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# Air Pollution Assessment of Vinylidene Chloride

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MITRE

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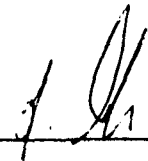
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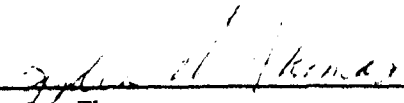
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# ABSTRACT

Vinylidene chloride is toxic to laboratory animals and can be fatal at sufficiently high dose levels. Liver is the prime target organ of vinylidene chloride in mammals. Hepatic injury can occur rapidly after inhalation exposure. Chronic exposure to low levels of vinylidene chloride can result in liver and kidney damage. Vinylidene chloride is a central nervous system depressant reported to have a narcotic effect. Mutagenicity of vinylidene chloride in microorganisms indicates a need for investigation of its carcinogenicity.

The population at risk due to vinylidene chloride exposure is composed primarily of workers in industrial or commercial operations manufacturing or using it. Airborne emissions of vinylidene chloride are not likely to pose a significant risk to the general population. Emissions during production, storage, and transport can be controlled by methods similar to those planned for control of vinyl chloride.

## ACKNOWLEDGEMENTS

# TABLE OF CONTENTS

## LIST OF TABLES

Table Number

## LIST OF FIGURES

Figure Number

TABLE OF CONTENTS (CONCLUDED)

LIST OF FIGURES (CONCLUDED)

I. SUMMARY, CONCLUSIONS, AND RECOMMENDATION

Vinylidene chloride, also known as 1,1-dichloroethene or 1,1-dichloroethylene, is used in combination with other monomers to produce co-polymers such as Saran B (vinylidene chloride - vinyl chloride), Saran C (vinylidene chloride - alkyl acrylate co-polymers), and Saran F (vinylindene chloride - acrylonitrile co-polymers). Saran, a Dow Chemical Corporation trademark, is now also used as a generic term for high vinylidene chloride content polymers. Vinylidene chloride is also used as an intermediate in the manufacture of 1,1,1-trichloroethane.

Vinylidene chloride is a clear liquid at room temperature. It is insoluble in water, but soluble in most other polar and non-polar solvents. Vinylidene chloride is a very volatile compound having a boiling point, at atmospheric pressure, of 31.56°C. It is flammable at vapor concentrations between 7 and 16 percent by volume in air. In the presence of air or oxygen, vinylidene chloride forms a highly explosive peroxide compound at temperatures as low as -40°C. Any dry composition containing more than 15 percent peroxide detonates from a slight mechnical shock or heat. For this reason, a peroxide formation inhibitor is usually added to vinylidene chloride prior to storage.

The toxicity of vinylidene chloride and to a lack of toxicological investigation by independent researchers. In view of toxic symptoms observed in animals exposed to vinylidene chloride and the similarity of this compound to vinyl chloride, the need for more toxicological

1

information concerning vinylidene chloride is urgent.

Vinylidene chloride has been found to be present in polyvinyl chloride manufacturing plants at concentrations of approximately 5 ppm. Workers in such plants have been reported to have an increased incidence of impaired liver function, angiosarcoma and overall malignancies. It is not possible to determine the extent to which vinylidene chloride present in workroom atmospheres may have contributed to these toxic syptoms generally attributed to vinyl chloride.

Vinylidene chloride is toxic to laboratory animals and can be fatal at sufficiently high dose levels through inhalation or oral exposure. The 24-hour oral $LD_{50}$* for a single dose of vinylidene chloride in rats is approximately 1,550 mg per kg body weight. The $LC_{50}$** for inhalation exposure of rats to vinylidene chloride has been reported as 15,000 ppm for deaths within 24 hours after exposure and 6,350 ppm for deaths within 14 days after exposure.

The acute toxicity of vinylidene chloride is similar to that for carbon tetrachloride and significantly greater than the acute toxicity of vinyl chloride. A threshold limit of 10 ppm is recommended by the American Conference of Governmental Industrial Hygienists for prevention of liver or kidney damage in humans from acute exposure to vinylidene chloride. No OSHA standard has been established.

