The Vinyl Institute
A Division of The Society of The Plastics Industry, Inc.

August 28, 1989

TO: The VI Health, Safety & Environment Committee

RE: California Air Resources Board Draft Report on Vinyl Chloride

Enclosed is material I received today from Peter de la Cruz relating to the development of a report by the California Air Resources Board outlining "Public Exposure To, Sources, and Emissions of Vinyl Chloride in California". As noted, the comment period on the preliminary draft closes on September 8th; a revised draft will then be developed with a 20-day comment period before the final draft is forwarded to the Scientific Review Panel.

If you have separately received this document and filed or plan to file comments, please let me know.

As the material I received notes that "landfills are the largest source category of vinyl chloride emissions in California", I have copied the appropriate VI Technical Committee contacts.

Meredith

Meredith N. Scheck
Assistant Director

MNS/pmb
enclosure
cc: C. Bush
J. Krokosky
R. Gottesman

CTL018228

OF CALIFORNIA
RESOURCES BOARD
STREET
X 2815
ENTO, CA 95812

George Deukmejian, Governor

August 1, 1989

Dear Sir or Madam:

Draft Report on Vinyl Chloride

As you requested, enclosed for your review and comment is the preliminary draft report on vinyl chloride.

Public review of this report includes two comment periods. The first comment period begins with the mailing of this report and you have until September 8, 1989 to submit your written comments to us. The staffs of the Air Resources Board (ARB) and Department of Health Services (DHS) will review and respond to all the comments received. Your comments and our responses to them will be compiled in Part C of the report and where appropriate, the report will be revised.

The revised report will be mailed out to you and other members of the public for a second and final review. It will include Parts A, B and C of the report as well as an Executive Summary which summarizes Parts A and B. At this stage, the ARB and DHS staff will only accept comments on the Executive Summary and any revisions made to the draft report. A 20 day comment period will be given for your review; comments received and our responses will then be incorporated as an addendum to Part C. After reviewing all public comments, the final draft report along with Part C will then be submitted to the Scientific Review Panel for its review.

The Scientific Review Panel has requested that all public comments be directed to the ARB during these two comment periods. Please note that, in accordance with this process, the Panel will not receive or consider any comments submitted directly to it, or received by the ARB after the close of each comment period.

In order for your comments to be considered on this version of the report, they must be received by September 8, 1989 and submitted to:

Mr. Robert Barham, Chief
Toxic Air Contaminant Identification Branch
Air Resources Board
Attn: Vinyl Chloride
P. O. Box 2815
Sacramento, CA 95812

If you have any questions concerning the report or the comment procedure, please contact Mr. Richard Corey of the Substance Evaluation Section at (916) 323-8543.

Sincerely,

Peter D. Venturini, Chief
Stationary Source Division

Enclosures

cc: Scientific Review Panel

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PRELIMINARY DRAFT

TECHNICAL SUPPORT DOCUMENT

PART A

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

REPORT TO THE AIR RESOURCES BOARD ON VINYL CHLORIDE

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July 1989

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I.

INTRODUCTION

Part A of this report is an evaluation of vinyl chloride's
es, emission sources, ambient and indoor air concentrations, and
pulation exposure in California. Also included are discussions of the
ysical properties and atmospheric persistence of vinyl chloride.
lifornia Health and Safety Code Section 39655 states that substances
st[redacted]by the U.S. Environmental Protection Agency (EPA) as hazardous
- [redacted]utants (Section 112 of the Clean Air Act) shall be identified as
xic air contaminants (TACs) by the Air Resources Board (ARB).
erefore, because the EPA has listed vinyl chloride as a hazardous air
llutant, the ARB is directed by statute to identify vinyl chloride as
TAC.

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

In 1978, the ARB adopted an ambient air quality standard for
yl chloride of 10 ppb for a 24-hour average. The standard
resented the limit of detection for vinyl chloride at the time it was
pted.

