence of a commonly observed, species-specific lesion. Virtually safe exposure estimates for human exposure to TCE predicted with a PBPK model and a margin of exposure (MOE) approach were higher than those obtained by the conventional LMS approach by roughly a factor of 100. The MOE approach is recommended as an alternative to the LMS approach for chemicals with a carcinogenic mode of action which entails increased cell proliferation, leading to the expectation of a highly nonlinear cancer dose-response.

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INTRODUCTION

Assessing the potential risk associated with human exposure to carcinogenic environmental contaminants represents an uncomfortable admixture of scientific evaluation and political policy, with the potential for enormous impact on both the public health and the economic well-being of the nation. The principal challenge facing cancer risk assessors today is to realistically consider the implications of the chemical's mechanism(s) of carcinogenicity

developing a risk assessment approach for a particular carcinogenic effect. It is becoming increasingly difficult to justify the use of the same standard risk assessment approach with chemicals that act through a purely radiomimetic, genotoxic mechanism, as well as with chemicals for which carcinogenicity is mediated by increased cell proliferation secondary to cytotoxicity or receptor interaction. Mechanism-dependent risk assessment approaches are the only alternative for maintaining the credibility of cancer potency estimates in the face of increasing sophistication in the understanding of the mechanisms of carcinogenicity. The new draft revisions to the U.S. Environmental Protection Agency (USEPA) guidelines for cancer risk [1] would appear to provide the flexibility necessary to move forward in this area.

Risk assessments for chemical carcinogens must necessarily be iterative in nature. It is in the nature of scientific inquiry that understanding develops slowly, as experimental information accumulates and theories can be tested and refined. Risk assessments, however, cannot be postponed indefinitely until an adequate understanding of the carcinogenicity of a particular chemical has been achieved. Therefore, it is necessary to attempt to perform the most scientifically defensible assessment possible, given the information available at that time, and to be ready to revise the estimate, repeatedly, whenever important new information is developed. In the last few years there has been a significant improvement in the level of understanding regarding chemical carcinogenesis in general and the mechanisms of carcinogenicity of vinyl chloride (VC) and trichloroethylene (TCE) in particular. The purpose of the study reported here was to attempt to perform state-of-the-science risk assessments for VC and TCE, using to as great an extent as possible the information currently available on pharmacokinetics, metabolism, and carcinogenic mechanism of action.

VINYL CHLORIDE

When it became evident that VC was carcinogenic both in animals and in humans, many of its uses were discontinued; the current use of VC is limited to serving as a chemical precursor in the production of such materials as polyvinyl chloride (PVC) and copolymer resins. However, VC is also produced from the biodegradation of trichloroethylene by bacteria in the soil. Thus past spills of trichloroethylene may lead to current or future exposures of the public to VC in drinking water or other environmental media. The current potency estimates for VC published by the USEPA do not quantitatively incorporate pharmacokinetic information on VC into the risk calculations [2]. To provide a more accurate assessment of human risk from exposure to VC, a physiologically-based pharmacokinetic (PBPK) model was developed which describes the uptake, distribution and metabolism of VC in the mouse, rat, hamster, and human following inhalation or oral exposure. The PBPK model was used to predict the total production of reactive metabolites from VC both in the animal bioassays and in human exposure scenarios. These measures of internal exposure were then used in the linearized multistage (LMS) model [3] to predict the risk associated with lifetime exposure to VC in air or drinking water.

Evidence for carcinogenicity: The carcinogenicity of VC has been well established in several animal species by a number of routes of exposure [2]. Of the many different tumor types which have been reported in animal bioassays of VC, four are of greater concern because they have been seen reproducibly at low concentrations (250 ppm and below): liver angiosarcoma, hepatocellular carcinoma, neurroblastoma, and mammary gland adenocarcinoma. Of these, two are particularly notable in that they are rarely seen in untreated animals: liver angiosarcoma and neurroblastoma. Greater than expected incidences of angiosarcoma of the liver have also been reported in a number of cohorts of workers occupationally exposed to VC [2]. Angiosarcoma of the liver is considered to be a very rare type of cancer, with only 20-30 cases per year reported in the U.S. [4]. Increased death due to cancer associated with human VC exposure has also been reported for brain, lung, and hematopoietic systems, as well as for other tissues, but several analyses have concluded that liver angiosarcoma show the clearest evidence for causal association and also demonstrate the highest relative risk [5]. The correspondence across species for liver hemangiosarcoma is quite striking and has made this tumor the primary focus for VC risk assessments in recent years.

Metabolism: Based on the elimination of VC observed following administration by various routes of exposure, the metabolism of VC appears to be a dose-dependent, saturable process. The primary route of metabolism of VC is by the action of the mixed function oxidase (MFO) system, now referred to as Cytochrome P450 or CYP, on VC to form chloroethylene oxide. Chloroethylene oxide (CEO) is a highly reactive, short-lived epoxide that rapidly rearranges to form chloroacetaldehyde (CAA), a reactive α-halocarbonyl compound [6]. The main detoxification of these two metabolites is conjugation binding with glutathione (GSH), as evidenced by the observation of decreased non-protein sulfhydryl concentrations at high VC exposure concentrations [7].

Mechanism of carcinogenicity: It has long been a tenet of carcinogenic risk assessment that the mechanism of carcinogenicity for "genotoxic" carcinogens (sometimes referred to as initiators) involves reaction with DNA, leading to misstranscription during subsequent cell division, causing a loss or change in heritable information which results in a neoplastic daughter cell. As early as 1978, it was demonstrated that binding of VC to liver macromolecules following inhalation exposure of rats correlated well with both total metabolism and the observed incidence of angiosarcoma [8]. It was suggested that the carcinogenicity of VC was due to binding of a reactive metabolite with DNA and subsequent miscoding during cell reproduction. The *in vivo* formation of four etheno-DNA adducts have since been demonstrated following exposure of animals to VC: 1,N^2-ethenoguanine; N^2,3-ethenoguanine; 1,N^6-etheno-2'-deoxyadenosine, and 3,N^4-etheno-2'-deoxycytidine [9]. These etheno-adducts are highly persistent and can lead to defective transcription [10].

Selection of a risk assessment approach: Based on the information described above on the metabolism and mechanism of carcinogenicity of VC, it is necessary to determine the appropriate approach for conducting a human risk assessment. The evidence is strong that the carcinogenicity of VC is related to the production of

reactive metabolic intermediates. The most appropriate pharmacokinetic dose metric for a reactive metabolite is the total amount of the metabolite generated divided by the volume of the tissue into which it is produced [11]. In the case of VC, a reasonable dose metric for angiosarcoma would be provided by the total amount of metabolism divided by the volume of the liver. The assumption underlying the use of this dose metric is that the concentration of the actual carcinogenic moiety, or the extent of the crucial event associated with the cellular transformation, is linearly related to this pseudo-concentration of reactive intermediates, and that the relationship of the actual carcinogenic moiety or crucial event to the dose metric is constant across concentration and species. Specifically, the average amount generated in a single day is used, averaged over the lifetime (i.e., the lifetime average daily dose, or LADD). The use of a dose rate, such as the LADD, rather than total lifetime dose, has been found empirically to provide a better cross-species extrapolation of chemical carcinogenic potency [12]. Subsequent steps in the carcinogenic mechanism related to specific adduct formation, detection, and repair, as well as to the consequences of DNA mistranscription and the potential impact of increased cell proliferation, have not yet reached the point where they can be incorporated into a risk assessment in any quantitative form. However, there appears to be sufficient evidence to justify the assumption that VC acts as a classic initiator, producing genetic transformations through direct reaction of its metabolites with DNA. Therefore the traditional assumption of low-dose linearity of risk appears to be warranted, and the LMS model would seem to be the most appropriate approach for low-dose extrapolation.