The susceptibility of animals to vinylidene chloride poisoning is increased during fasting or at night.

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*LD50:   Mean lethal dose
**LC50:   Mean lethal concentration

Based on animal experiments, chronic exposures to low levels of vinylidene chloride would be expected to result primarily in liver and kidney damage. Vinylidene chloride is a potent hepatotoxin. the hepatotoxicity of vinylidene chloride is, qualitatively and quantitatively, roughly comparable to that of carbon tetrachloride. Due to its structural similarity to vinyl chloride and to some indications of carcinogenicity in laboratory animals, vinylidene chloride may be suspected of being a human carcinogen. Long term carcinogenicity bioassays of vinylidene chloride are strongly recommended.

Human exposure to vinylidene chloride is expected to occur mainly through inhalation. Animal studies have shown that toxic symptoms can result from chronic exposures to low levels of vinylidene chloride. During continuous 90 day exposure, fatalities have been observed in rats, guinea pigs and monkeys exposed to 5 ppm vinylidene chloride.

The liver is the prime target organ of vinylidene chloride in most laboratory mammals and, apparently, in humans. Hepatic injury occurs quite rapidly after inhalation exposure (2 to 4 hours of exposure to 2,000 ppm in rat). Single oral doses of 4.0 mg per kg body weight were hepatotoxic to cats. Morphological changes in liver cells and large quantities of neutral fat within the liver indicate vinylidene chloride hepatotoxicity. The most sensitive indicators of vinylidene chloride injury to liver are disruptions of normal microsomal enzyme activity.

3

Kidney damage resulting from vinylidene chloride exposure has been observed in experimental animals. Vinylidene chloride is a central nervous system depressant reported to have a narcotic effect on mice, cats, rabbits and guinea pigs at vapor concentrations from 12 to 1125 ppm. Lung damage has occasionally been observed during prolonged inhalation exposures.

Vinylidene chloride has been shown to be mutagenic in microbial systems mediated by microsomal enzymes from various organs. Mouse liver, kidney and lung fractions converted vinylidene chloride into mutagenic matabolites in vitro. Although mutagenicity in micro-organisms cannot be assumed to indicate carcinogenicity in humans, mutagenicity tests are currently used to select environmental chemicals for carcinogenicity tests. On this basis, the mutagenicity of vinylidene chloride in microorganisms indicates a need for investigating its carcinogenicity. Preliminary reports from Dr. P. L. Viola, one of the first researchers to associate vinyl chloride with liver tumors in animals, indicate that inhalation exposure to vinylidene chloride may cause liver tumors in rats.

The current domestic production of vinylidene chloride is approximately 260 million pounds per year. An industrial growth rate of 5 to 10 percent has been predicted for the next 5 years.

Vinylidene chloride monomer is produced at only three locations. Total vinylidene chloride emissions to the environment have been estimated to be between 3 and 4 million pounds per year. Due to

vinylidene chloride's volatility most of the emissions will be
rapidly airborne even if they originated as spills of liquid monomer.
If the predicted improvements are assumed for the PPG plant at Lake
Charles, Louisiana, the emissions at ground level 500 m downwind
range at all facilities from 0.16 $mg/m^3$ to 0.31 $mg/m^3$ for a one hour
exposure and 3.72 $mg/m^3$ to 7.32 $mg/m^3$ for a 24 hour exposure. Without
the control technology at the PPG plant, the recommended 8 hour dose
of 40 $mg/m^3$ is reached in 13.1 hours, making it possible to receive
almost twice the recommended dose in a 24 hour period. This suggests
that the controls are imperative.

The potential hazard from emissions of vinylidene chloride is
probably not significant for the general population. The population
at risk due to vinylidene chloride exposure is composed primarily of
workers in industrial or commercial operations manufacturing or using
vinylidene chloride. Long term low level exposures may prove to be
significant. Control procedures are needed to ensure that the time
weighted average concentration in workroom atmospheres does not
exceed 10 ppm.