Vinyl chloride is an extremely volatile compound that is
marily used for the production of polyvinyl chloride (PVC). PVC is
ricated for use in several products of which many are used by the

construction industry. In California, the identified sources of vinyl chloride emissions are landfills, PVC production and fabrication facilities, and sewage treatment plants.

Available information indicates that landfills are the largest source category of vinyl chloride emissions in California. Vinyl chloride has been measured in the ambient air near hazardous waste and municipal waste landfills. Numerous studies have documented the presence of vinyl chloride in the landfill gas of these and other landfills, and have shown that vinyl chloride can be formed in landfills where chlorinated organic compounds have been disposed. Therefore, because disposal of such chlorinated compounds is prevalent, the staff recommends that all landfills (hazardous and municipal) in the state be regarded as potential vinyl chloride emission sources.

In this report, ambient monitoring data and meteorological data are used with an atmospheric dispersion model to estimate population exposure to vinyl chloride near two California landfills. The modeling results show that people living near these landfills are exposed to elevated levels of vinyl chloride. The results also imply that people residing near other landfills in the state may be exposed to elevated levels of vinyl chloride. In addition to estimating ambient air exposure, this report also evaluates indoor air exposure to vinyl chloride.

Based on limited monitoring data, indoor air exposure to vinyl chloride is probably not significant for the majority of the population. However, for people residing near landfills, inhalation of indoor air may represent the most significant source of vinyl chloride exposure. This is because vinyl chloride can migrate underground from landfills and accumulate in nearby structures. The concentrations of vinyl chloride measured in homes located near landfills have been reported to be several times greater than the corresponding ambient concentrations.

Part B

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1.0 Executive Summary

Vinyl chloride is a short-chain halogenated hydrocarbon used predominantly in the manufacture of polyvinyl chloride and various packaging and construction products. Vinyl chloride has a very low degree of acute toxicity, with two-hour inhalation LD_{50} values ranging from 27,419 ppm in mice to 236,215 ppm in rabbits and guinea pigs. Exposure to high concentrations can lead to narcosis, cardiovascular and respiratory irregularity, convulsions, cyanosis and death. Several human deaths have been attributed to occupational exposure to very high levels of vinyl chloride. Autopsies of these patients revealed congestion of the liver, spleen and kidneys. Acute toxicity symptoms are thought to occur above 100 ppm.

Chronic exposure of workers to vinyl chloride has been shown to lead to "vinyl chloride disease", characterized by occupational acro-osteolysis, vasospasm of the hands similar to Raynaud's syndrome, dermatitis, circulatory and central nervous system alterations, thrombocytopenia, splenomegaly and changes in liver function. Eight symptoms commonly reported by workers exposed to vinyl chloride (including dizziness, headaches and nausea) were observed even at dose levels below 50 ppm.

Vinyl chloride has been shown to induce cancer in animals in utero, but has not been shown to cause any other reproductive or

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developmental effects in rats, mice and rabbits. Epidemiologic 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.

The noncarcinogenic effects occur at concentrations near or above 10 ppm, which is greater than four orders of magnitude above possible general ambient levels in California (0.5 ppb). The noncarcinogenic effects also occur at concentrations greater than 3 orders of magnitude above the highest concentrations measured near landfills (10 ppb). Consequently, DHS staff do not expect noncarcinogenic adverse health effects to occur from acute or chronic exposures to vinyl chloride in ambient air.

The International Agency for Research on Cancer (IARC), the United States Environmental Protection Agency (EPA) and the California Department of Health Services (CDHS) have identified vinyl chloride as a chemical for which there is sufficient evidence of carcinogenicity in both humans and experimental animals. Chronic inhalation and oral exposures of rats, mice and hamsters to vinyl chloride have been associated with an increased incidence of malignant and benign tumors at several sites including the liver, lung, mammary gland and th nervous system. In humans,

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epidemiological studies of occupationally exposed workers have linked vinyl chloride exposure to development of a rare cancer, liver angiosarcoma, and have suggested a relationship between exposure and lung and brain cancers.