Description of PBPK model: The PBPK model for VC used in this study is an adaptation of a previously described PBPK model for vinylidene chloride [13]. For a poorly soluble, volatile chemical like VC, only four tissue compartments are required: a richly perfused tissue compartment which includes all of the organs except the liver, a slowly perfused tissue compartment which includes all of the muscle and skin tissue, a fat compartment which includes all of the fatty tissues, and a liver compartment. The physiological parameters used in the model are the current USEPA reference values [14]. The model assumes flow-limited kinetics, or venous equilibration; that is, that the transport of VC between blood and tissues is fast enough for steady state to be reached within the time it is transported through the tissues in the blood. The partition coefficients are based on in vitro studies with tissue suspensions [15]. All metabolism is assumed to occur in the liver, which is a good assumption in terms of the overall kinetics of VC, but which would have to be revised to include target-tissue-specific metabolism if a serious attempt were to be made to perform a VC risk assessment for a tissue other than the liver [11]. Metabolism of VC is modeled by two saturable pathways: one high affinity, low capacity, representing P450 2E1, and one low affinity, high capacity, representing the other P450 isozymes (e.g., 2C11/6 and 1A1/2). The parameters for the two oxidative pathways in the mouse, rat, hamster, and human were estimated by fitting the model to data from closed-chamber inhalation exposures with each of the species and strains of interest [16].

In the model, the reactive metabolites produced by these pathways (whether CEO, CAA, or other intermediates) may then either be metabolized further, leading to CO_2, react with GSH, or react with other

cellular materials, including DNA. Because exposure to VC has been shown to deplete circulating levels of GSH, a simple description of GSH kinetics was also included in the model [13]. Initial estimates for the subsequent metabolism of the reactive metabolites and for the glutathione submodel in the rat were taken from the model for vinylidene chloride [13]. These parameter estimates were then refined for the case of VC with data on glutathione depletion [17,7], total metabolism [18], and CO_2 elimination [19]. The parameters obtained for this portion of the model in the rat were used for the other species with appropriate allometric scaling (i.e., the first-order rate constants were scaled by body weight raised to the -1/4 power).

Pharmacokinetic risk assessment: The model just described was used to calculate the pharmacokinetic dose metrics for angiosarcoma in the most informative of the animal bioassays [20,21,22], as well as for human inhalation exposure. The 95% upper confidence limits (UCLs) on the human risk estimates for lifetime exposure to 1 part per billion (ppb) VC were then calculated on the basis of each of the sets of bioassay data, using the LMS model, and the resulting risk estimates are shown in Table 1.

Table 1: Human risk estimates (per million) for lifetime exposure to 1 ppb vinyl chloride in air based on the incidence of liver angiosarcoma in animal bioassays

Animal Bioassay Study	95% UCL Risk / million / ppb	
	Males	Females
Maltoni *et al.* - Mouse Inhalation [20,21]	1.52	3.27
Maltoni *et al.* - Rat Inhalation [20,21]	5.17	2.24
Feron *et al.* - Rat Diet [22]	3.05	1.10
Maltoni *et al.* - Rat Gavage [20,21]	5.68	15.70

The risk estimates based on inhalation studies with mice (1.5×10^{-6} and 3.3×10^{-6}) agree very well with those based on inhalation studies with rats (5.17×10^{-6} and 2.24×10^{-6}), demonstrating the ability of pharmacokinetics to [illegible] dose-response information across species. The risks estimated from the dietary administration of VC ([illegible]$\times 10^{-6}$ and 1.1×10^{-6}) are also in good agreement with those obtained from the inhalation bioassays, showing good route-to-route correspondence of potency based on the pharmacokinetic dose metric. However, the estimates based on oral gavage of VC in vegetable oil (5.68×10^{-6} and 15.7×10^{-6}) are about 6-fold higher than either dietary or inhalation exposure. Incorporation of corn oil into the diet increased the yield of aflatoxin B$_1$-induced tumors in rats [23]; a similar phenomenon could be responsible for the apparently higher potency of VC when administered by oil gavage compared to incorporation in the diet.

Epidemiological analysis: In order to evaluate the plausibility of the risks predicted on the basis of the animal data, risk calculations were also performed on the basis of the best available epidemiological data [24,25,26]. A linear relative risk dose-response model was used for analysis of the human data. To obtain pharmacokinetic, human-based risk estimates, the PBPK model was run for the exposure scenario appropriate to each of the selected subcohorts from each of the studies. The resulting internal dose metrics were multiplied by the appropriate durations to obtain the cumulative internal doses, which were then input into the relative risk model, along with the observed and expected liver cancer deaths for each subcohort, to obtain an estimate of the carcinogenic potency. Then, to determine the risk associated with a continuous lifetime exposure to 1 ppb for comparison with the animal results, the PBPK model was run for a 1 ppb continuous exposure and the average daily value of the internal dose metric was calculated. Using the 95% upper bound on the estimate for the potency provides a 95% upper confidence limit on the lifetime risk per ppb of vinyl chloride for comparison with the animal-based results obtained with the *LMS* model.

Table 2: Human risk estimates (per million) for lifetime inhalation of 1 ppb vinyl chloride in air based on the incidence of liver angiosarcoma in human epidemiological studies

Epidemiological Study	95% UCL Risk / million / ppb
Fox & Collier [24]	0.71 - 4.22
Jones *et al.* [25]	0.97 - 3.60
Simonato *et al.* [26]	0.40 - 0.79

A comparison of the results of the analyses of the three sets of data, shown in Table 2, gives some indication of the consistency of the human results, even before the comparison with the animal predictions. It is encouraging that the lifetime risk of liver cancer per ppm VC exposure estimated from the three studies only ranges over about one order of magnitude: from 0.4×10^{-6} to 4.2×10^{-6}. Moreover, these estimates are in remarkable agreement with the estimates based on animal data shown in Table 1. However, any confidence produced by this agreement should be tempered by the likelihood that misclassification of exposure in the human studies tends to underestimate the true risk at lower doses. Nevertheless, the agreement of the pharmacokinetic animal-based risk estimates with the pharmacokinetic human-based risk estimates provides strong support for the assumption used in this study: that cross-species scaling of lifetime cancer risk can be performed on a direct basis of lifetime average daily dose (without applying a body surface area adjustment) when the risks are based on biologically appropriate dose metrics calculated with a validated PBPK model.

Conclusions: Giving priority to the animal studies most closely approximating the human route of exposure, the best conservative estimate of the carcinogenic risk of angiosarcoma from lifetime exposure to 1 ppb

VC in air is 5.2×10^{-6}, or 2.0×10^{-6} $(\mu g/m^3)^{-1}$, based on inhalation studies in male rats (20,21). This value is consistent with the range of estimates from epidemiological studies of 0.4×10^{-6} to 4.2×10^{-6} risk per ppm VC, but is roughly a factor of 30 below the currently published inhalation unit risk of 8.4×10^{-5} $(\mu g/m^3)^{-1}$. The model was also used to estimate the daily internal dose for human drinking water consumption. The resulting best conservative estimate of the carcinogenic risk of angiosarcoma from lifetime exposure to 1 $\mu g/L$ VC in drinking water is 1.14×10^{-6} $(\mu g/L)^{-1}$, based on studies with male rats of the dietary administration of VC [22]. This value is roughly a factor of 50 below the currently published unit risk of 5.4×10^{-5} $(\mu g/L)^{-1}$.