Airborne emissions of vinylidene chloride during monomer pro-
duction, storage and transport can be controlled by methods similar
to those planned for control of vinyl chloride emissions, since the
compounds are chemically similar and are generally both present
during polymerization. These methods include proper design of vents
and vapor condensing apparatus, proper location of vents within

5

the plants, and proper storage, handling and transporatation procedures.
The monitoring of vinylidene chloride emissions during distribution
operations is recommended.

Vinylidene chloride may be present in liquid effluent from produc-
tion plants. Since vinylidene chloride has low solubility in water,
it would soon vaporize. Wastewater could therefore become a source
of airborne emissions. Monitoring of liquid effluent from plants
manufacturing or using vinylidene chloride is recommended to determine
the need for control.

II. AIR POLLUTION ASSESSMENT REPORT

   A. PHYSICAL AND CHEMICAL PROPERTIES

   1. Physical Properties

      Vinylidene chloride is also known as 1,1-dichloroethene,

1,1-dichloroethylene, VDC, vinylidene chloride monomer, and vinylidene

dichloride. It is a clear liquid with a low viscosity at room

temperature (1-5). It possesses a pleasant, sweet odor similar to

those of carbon tetrachloride and chloroform (1,3,4). Most persons

find that at concentrations of 1000 ppm in air, it has a mild but

definite odor. Many people can detect levels of 500 ppm. Vapors

containing decomposition products have a disagreeable odor and can be

detected at concentrations of vinylidene chloride considerably less

than 500 ppm (4). The odor is generally inadequate to prevent

excessive exposures since the threshold limit value has been set by

the American Conference of Governmental Industrial Hygienists at 10

ppm or approximately 40 mg/m$^3$ (6).

      Vinylidene chloride is a very volatile compound (660.4 mm Hg)

when compared to the standard in the Federal Code of Regulations of

77.6 mm Hg at storage conditions, and hence must be handled with

extreme care. Volatility is such that vapor concentrations exceeding

2 percent (20,000 ppm) may readily occur in instances of a large

spill in a poorly ventilated area (5).

      Vinylidene chloride is rated as a high fire hazard (7) due to its

low flash point and as a moderate explosion hazard (8). It is

7

flammable at vapor concentrations between 7 percent and 16 percent by volume in air. Vapors of the liquid monomer, once ignited, readily burn (5). It ignites less easily than benzene, gasoline, or common hydrocarbons, but after a short period, it burns in a similar fashion (4). Vinylidene chloride is insoluble in water. However, it is soluble in most other polar and non-polar solvents (2,3).

Physical properties of vinylidene chloride are listed in Table I (1,3,4,7,9).

### 2. Chemical Properties

Vinylidene chloride forms an azeotrope with 6 percent methanol. Distillation of the azeotrope, followed by extraction of the methanol with water, yields pure vinylidene chloride (10).

Polymers containing vinylidene chloride show thermal stability and impermeability, hence vinylidene chloride is used in combination with other monomers to produce copolymers with desirable properties (3). Pure vinylidene chloride polymer is flexible at $70^{\circ}C$, but at $180^{\circ}C$ forms a thermoplastic solid. If the crystals are not oriented, this polymer has a tensile strength of about 8000 psi but if they are oriented, the tensile strength increases to 60,000 psi (11).

The homopolymer, Saran A, is difficult to fabricate and for this reason has not been extensively used. Many copolymers are possible, but only three are of commercial interest: Saran B, vinylidene chloride-vinyl chloride copolymers; Saran C, vinylidene chloride-alkyl acrylate copolymers; and Saran F, vinylidene chloride-acrylonitrile

TABLE I

PHYSICAL PROPERTIES

Molecular Formula:           $CH_2 = C\ Cl_2$

Structural Formula:

$$\begin{array}{c} Cl \\ Cl \end{array}\!\!\!\!\!>\!\!C ===== C\!\!<\!\!\!\!\!\begin{array}{c} H \\ H \end{array}$$