Although pharmacokinetic studies in humans exposed to vinyl chloride are rare, limited evidence indicates that, following inhalation of low levels of vinyl chloride (3 to 24 ppm), up to 71% (with a mean value of 42%) of the given dose may be absorbed. Vinyl chloride absorption appears to depend on its metabolism, which is a dose-dependent, saturable process. Due to saturation of the enzyme systems responsible for the metabolism of vinyl chloride (cytochrome P-450 and alcohol dehydrogenase), exposure to concentrations above approximately 250 ppm will not necessarily lead to an increasing incidence of tumor development. Metabolism of vinyl chloride leads to formation of chloroethylene oxide and chloroacetaldehyde, two reactive intermediates which undergo covalent binding to cellular macromolecules and are thought to be responsible for the toxic effects of vinyl chloride. These and other metabolites may be further metabolized and excreted in the urine. Unmetabolized vinyl chloride is eliminated primarily in exhaled air.

Vinyl chloride is mutagenic in both prokaryotic and eukaryotic test systems, with significantly greater genotoxicity seen after metab lic activation. DHS staff have found no evidence of a carcinogenic thresh ld level and because vinyl chlorid is

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mutagenic, the staff recommends that vinyl chloride be considered as not having a threshold for carcinogenicity.

Several animal carcinogenicity and human epidemiological studies f occupationally exposed workers have been analyzed for risk assessment purposes. Although actual exposure levels are not known, exposure estimates have been used to evaluate the Waxweiler et al. (1976) study of vinyl chloride workers. Based on these estimates, DHS staff has calculated that a lifetime exposure to 0.485 ppb might result in an incremental individual cancer risk of 1×10^{-6} (assuming liver, brain and lung cancer are all related to vinyl chloride exposure). This yields a risk estimate of 2.1×10^{-6}/ppb. In the case that only liver cancer is assumed to be linked to exposure, a lifetime exposure to 1.0 ppb may be expected to result in a risk of 1.0×10^{-6}. Due to inadequate exposure data, follow-up time and other methodological problems, DHS staff suggest that the human risk estimates be used only for comparative purposes. Evaluation of animal experiments by the linearized multistage model yields a range of human risks spanning from 1.8×10^{-3}/ppb to 3.9×10^{-6}/ppb, with most estimating a risk of between 10^{-4} and 10^{-5}/ppb. Evaluation of animal tumorigenicity data indicates that vinyl chloride's carcinogenic potency is dependent on sex, tumor site and age of exposure. Taking these factors int account, DHS staff believe that the human risk estimates are consistent with those obtained for laboratory animals. The staff of DHS recommends that the animal data be used to evaluate the risks resulting from vinyl chlorid exposure. Consequently, the

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range of risks estimated from analyses of animal studies and recommended by DHS for regulatory purposes lie between 3.9 x 10^{-6}/ppb and 1.8 x 10^{-3}/ppb.

Vinyl chloride has not been detected in the ambient air of California (limit of detection = 0.5 ppb) except at certain "hot spots". Air Resources Board (ARB) staff has monitored vinyl chloride emissions from the BKK hazardous waste site in West Covina and the OII landfill in Monterey Park. Estimates of peak exposure concentrations for maximally exposed receptors range from 2 to 10 ppb at the BKK landfill and from 0.6 to 9 ppb at the OII site. Air Resources Board staff has estimated that between 17,000 and 131,000 individuals may be exposed to 1 ppb at the BKK site. A lifetime exposure of 131,000 residents to 1 ppb would be associated with an upper bound estimate of 0.5 to 236 excess cancer cases. The calculations represent the upper range of plausible excess cancer risk: the actual risk, which cannot be calculated, may be insignificant. Based on the finding of vinyl chloride-induced carcinogenicity and the results of the risk assessment, DHS staff finds that vinyl chloride is an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.