Although VC has often been cited as a chemical for which saturable metabolism should be considered in the risk assessment, saturation appears to become important only at very high exposure levels (greater than 250 ppm by inhalation or 25 mg/kg/day orally) compared to the lowest carcinogenic levels, and thus has little impact on the quantitative risk estimates. The important contribution of pharmacokinetic modeling is to provide a more biologically plausible estimate of the effective dose: total production of reactive metabolites at the target tissue. The ratio of this biologically effective dose to the administered dose is not uniform across routes and species. Therefore any estimate of administered dose is less accurate for performing route-to-route and interspecies extrapolation of risk. The risk estimates obtained for VC using the pharmacokinetic dose metric are lower than those obtained with conventional external dose calculations by a factor of 30 to 50, and appear to be more consistent with human epidemiological data.

TRICHLOROETHYLENE

TCE has been widely used in industry for many years because of its excellent solvent properties and its nonflammability. The ACGIH has recently announced its intention of classifying TCE into a new carcinogenicity group, A5 (not suspected as a human carcinogen), based on a well-conducted, negative epidemiological study performed in an aircraft maintenance facility at Hill Air Force Base by the National Cancer Institute [27,28]. The USEPA, on the other hand, has for a number of years regulated TCE on the basis of its carcinogenicity, although it has wavered between group 2B (sufficient evidence in animals) and C (limited evidence) in trying to classify the likelihood of carcinogenicity from TCE [29,30,31]. However, in 1989 the International Agency for Research on Cancer classified TCE as a group 3 animal carcinogen (limited evidence) [32], and the USEPA has since withdrawn the classification of TCE from its IRIS database for consideration. Nevertheless, regardless of the formal classification the USEPA cancer risk estimates for TCE [30,31], calculated on the basis of metabolized dose with the LMS model, have continued to be used for environmental decision-making since 1985. In contrast to the case of VC, the most recent potency estimates for TCE published by the USEPA do attempt to incorporate pharmacokinetic information on TCE into the risk calculations [30,31]. The current USEPA unit risks for TCE, 1.7×10^{-6} $(\mu g/m^3)^{-1}$ and 0.32×10^{-6} $(\mu g/L)^{-1}$, are based on total metabolized dose in mg/kg/day, adjusted by body surface area (i.e., by the ratio of the body weights raised to the negative 1/3 power), which provides a reasonable

approximation to the internal exposure (area under the concentration curve) for the metabolites. However, it is disconcerting to note that these pharmacokinetically based potencies for TCE are very similar to those shown above for VC. In spite of the strong epidemiological evidence suggesting that VC is a more potent human carcinogen than TCE. Clearly, pharmacokinetics alone is inadequate to provide a reasonable comparison of the human risk for cancer from these two chemicals. Just as the pharmacokinetics of a chemical must always be considered in order to obtain a realistic measure of internal exposure to the chemical, the pharmacodynamics of the chemical (that is, the mechanism by which the chemical causes cancer) must also be considered in order to obtain a realistic measure of the response to the chemical.

Several steps are involved in performing a risk assessment for TCE that considers both pharmacokinetics and mechanism. Information must first be gathered on the pharmacokinetics and metabolism of TCE, as well as on each of its key metabolites: chloral (CHL), trichloroacetic acid (TCA), trichloroethanol (TCOH), dichloroacetic acid (DCA), and dichlorovinylcysteine (DCVC). This pharmacokinetic and metabolism data can then be used in a PBPK model to provide a prediction of the concentration profiles for TCE and its metabolites in each of the target tissues, whether associated with exposure to TCE in the animal bioassays or in potential human exposure scenarios. Mechanistic information specific to each of the tumors of concern must then be incorporated to provide a link between target tissue chemical exposure and biological or biochemical effects in the target tissue leading to the observed cancer response. The specific mode of action associated with the production of a particular tumor provides the basis for expectations regarding both the dose-response for tumor incidence and the nature of cross-species scaling. These expectations, in turn, should drive decisions concerning the most appropriate risk assessment approach and the assumptions to be made where chemical-specific data are lacking.

Evidence for carcinogenicity: By far the most common carcinogenic outcomes associated with TCE exposure are liver and lung tumors in several strains and both sexes of mice [33]. Statistically increased tumor outcomes observed in only a single study include malignant lymphoma in HAN:NMRI mice exposed by inhalation, renal tubular cell adenoma and carcinoma in male F344 rats exposed by oral gavage, and benign testicular (leydig cell) tumors in Sprague-Dawley rats exposed by inhalation. Of these, the kidney tumors have raised the greatest concern since they were not observed in control animals. Direct human evidence of carcinogenicity from TCE exposure is equivocal at best: epidemiological studies have generally been negative, although most are limited by problems due to small cohorts, inadequate latency periods, and co-exposure to other contaminants [33]. The largest study, mentioned above [27,28], was unable to link TCE exposure with increased cancer incidence in any tissue. A few epidemiological studies have, however, tentatively linked TCE exposure with increased incidence of urinary tract tumors and lymphoma in workers, as well as with childhood leukemia [33].

Metabolism: Based on both *in vitro* and *in vivo* studies, the metabolism of TCE has been suggested to consist of initiate oxidation of TCE to CHL by the MFO system, followed by either oxidation of CHL to TCA by an aldehyde oxidase or reduction to TCOH by alcohol dehydrogenase (ADH) with subsequent glucuronidation; oxidation of TCOH to TCA was also proposed [34]. DCA has been identified as a minor urinary metabolite of TCE (on the order of 1%) in both rats and mice, but has not been detected as a metabolite of TCE in the human. Significantly, the clearance of DCA in humans appears to be much more rapid than would be expected from allometric scaling of animal data; the extremely high rate of clearance of DCA in humans is probably responsible for the failure of investigators to detect it as a metabolite of TCE.

Mechanism of carcinogenicity in the liver: It has been suggested that both TCA and DCA play a major role in the tumor incidence observed in mice dosed with TCE [35]. Both compounds have been shown to produce focal hyperproliferative lesions, adenomas, and carcinomas on chronic administration. The typical sequence of events for tumorigenicity can be described as follows: initially upon treatment with DCA or TCA, there is evidence of a slight, but generalized liver hyperplasia, consistent with the induction of a mitogenic signal by the chemical. However, this increased cell proliferation soon returns to a normal liver turnover rate in spite of continued exposure to the mitogen, presumably in response to the expression of endogenous negative growth factor (TGF-β) by stromal cells. Upon repeated exposure for about 30 weeks, however, there is a sudden appearance of hyperplastic nodules, which eventually progress to neoplastic lesions. This sequence of events is entirely consistent with a "suppression escape" mechanism for promotional carcinogenicity [36]. The sudden appearance of rapidly dividing cells, representing an escape from cytostatic suppression, produces a greatly increased probability of mutational events leading to an increased tumorigenicity.

The observation that similar concentration-time profiles of DCA and TCA produce the hyperplastic and tumorigenic responses in the mouse but not in the rat apparently reflects a difference in the susceptibility of the two species to the onset of hyperplasia. Since *in vitro* studies with rat hepatocytes have demonstrated the mitogenic response, the most likely possibility for the observed difference in susceptibility is a differential genetic predisposition for the escape from suppression. The maternal imprinting of the gene for a negative growth factor receptor in the mouse [37] provides one such possible explanation, if the rat is not similarly predisposed genetically. Since the human appears to have both alleles for this gene [37], it is possible that the much lower potency of TCA and DCA in the rat provides a more realistic estimate of the potency that could be expected in the human.