Physical State:              Colorless liquid

Molecular Weight:            96.95

Freezing and Melting Point:  -122.5°C

Boiling Point:

| °C | at | Pressure (mmHg) |
|---|---|---|
| 31.56 | | 760 |
| 14.43 | | 400 |
| -1.78 | | 200 |
| -16.04 | | 100 |
| -25.46 | | 60 |
| -32.44 | | 40 |
| -43.31 | | 20 |
| -53.10 | | 10 |
| -61.94 | | 5 |
| -79.54 | | 1 |

TABLE I (Continued)

Vapor Density:                                    3.34 (air = 1)


Liquid Density:          Temperature ($^{o}$C)   Density (gm/cc)

                              -20                    1.2902

                               0                     1.2517

                              +20                    1.2132



Percent in "Saturated" air:        78%



Density of "Saturated" air:        2.8 (air = 1)



Flash Point (Cleveland Open Cup):   5°F (4)
                                    3°F (3)
   (Tag Closed Cup):               55°F (4)
                                   -2°F (3)



Explosive Limits:                  7.3 - 16.0 at 28°C
   (% by volume)                   5.6 - 11.4
                                   6.5 - 15.5



Autoignition Temperature:          1058°F
                                   1031°F



Latent Heat of Vaporization    6, 328 $\pm$ .3% cal./mole at 25$^{o}$C:
                               6, 257 $\pm$ .3% cal./mole at BP:

10

TABLE I (Continued)

Latent Heat of Fusion:                          1,557 cal./mole


Heat of Polymerization ( Hp):                   -18.0 $\pm$ .9k cal./mole at 25°C


Heat of Combustion, Liquid Monomer
( Hc):                                          261.93 $\pm$ .3k cal./mole


Heat of Formation ( Hf):
    Liquid Monomer:                             -6.0 $\pm$ .3k cal./mole
    Gaseous Monomer:                            + .3 $\pm$ .3k cal./mole


Heat Capacity Liquid Monomer (Cp):              26.745 cal./mole/deg.
                                                at 25.15°C


Heat Capacity Ideal Gas State (Cp):             16.04 cal./mole/deg.
                                                at 25.15°C


Critical Temperature ($T_c$):                   222°C

Critical Volume ($V_c$)                         219 cc/mole

Critical Pressure ($P_c$):                      51.3 atmospheres


Index of Refraction ($n_d^{20}$):               Temperature (°C)

|     |         |
| --- | ------- |
| 10  | 1.43062 |
| 15  | 1.42777 |
| 20  | 1.42468 |

TABLE I (Concluded)

| Absolute Viscosity: | Temperature (°C) | Viscosity (cps) |
|---|---|---|
| | -20 | 0.4478 |
| | 0 | 0.3939 |
| | +20 | 0.3302 |

Solubility of Vinylidene Chloride
in Water:                             .021 gm/liter
at 25°C Wt %:                     .055 gm/liter

Solubility of Water in Vinylidene
Chloride Monomer:                       .035
at 25°C Wt %:

| Solubility Profile: | $H_2O$ – insoluble | Acetone – soluble |
|---|---|---|
| | Ethanol – soluble | Benzene – soluble |
| | Diethyl Ether – very soluble | Chloroform – very soluble |

Dielectric Constant:                          4.67 at 16°C

1 mg/liter ≡ 252 ppm and 1 ppm ≡ 3.97 mg/m$^3$ at 25°C, 760 mm Hg

copolymers (3). In the U.S., Saran is now a generic term for high vinylidene chloride content polymers regardless of composition, in addition to being a Dow trademark.

Vinylidene chloride polymerizes by both ionic and free radical reactions. However, free radical reactions predominate. When compared with other monomers, vinylidene chloride is of average reactivity. The clorine substituents stabilize the radicals in the intermediate state of an addition reaction. Since the chlorine atoms are strongly electrophilic, they polarize the double bond and make it susceptible to anionic attack. For the same reason, a carbonium ion intermediate would not be favored (3). Free radical polymerization requires the presence of a small amount of an initiator such as peroxide. This radical adds to a monomer molecule and generates another free radical (Rad') (12).