Mechanism of carcinogenicity in the lung: Tumors have also been observed in the lungs of mice exposed to TCE by inhalation. The mechanism in this case appears to be entirely different from that just described for the liver. In a well-designed experimental effort [38], which provides an excellent example of the kind of studies needed to support biologically-based risk assessments, investigators at ICI combined *in vivo* and *in vitro* experi

elucidate the mechanism of TCE carcinogenicity in the mouse lung. In the *in vivo* studies, female mice and rats were exposed to TCE at a range of inhaled concentrations at and below the concentrations at which tumors are observed in mice, and the effects of TCE in the lung were determined. A specific lesion, characterized by vacuolization of lung Clara cells, was observed in mice, but not rats. There was evidence of a threshold for the Clara cell effects at about 20 ppm. Mice exposed to 100 ppm CHL by inhalation displayed Clara cell lesions similar to those observed with 1000 ppm TCE. In contrast to these results, only mild effects were observed with TCOH inhaled at 100 ppm, and none were observed with 500 mg/kg TCA given intraperitoneally (the effects had been observed with intraperitoneally administered TCE at 2000 mg/kg). These results suggested that CHL was responsible for the toxicity.

In the *in vitro* studies, mouse lung Clara cells were shown to metabolize TCE to CHL, TCOH, and TCA, with CHL being the major metabolite. Significantly, no TCOH glucuronide was detected. In comparison with mouse Clara cells, mouse hepatocytes were shown to produce primarily TCOH and its glucuronide. In both cell preparations, a steady state concentration of CHL was achieved. Separate *in vitro* studies demonstrated that mouse Clara cells possess a relatively low activity for the glucuronidation of TCOH as compared either to the glucuronidation of other substrates in the lung or to the glucuronidation of TCOH in the liver. It has also been determined that ADH, the enzyme which converts CHL to TCOH, has a low activity in the mouse lung, consistent with the relatively low production observed in the Clara cells. On the basis of this evidence, the investigators concluded that the observed acute toxicity in the lung was a result of accumulation of CHL in Clara cells resulting from a limitation in the formation of TCOH and its glucuronide. The specificity of this lesion for the Clara cells can be rationalized in terms of their relatively high Cytochrome P450 activity, coupled with limited ADH and UDP glucuronosyl transferase (UGT) activities.

The implications of these results for the lung tumorigenicity of TCE are two-fold. First, the accumulation of CHL, if it does occur *in vivo*, has clear carcinogenic implications, since CHL has been shown to be genotoxic in a number of studies [38]. Secondly, the recurrent toxicity observed with intermittent exposure is likely to produce compensatory cell proliferation, exacerbating the genotoxic effect. The fact that the lung tumors were generally benign is also significant: the production of primarily benign tumors is more consistent with a nongenotoxic, cell-proliferative mechanism.

Mechanism of carcinogenicity in the kidney: While both of the tumors discussed thus far are observed in the mouse but not in the rat, the reverse is true for the kidney tumors produced by TCE. A mechanism for the induction of these tumors has been proposed, in which direct conjugation of TCE with glutathione (GSH) in the liver is followed by further metabolism in the kidney to a cysteine conjugate which can then be cleaved to a reactive intermediate in the kidney tubular cells [39]. The cysteine conjugate formed from TCE, dichlorovinylcysteine (DCVC), has been shown to be highly nephrotoxic as well as mutagenic in the Ames test.

Detoxification and clearance of DCVC takes place by urinary excretion of the N-acetyl derivative: the fact that N-acetyl-DCVC has been identified in the urine of humans exposed to TCE occupationally [39], indicates that exposure of the kidney to DCVC does occur in the human.

As with the two previous cases, cell proliferation also appears to play a role in this tumor outcome. In the only bioassay that reported a significant increase in kidney tumors from TCE, cytotoxicity was observed in the kidney at both the low and high doses, while tumors were observed only at the high dose. Kidney cytotoxicity was also reported in association with a non-statistically-significant incidence of kidney tumors in the only other study demonstrating the tumor response.

Selection of a risk assessment approach: With regard to the use of a cancer dose-response model, the highly nonlinear dose-response expected for the receptor-mediated, promotional mechanism suggested for the liver carcinogenicity of TCE argues against the use of the usual linear extrapolation to low-dose risk associated with the use of the *LMS* model [3]. In the case of the lung and kidney, although genotoxicity may lead to a small but finite residual risk component which is linear at low dose, there is also evidence for cytotoxicity at the high doses where tumors are actually observed. The extreme nonlinearity of the impact of cytotoxicity driven, compensatory cell-proliferation on risk at the doses where tumors are observed is incompatible with the behavior and underlying assumptions of the *LMS* model, even if a pharmacokinetic dose metric is used. It has frequently been suggested that the *LMS* model may simply be inappropriate for use with chemicals whose carcinogenicity is mediated by changes in cell proliferation, and that a promising alternative in such cases is a biologically based dose-response (BBDR) model of cancer which incorporates cell proliferation; it is also possible to link such cancer models to PBPK descriptions of target tissue exposure to provide a more complete description of the carcinogenic process for cytotoxic or mitogenic chemicals [40,41]. However, there are at least two difficulties associated with the use of these alternatives to the *LMS* model. First, the parameters for the cell proliferation models are often not available from direct experiment, but must be estimated by fitting bioassay data. Unfortunately, it has been shown that the parameters in the BBDR model are not independently identifiable under such conditions, and therefore the estimates of risk at low dose could vary widely depending on the specific parameterization chosen [42]. Secondly, even when experimental data on hyperplastic nodules or altered hepatic foci permit a more independent estimate of the cancer model parameters, low-dose risk estimates with the typical 2-stage BBDR model are exquisitely sensitive to the estimated dose-response for the model parameters [43].

Another alternative which has been suggested for risk assessments with non-genotoxic carcinogens is the use of a threshold approach based on the underlying process which is required for carcinogenicity [44]. The rationale for the existence of a threshold in non-genotoxic carcinogenicity is that, unlike the case for direct reaction with DNA, the nonlinear process underlying carcinogenicity in these cases (e.g., cytotoxicity or response to receptor binding) is not one which would be expected to be active at very low doses. The greatest difficulty in

gaining acceptance for the threshold approach is that, strictly speaking, it implies that no increased risk is incurred from exposures below the threshold, but only an apparent (experimentally observable) threshold can be determined. Residual carcinogenic activity below the apparent threshold for the nonlinear process could result from insufficient experimental power to detect the effect at lower incidence, from secondary mechanisms (e.g. from the mutagenic activity of a chemical which is also cytotoxic at higher concentrations), or from the inherent nature of the dose-response for the effect (e.g. for receptor-mediated effects possessing a linear dose-response in the low-dose regime).

The margin of exposure (MOE) approach is similar in practice to the threshold approach, but the assumptions underlying its use are not as constraining. Rather than estimating a human exposure threshold below which no risk is expected, the MOE approach merely estimates the human exposure producing a dose-metric value which is a specified factor ("margin") below the value of the dose metric at which a minimal tumor response (e.g. 10% – the ED_{10}) was observed in animals. The MOE approach admits the possibility that in spite of the presence of a highly nonlinear dose-response in the experimental regime there may still be residual risk, and even low-dose linear behavior, below the apparent threshold for the nonlinear process. However, it relies on the nonlinearity of the process underlying the carcinogenic mode of action to assure that the margin of risk between the human and animal exposures is much greater than the MOE. That is, an MOE of 100 might be expected to provide a risk reduction of greater than 1000, while an MOE of 1000 might be expected to provide a risk reduction of greater than 100,000 (since it is expected that risk falls off at a much faster rate than exposure).

Description of PBPK model: The PBPK model for TCE used in this study is an expansion of a previously published model of TCE and its metabolite TCA [45], to include the other key metabolites: DCA, TCOH (which is the principal source of DCA), DCVC in the kidney, and CHL in the lung. The parent chemical portion of the model includes individual tissue compartments for the liver, gut tissue, fat, and tracheo-bronchial region of the lungs. All other tissues are lumped into rapidly perfused (kidney, brain, alveolar region of lungs, etc) and slowly perfused (muscle, skin, etc) compartments. The model includes both inhalation and oral routes of exposure. Oral gavage is modeled using a two-compartment description of the GI tract. Allometric scaling is used throughout the model (flows and capacities scaled by body weight to the three-quarters power, rate constants scaled by body weight to the negative one-quarter power) to simplify intraspecies and interspecies extrapolation.

The model includes three target tissues: lung, kidney, and liver. The dose metrics provided in the lung are the instantaneous concentration and area under the curve (AUC) for CHL in the tracheo-bronchial region, which is assumed to be produced by saturable production and clearance of CHL in Clara cells. The dose metric in the kidney is total production of the thioacetylating intermediate from DCVC divided by the volume of the kidney. The model implicitly assumes that all glutathione conjugation of TCE leads eventually to the appearance of DCVC in the kidney. Clearance of TCE by N-acetyl-transferase into the urine is also modeled. Two dose metrics are

included in the description of the liver AUC for DCA and AUC for TCA. The model assumes that all oxidative metabolism proceeds through CHL, which is further metabolized to TCA and TCOH. TCOH can subsequently be oxidized to TCA, conjugated with glucuronic acid, or reduced to DCA. DCA is also produced from the reduction of TCA. Biliary excretion of TCOH glucuronide and enterohepatic recirculation of free TCOH is described, with only the glucuronide being excreted in the urine. The model is able to reproduce data on TCE, TCOH and TCA kinetics in the mouse, rat, and human, as well as DCA kinetics in mice, for both inhalation exposure and oral gavage.

Table 3: Comparison of virtually safe lifetime exposure levels (ppb in air or µg/L in water) for TCE based on the Margin of Exposure (MOE) approach and the Linearized Multistage (LMS) approach

MOE[a] = 1000	ED_{10} / MOE	MOE Level	10^{-6} Risk Level[b]
• Inhalation (ppb):			
Lung	0.009	6000 (9)[c]	41 (0.06)
Kidney	9.02	15000 (36)	300 (0.64)
Liver	1.59	88 (12.5)	0.35 (0.05)
• Drinking Water (µg/L):			
Lung	0.009	600,000 (900)	4000 (6.0)
Kidney	9.02	225,000 (540)	4500 (9.6)
Liver	1.59	390 (56)	5.6 (0.8)

[a] Margin of exposure below ED_{10} (dose corresponding to an extra risk of 10%)
[b] Lifetime extra cancer risk based on the Linearized Multistage Model
[c] Alternate (worst-case) calculation -- see text

Pharmacokinetic risk assessment: The results of the dose metric calculations with the PBPK model are summarized in Table 3. In this table, the most plausible estimates of acceptable exposure levels are shown for each target tissue and [illegible] exposure scenario of concern. The numbers in parentheses represent alternative, worst-case risk estimates. The purpose for including these alternative estimates is to demonstrate the broad uncertainty in the current risk estimates. In every case the discrepancy between the best estimate and worst-case estimate could be greatly reduced by experiments which are well within the state of the science. In the case of lung tumors, the numbers in parentheses represent the calculations which assume that the cross-species scaling for the clearance of CHL in the lung parallels that of P450 (which falls off dramatically) rather than following allometric expectations. In the case of kidney tumors, the numbers in parentheses represent the calculations which

assume an extremely low GST pathway production in the rat compared to the human (based on one animal study) rather than assuming a production more in keeping with allometric expectations (based on another animal study). Both of these uncertainties can readily be addressed by *in vitro* studies similar to those which have been performed with methylene chloride [47]. In the case of liver tumors, the numbers in parentheses represent the calculations which assume that the human susceptibility to mutagenic carcinogens is similar to that of the mouse, as opposed to the most plausible estimates, which assume that the human susceptibility is more similar to the rat. The studies needed to resolve this question are more difficult to define, but the importance of this question goes well beyond TCE, and the benefits of such studies would be significant.

Table 3 also demonstrates two different approaches for estimating acceptable exposure levels for the public. In the traditional quantitative risk estimate approach, the LMS model is used to obtain quantitative estimates of the risk associated with a given human exposure scenario, based on the bioassay dose-response data. Typically, USEPA considers an increased lifetime risk of cancer on the order of 10^{-6} to be acceptable for the public [29,30,31]. Depending on the target tissue, the TCE exposure levels associated with an increased lifetime risk of 10^{-6} range from 0.35 to 300 ppb in air or 3.6 to 4500 $\mu g/L$ in water. For comparison, the most recently published risk estimates from USEPA would equate to lifetime 10^{-6} risk exposure levels of 0.11 ppb and 3.1 $\mu g/L$.

A second approach for estimating acceptable levels is to simply relate the human exposure level to the animal bioassay results by determining the ratio between the ED_{10} in the animals (calculated from the bioassay data using the multistage model) and the dose metric for the human exposure. Alternatively, an acceptable ratio, or MOE, can be set and the corresponding human exposure can be calculated directly from the ED_{10} and the MOE. This is the approach shown in the left side of Table 3. In each case, the acceptable dose metric levels were calculated by dividing the appropriate ED_{10} by the desired MOE. The model was then used to translate the acceptable dose metric level into an acceptable exposure level. Based on an analogy between nongenotoxic carcinogenicity and noncancer toxicity, a minimum MOE of 100 would seem to be justified on the basis of 10 for human variability (particularly for variability in the activities of the key metabolizing enzymes) and 10 for uncertainty in the animal to human extrapolation. For exposures of the public, it might sometimes be appropriate to add an additional margin of 10 because of the potentially large number of individuals exposed. For the purpose of this illustration an MOE of 1000 was used in obtaining the public exposure levels in Table 3. Depending on the target tissue, the TCE exposure levels which provide an MOE of 1000 range from 85 to 15000 ppb in air or 390 to 600,000 $\mu g/L$ in water. In general, the MOE approach results in acceptable levels which are higher than those obtained by the LMS approach by roughly two orders of magnitude.

Conclusions: The basis for determining which of the two approaches is the most appropriate is the mode of action of the chemical carcinogenicity being considered. For example, it would seem clear that the use of the LMS approach is both justified and preferable in the case of a carcinogen such as vinyl chloride for which the evidence regarding mode of action includes (a) direct reaction of a metabolite with DNA that results in DNA

adducts and misrepresentation; (b) no evidence of enhanced cell proliferation, receptor interaction, or cytotoxicity at clearly tumorigenic doses; and (c) cross-species target-tissue correspondence of a rare tumor type. The cancer risk from a genotoxic chemical like vinyl chloride is very likely to fall off linearly with dose, even to very low exposure levels. On the other hand, the MOE approach seems quite applicable, and more appropriate than the LMS approach, for a carcinogen such as TCE for which (a) there is no similar evidence of direct interaction with DNA, (b) there is evidence of enhanced cell proliferation due to receptor interaction or cytotoxicity associated with every target tissue, and (c) there is little evidence of cross-species correspondence or the production of rare tumor types (the kidney tumors providing the possible exception). The cancer risk from a chemical like TCE, for which the mode of action appears to involve the highly nonlinear impact of enhanced cell proliferation, is very likely to fall off much faster than dose, producing an incremental reduction in risk far exceeding the reduction in exposure.