$$\text{Rad}^\cdot + CH_2 = C \overset{Cl}{\underset{Cl}{\big<}} \longrightarrow \text{Rad} - CH_2 - C^\cdot \overset{Cl}{\underset{Cl}{\big<}} \quad \text{Chain initiating steps}$$

Peroxide     Rad'

$$\text{Rad-}CH_2 - C^\cdot \overset{Cl}{\underset{Cl}{\big<}} + CH_2 = C \overset{H}{\underset{R}{\big<}} \longrightarrow \text{Rad-}CH_2 - \overset{Cl}{\underset{Cl}{\overset{|}{C}}} - \overset{H}{\underset{R}{\overset{/}{C^\cdot}}} = \text{etc.} \quad \begin{array}{l}\text{polymer-}\\\text{ization.}\end{array}$$
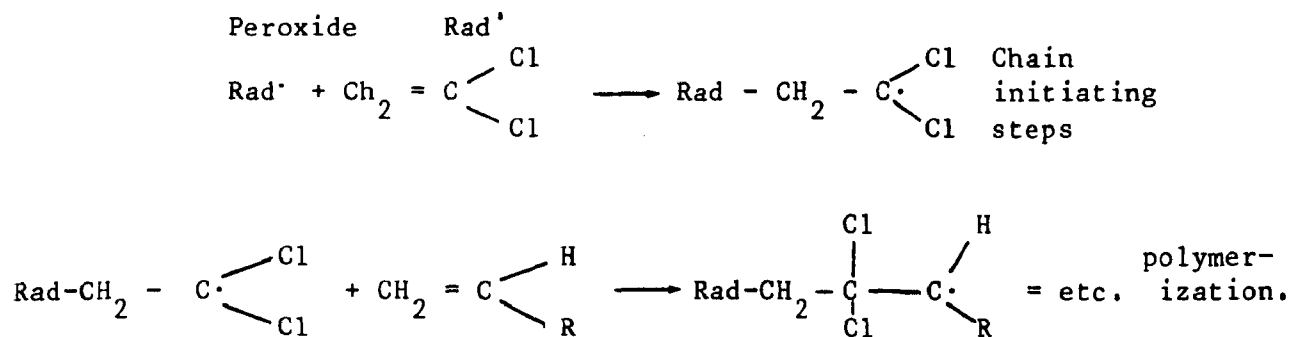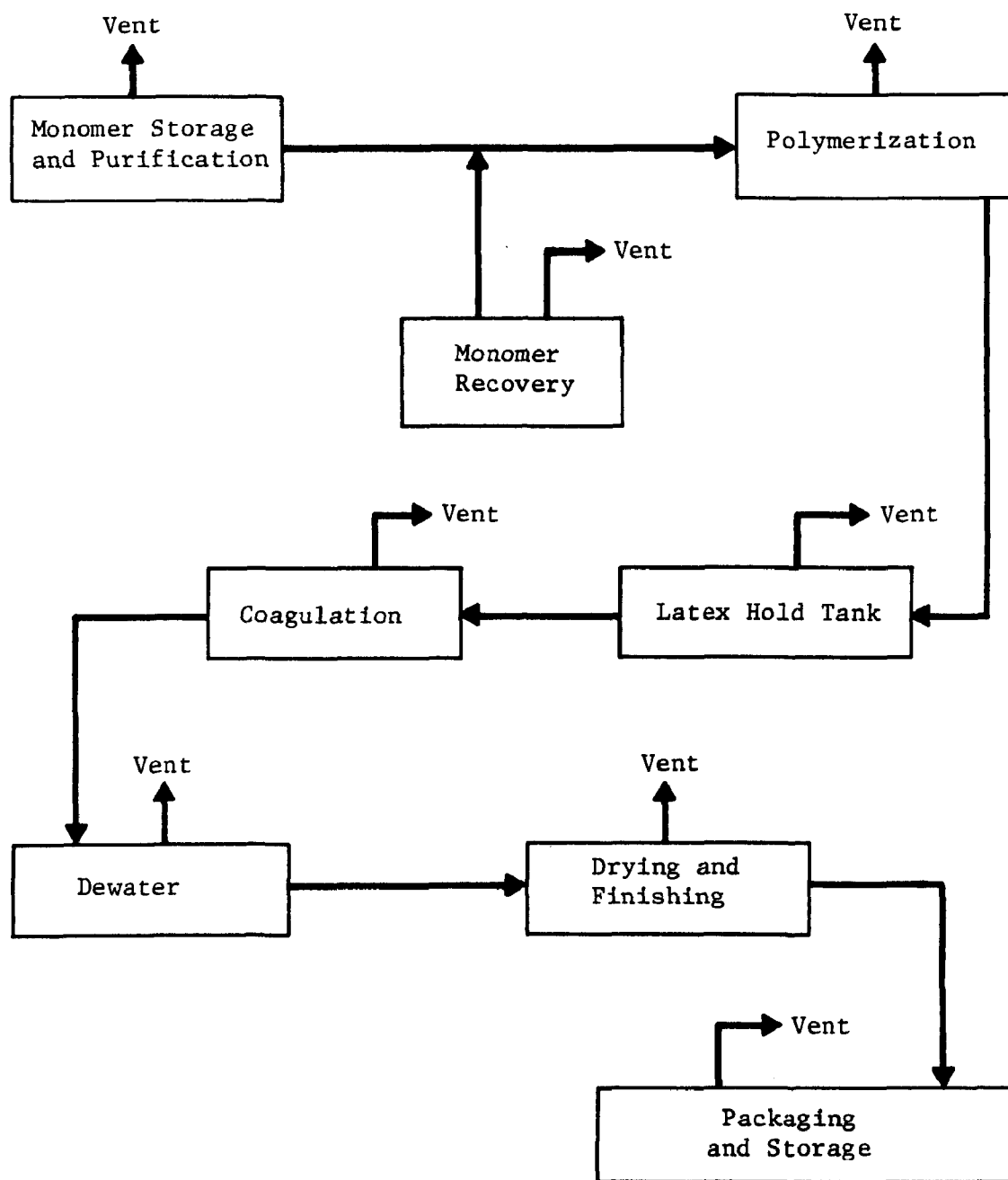
The 1,1-disubstitution causes significant steric interactions in the polymer (13) as evident from the heat of polymerization (see Table I). If this is corrected for the heat of fusion, a value significantly less