Acknowledgements

This study was supported by the USEPA Office of Health and Environmental Assessment and the U.S. Occupational Safety and Health Agency (USOSHA) Department of Health and Environmental Policy. However, the views presented in this paper are strictly those of the authors and do not necessarily reflect the position of either of the agencies. The authors are greatly indebted to the USEPA and USOSHA project officers, Lorenz Rhomberg and Christine Whitaker, for their guidance, support, and thoughtful discussions.

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OEHHA'S RESPONSE TO COMMENTS FROM WILLIAM F. POLICH, PH.D.

1. For a pathway of exposure to humans to be complete there needs to be a source of release, a route of exposure, and a point of human contact. While the Class I Unit was the source of vinyl chloride emissions from the B.K.K. Landfill, and air is a route of exposure, the point of human contact is not clearly established until the development of homes north of Amar near Nogales beginning in 1977. According to the USEPA aerial photographic analysis of the landfill dated July 28, 1964, the nearest residential development in 1964 was approximately 1,000 feet to the southwest of the landfill. Concentrations of gases emitted into the air will decrease with distance from the landfill. Residential areas other than the southeast and south either have not been impacted at all or by as high and as frequent detections of vinyl chloride. The "Scope" section of the Introduction states the refined nature of the addendum report. This is summarized in the second paragraph of the Executive Summary.

2. Please see Risk Characterization, Exposed Population.

3. A worst-case (70-year; 1980-2050) exposure estimate has been added to TABLE 10 to facilitate comparison with the interim report.

4. Please see Hazard Identification and Exposure Assessment, Other Hazardous Chemicals Detected in Ambient Air.

5. a) Please see TABLE 10 for risk estimates using 5 ppb as the substituted value for samples with nondetectable concentrations in the 1981-1982 data set.
   b) Please see TABLE 10 for risk estimates using 5 ppb as the assumed exposure concentration in 1980.
   c) Discussion of potential underestimation of the exposure concentration at Station MY for the years 1980-1983 has been expanded.
   d) We assume an exposure concentration for 1980 because the 1981-1982 data suggests concentrations at Stations A, B, and MY were not likely zero. However, there is no way of knowing quantitatively what exposures were prior to monitoring data.

6. We recognize your concern regarding past exposures. The inability to use flare data has been discussed in the addendum report. Manifest data is categorical data, as was shown in Table I of the interim report. There is no generally accepted scientific method to convert such categorical data to specific chemical concentrations in air at a receptor location for use in exposure assessment and quantitative risk estimation.

February 9, 1996

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West Covina, CA 91791

Office of Environmental Health Assessment
Hazardous Waste Toxicology Section
601 North 7th Street
P O Box 942732, MS 241
Sacramento, CA 94234-7320

Attention: Dr David M. Siegel

Subject: Review of Addendum Health Risk Assessment of Ambient Fugitive Vinyl Chloride Emissions from the Class 1 Unit of the BKK Landfill, West Covina, California

Dear Dr Siegel,

The subject document has been reviewed and a list of comments and suggested modifications is attached. Please contact me at 818-919-8272 if there are any questions on my comments.

Sincerely,

William F Polien

William F Polien, Ph.D.

FEB 22 1996

Hazardous Waste Toxicology

CMA 114513

Draft Addendum Assessment of Ambient Fugitive Vinyl Chloride Emissions
From the Class I Unit of the BKK Landfill, West Covina, California
Comments and Suggested Modifications

1 The rationale given for assuming 1980 as the start of the exposures because no one was then living within a few hundred feet of the location of the monitoring stations sounds ridiculous. There were more than 30,000 people living within a mile of the landfill in 1979 and many within a few hundred yards of the monitoring locations. With this line of reasoning, if the monitoring locations had been located a few hundred feet to the north on BKK property, no exposures would have occurred to date and the risk would be zero regardless of the magnitude of the emissions or the number of people living nearby. The real reason for selecting 1980 was the lack of emission data prior to 1980 that met EPA analysis standards. Surely, if adequate emission data were available, the assessment would have started in 1963. The analysis should not assume the exposure to residents living near Bkk started in in 1980 because that is not true. It should say that the assessment started in 1980 because adequate data to evaluate the risk for the period from 1963 through 1979 are not available. The report should clearly state in the Executive Summary that the carcinogenic exposures to the residents living within a mile of the landfill started in 1963 but adequate data to estimate the risk was not available for the years prior to 1980.

2. The report is very misleading. It gives the impression that the total cancer risk for all residents who ever lived near the landfill has been estimated and shown to be less than the values given in the report. The scope and limitations of the assessment should be clearly defined in the report. The dimensions of the areas surrounding the monitoring stations for which the assessment results are applicable and the number of residences included within these areas should be precisely defined. The report should state residents living in other areas near the landfill could have received greater exposure levels and acrued higher risks because of higher annual emission levels in previous years or the cumulative effects of exposures since 1963. An estimate of the number of people living within a mile of the landfill as a function of time since 1963 should be made and included in the report. This would help show how the analysis had to be limited in scope because of the lack of data.

3. The report should point out that the use of the 30 and 9 year exposure durations when calculating the the cancer risk is not adequate for a substantial portion of the people living near BKK. Many people living within a mile of BKK have already been exposed to carcinogenic emissions for 33 years and surely some now young adults will be exposed for more than 60 years.

4 The cancer risk due to carcinogens other than vinyl chloride present in the landfill gas at BKK is minimized and quickly dismissed in the report. Analyses of the of the composition of the landfill gas at the inlet to the flares consistently show enough carcinogens other than vinyl chloride at high enough concentrations to cumulatively equal or exceed the cancer risk due to vinyl chloride. There is no mechanism by which these chemicals will not enter into the neighborhoods in roughly the same ratio to vinyl chloride as present in the inlet

gas. The presence of some of these carcinogens in the background is not important. The emitted concentrations will always be higher than the background near the source. The Executive Summary should contain the information that carcinogens other than vinyl chloride are present in the landfill gas but were not included in the assessment. It should also contain the information that, based on concentration measurements of the landfill gas at the flare inlets, the presence of these other carcinogens cause a cancer risk similar in magnitude to vinyl chloride. The estimated cancer risk would be at least twice that reported if the other carcinogens were included in the assessment.

5. The report claims that the estimated excess cancer risks are upper bound and actual excess cancer risks are likely to be lower. Actually, the opposite is true. The analysis contains many assumptions which substantially minimize the risk. These include the following:

a. The use of 1 ppb for the samples with nondetectable concentrations for the 1981-1982 data. The reason given is that by using 1ppb, the average annual concentrations for 1981 and 1982 are then more consistent with the 1983 annual average. The average annual concentration was lower in 1983 because of improvements in the gas collection and incineration system. It is not reasonable to assume that the daily and day to day variations in the measurements were as high as 10 to 1. A more reasonable assumption is 1/2 of the detection limit (5 ppb for this data) for the samples with nondetectable concentrations as was done for all the other measurement data.

b. The assumption of 2 ppb as the annual average concentration of vinyl chloride in 1980 underestimates the actual value. The plot of average annual vinyl chloride in Figure 17 should have included the 1981 and 1982 data. If it had, entirely different conclusions would have had to be drawn since the average concentrations rapidly increased from 1983 through 1981. A concentration value for 1980 which was lower than 1983 never could have been selected. The implied correlation between the tons of hazardous waste deposited and the measured average annual concentrations.in addition to ignoring the 1981 and 1982 data, does not include the effect of emission control measures implemented at the landfill which substantially altered the emissions data.

The average annual vinyl chloride concentration in 1980 had to be much larger than the average of the concentrations measured from June through December of 1981. From November 1980 to June 1981, the gas collection system was expanded, vinyl chloride was banned and a maintenance program initiated to add fill and groom the slopes to reduce escaping gases. The USC report of September 1980 found the working face at BKK to be the major source of volatile organic compound emissions. A conservative assumption would be that the banning of vinyl chloride reduced the emissions by at least 15 to 20 percent. In December 1980 the operating gas collection and incineration system had 15 wells and 2 flares. By June 1981, the system had 55 wells and 4 flares: a 3.67 fold increase in capacity. The January 1981 Eutek report states that the existing gas recovery system of 15 wells and 2 flares had the capability of extracting 2200 cfm of landfill gas. The report also states that BKK estimated that the total gas production at the site in January 1981 was 6600 cfm. The expanded gas collection system of 55 wells and 4 flares completed by

June 1981 had the capability of extracting 8100 cfm which is 1500 cfm more than what was estimated to be produced by the landfill. Thus, since the banning of vinyl chloride eliminated emissions from the working face, if the gas control system was 100 percent efficient, the vinyl chloride emissions would have been reduced to zero in June 1981. To estimate the actual reduction, a gas control system efficiency of 50 percent in 1980 and 55 percent in 1981 was assumed. A higher efficiency was used for 1981 because of improved maintenance of the slopes and the addition of 2 new more efficient flares. Using these assumptions, a 61 percent reduction in the1980 vinyl chloride emissions was achieved by the expansion of the gas control system installed between January and June 1981. Eutek in their January 30, 1981 report estimated that the installation of a planned gas control system expansion which would increase the capacity to 5500 cfm would reduce the odor emissions by 68 percent which is general agreement with the above estimate. Combining the emission reductions due to the gas control system expansion and the elimination of working face emissions gives a total reduction of 76 to 81 percent from 1980 to June 1981. Thus, the average annual vinyl chloride concentrations in 1980 are about 4 times greater than the average of the concentrations measured from June through December 1981. There obviously was a lot of vinyl chloride brought to BKK in 1980 since banning it in 1981 reduced the total amount of hazardous waste deposited in 1981 by 180,000 tons.

c. The assumption that the average annual concentration for Station MY for the period 1980-83 was equal to the Station A values is not reasonable and lowers the estimated risk for Station MY. Since the measured concentrations at Station MY are consistently higher than at Station A for the years prior to 1983, it is reasonable to assume the concentration values were higher at Station MY than at Station A in 1980-83 when no measurement were made at Station MY. A comparison of the 1984 -1987 measurements shows that the Station A data are on average 49 percent higher than Station MY. Therefore, the average annual vinyl chloride concentrations at Station MY are assumed to be 49 percent greater than those at Station A for the years prior to 1984

d. Tables 1 and 2 were prepared to show what the 1981 and 1982 monthly vinyl chloride concentrations for Stations A and B and the 1980 through 2009 average annual vinyl chloride concentrations would be if the changes in assumptions discussed in a, b, and c above were implemented. Table 1 was developed by substituting a value of 5 ppb (as discussed in Comment 4 a) for the samples with nondetectable concentrations for the months of June 1981 through September 1982 for Stations A and B. The 1980 average annual concentrations for Stations A and B were obtained by multiplying the average of the concentrations for the months June 1981 through December 1981 by 4 (for the reasons given in Comment 4 b) to give 38.4 (4x9.6) for Station A and 34 4 (4x8.6) for Station B. A linear decrease in concentration from 1 January 1981 to 1 June 1981 (from 38.4 to 9 6 for Station A and 34 4 to 8.6 for Station B) was assumed to determine the concentrations for January through May. This is a reason able assumption since the changes in the gas collection system were incrementally added from January through May in 1981 The average annual vinyl chloride concentrations for Station MY were obtained by increasing the corresponding Station A values by 46 percent as discussed in Comment 4 c.

Using the average annual concentrations from Table 2, estimates of the excess cancer risk were made for Stations A, B and MY. The results are shown in Tables 4, 5 and 6. The risks are seen to be 2 to 3 times higher when the effect of emission control measures implemented at BKK prior to June 1981 are considered in the analysis.

6. What is really needed are cancer risk estimates for all the residents living within a mile or so of the landfill. The residents have a right to know what they have been exposed to. The risk should be determined as a function of time since 1963 and should include all of the carcinogens known to be present at BKK. This was the original request of the residents and the original objective of this study. Perhaps this is felt to be impossible to do because of a combination of lack of data and analysis requirements dictated by EPA. However, there should be enough information to make reasonable estimates of the risks since 1972 when all toxic landfills and government agencies had to start maintaining records of all toxics deposited in landfills. Water Quality should have records of the amount of waste deposited since 1963 from which the amount of landfill gas produced each year can be estimated. Documentation should be available at AQMD on the development of the gas collection system and other controls to reduce emissions to estimate the amount of landfill gas leaving BKK. AQMD should also have measurements of the composition of the landfill gas at the inlets of the flares since the experiments with the small test flare in 1975 from which the concentrations of the carcinogens can be determined. The waste producers manifests provide information on the amount of toxics deposited each year and their general composition. If all the information available at the landfill monitoring agencies was used, a reasonable estimate satisfying EPA requirements could probably be accomplished. The report should be expanded to include an assessment of the excess cancer risk starting in 1972.

For the years prior to 1972, hazardous waste was not defined nor controlled so that no data are available for cancer risk evaluation. However, all Class 2 landfills accepted industrial waste some of which was later classified as hazardous. Most of the Class 2 landfills, which never accepted toxics after 1972, are now emitting vinyl chloride. Operating Industries Landfill has been found to be emitting enough vinyl chloride to exceed the air quality standard of 10 ppb for a 24 hour average in the surronding neighborhood although it never accepted toxics. Carcinogens were obviously deposited at BKK prior to 1972 and risk estimates starting in 1972 will underestimate the risk to residents living near Bkk in prior years. The report should also be expanded to contain a discussion of the probable carcinognic exposures and cancer risks from living near the landfill from 1963 to 1972.

Estimates of the excess cancer risks acrued since 1972 at Stations A, B and MY are presented in Tables 5, 6 and 7. These estimates were derived by estimating the average annual vinyl chloride concentrations for the years 1977 -1979 based upon the development of the gas control system. In Comment 4 b, it was determined that the 1980 average annual concentration was higher than in 1981 primarily because of the expansion of the gas control system. Similarly, the concentration was probably higher in 1979 than in 1980 because the number of gas extraction wells was increased from 9 to 15 in 1980. In 1979

the capacity was tripled by the addition of a new flare and 5 gas wells so that the 1978 concentration was probably higher than the 1979 value. Also, the concentration in 1977 could have been higher than 1978 because a gas control system of 4 wells and 1 flare were installed in 1978. Thus, the concentrations in 1980 were higher than in 1981 and each prior year could have been higher until 1977 because of the reductions in emissions achieved by an expanding gas control system. In the analysis, the concentrations for the years 1977 -1980 were assumed equal because the amount of landfill gas would probably be increasing and compensate for the increased gas collection. A linear extrapolation to a concentration of zero on 1 January 1972 was used to determine the concentrations for the years 1972-1976. The estimated excess cancer risks are about 10 times greater than those shown in the report.

CMA 114518

TABLE 1

1981 - 1982 AVERAGE VINYL CHLORIDE CONCENTRATIONS IN AMBIENT AIR (ppb)
BKK LANDFILL, WEST COVINA, CALIFORNIA

MONTH	STATION A		STATION B	
	1981	1982	1981	1982
January	36.0	5.7	31.0	6.1
February	30.6	5.6	27.3	6.1
March	25.3	5.8	23.2	6.1
April	20.0		17.8	
May	14.8		13.1	
June	12.1		10.0	
July	7.2	10.4	7.5	5.0
August	9.3	7.4	7.9	5.7
September	9.4	6.5	6.0	5.7
October	10.7	3.3	9.7	3.7
November	11.1	1.7	10.7	2.1
December	7.2	2.8	6.8	5.0
Annual Average	16.2	5.5	14.4	5.3

TABLE 2

AVERAGE ANNUAL VINYL CHLORIDE CONCENTRATIONS (PPB)

NEAR BKK LANDFILL

YEAR	STATION A	STATION B	STATION MY
1980	38.4	24.4	57.2
1981	16.2	14.4	24.1
1982	5.5	5.3	8.2
1983	2.8	3.6	4.2
1984	4.2	3.4	5.4
1985	3.5	2.3	5.4
1986	1.4	1.3	3.0
1987	1.7	1.2	2.6
1988	1.3	0.8	2.5
1989	0.8	0.8	0.9
1990 - 1994	0.05	0.01	0.25
1995 - 2009	0.05	0.01	0.25

TABLE 3

EXCESS CANCER RISK SINCE 1980

EXPOSURE SCENARIO 30 YEARS

STATION	EXPOSURE PERIOD	CONCENTRATION PPB	DURATION YEARS	EXCESS RISK x 10^{-5}	CUM EXCESS RISK x 10^{-5}
A	1980	38.4	1	8.79	8.79
	1981	16.2	1	3.71	12.50
	1982	5.5	1	1.26	13.76
	1983	2.8	1	.64	14.40
	1984	4.2	1	.96	13.36
	1985	3.5	1	.80	16.16
	1986	1.9	1	.44	16.20
	1987	1.7	1	.39	16.99
	1988	1.3	1	.30	17.29
	1989	0.8	1	.18	17.47
	1990-1994	0.05	5	.06	17.53
	1995-2007	0.05	15	.18	12.71
B	1980	34.4	1	7.87	7.87
	1981	14.4	1	3.30	11.17
	1982	5.3	1	1.21	12.38
	1983	3.0	1	.69	13.07
	1984	3.9	1	.89	12.96
	1985	2.3	1	.53	14.49
	1986	1.3	1	.30	14.79
	1987	1.2	1	.27	15.06
	1988	.8	1	.18	15.24
	1989	.5	1	.18	15.42
	1990-1994	.03	5	.03	15.09
	1995-2007	.03	15	.10	15.15

TABLE 3 (CONTINUED)
EXCESS CANCER RISK SINCE 1980
EXPOSURE SCENARIO 30 YEARS

STATION	EXPOSURE PERIOD	CONCENTRATION PPB	DURATION YEARS	EXCESS RISK $\times 10^5$	CUM EXCESS RISK $\times 10^5$
MW	1980	57.2	1	13.10	13.10
	1981	24.1	1	5.52	18.62
	1982	8.2	1	1.88	20.50
	1983	4.2	1	.96	21.46
	1984	5.4	1	1.24	22.70
	1985	5.4	1	1.24	23.94
	1986	3.0	1	.69	24.63
	1987	2.6	1	.60	25.23
	1988	2.5	1	.57	25.80
	1989	0.9	1	.21	26.01
	1990-1994	0.25	5	.29	26.30
	1995-2009	0.25	15	.86	27.16

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TABLE 4

EXCESS CANCER RISK SINCE 1972 FOR STATION A
EXPOSURE SCENARIO 30 YEARS

EXPOSURE PERIOD	CONCENTRATION PPB	DURATION YEARS	EXCESS RISK $\times 10^{-5}$	CUM EXCESS RISK $\times 10^{-5}$
1972	3.9	1	.59	.89
1973	11.6	1	2.66	3.55
1974	19.2	1	4.40	7.95
1975	27.1	1	6.21	14.16
1976	34.6	1	7.92	22.08
1977	38.4	1	8.79	30.87
1978	38.4	1	8.79	39.66
1979	38.4	1	8.79	48.45
1980	38.4	1	8.79	57.24
1981	16.2	1	3.71	60.95
1982	5.5	1	1.26	62.21
1983	2.8	1	.64	62.85
1984	4.2	1	.96	63.81
1985	3.5	1	.80	64.61
1986	1.9	1	.44	65.05
1987	1.7	1	.39	65.44
1988	1.3	1	.30	65.74
1989	.8	1	.18	65.92
1990-1994	.05	5	.06	65.98
1995-2001	.05	7	.08	66.16

TABLE 5

EXCESS CANCER RISK SINCE 1972 FOR STATION B

EXPOSURE 30 YEAR SCENARIO

EXPOSURE PERIOD	CONCENTRATION PPB	DURATION YEARS	EXCESS RISK $\times 10^{-5}$	CUM EXCESS RISK $\times 10^{-5}$
1972	3.8	1	.87	.87
1973	10.5	1	2.40	3.27
1974	17.1	1	3.92	7.25
1975	24.3	1	5.56	12.81
1976	31.2	1	7.14	19.95
1977	34.4	1	7.87	27.82
1978	34.4	1	7.87	35.70
1979	34.4	1	7.87	43.70
1980	34.4	1	7.87	51.44
1981	14.4	1	3.30	54.74
1982	5.3	1	1.21	55.95
1983	3.0	1	.69	56.64
1984	3.9	1	.89	57.53
1985	2.3	1	.53	58.06
1986	1.3	1	.30	58.36
1987	1.2	1	.27	58.63
1988	.8	1	.18	58.81
1989	.8	1	.18	58.99
1990 - 1994	.03	5	.03	59.02
1995 - 2001	.03	7	.05	59.07

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TABLE 6

EXCESS CANCER RISK SINCE 1972 FOR STATION M4

EXPOSURE SCENARIO 30 YEARS

EXPOSURE PERIOD	CONCENTRATION PPB	DURATION YEARS	EXCESS RISK $\times 10^{-5}$	CUM EXCESS RISK $\times 10^{-5}$
1972	5.7	1	1.31	1.31
1973	17.2	1	3.94	5.05
1974	28.6	1	6.55	11.60
1975	40.0	1	9.16	20.76
1976	51.5	1	11.79	32.55
1977	57.2	1	13.10	45.65
1978	57.2	1	13.10	58.75
1979	57.2	1	13.10	71.85
1980	57.2	1	13.10	84.95
1981	24.1	1	5.52	90.47
1982	8.2	1	1.88	92.35
1983	4.2	1	.96	93.31
1984	5.4	1	1.24	94.55
1985	5.4	1	1.24	95.79
1986	3.0	1	.69	96.48
1987	2.6	1	.60	97.08
1988	2.5	1	.57	97.65
1989	.9	1	.21	97.86
1990 - 1994	.25	5	.29	98.15
1995 - 2001	.25	7	.40	98.55

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