13

than the theoretical value of 20 Kcal/mole for the conversion of a double bond to two single bonds is attained. The steric strain does not appear to affect the addition step nor does it favor depolymerization (3).

Commercial polymerization procedures are dictated to some extent by the desired polymer characteristics. Emulsion polymerization and suspension polymerization are the preferred industrial processes. The emulsion polymerization procedure is used directly to make a latex coating or the latex may be recovered in dry form. This process has the advantage over suspension polymerization of shorter reaction times (7-8 hr. at 30 C) and a high degree of control over the polymer composition. The suspension polymerization procedure, used for molding and extrusion resins, occurs more slowly but requires fewer ingredients. With suspension polymerization, improved stability and decreased water sensitivity are obtained (3). Figures 1 and 2 illustrate representative process diagrams (14).

Inhibitors are generally added to vinylidene chloride monomer preparations shortly after production. These inhibitors prevent polymerization and preserve the quality of the product during storage and shipment. There are two inhibitors currently in use: phenol at a concentration of 0.6 to 0.8 percent by weight, or MEHQ (monomethyl ether of hydroquinone) at 100 ppm. MEHQ inhibited vinylidene chloride is generally preferred for most applications since this level of MEHQ is generally overcome by polymerization initiation. To use the

**FIGURE 1**
**SARAN EMULSION RESINS**

Vent

Vent

Monomer Storage
and Purification

Polymerization

Vent

Monomer
Recovery

Vent

Vent

Coagulation

Latex Hold Tank

Vent

Vent

Dewater

Drying and
Finishing

Vent

Packaging
and Storage

Source:  Strasser, J.P., Dow Chemical, U.S.A., personal communication.

**FIGURE 2**
**SARAN SUSPENSION RESINS**
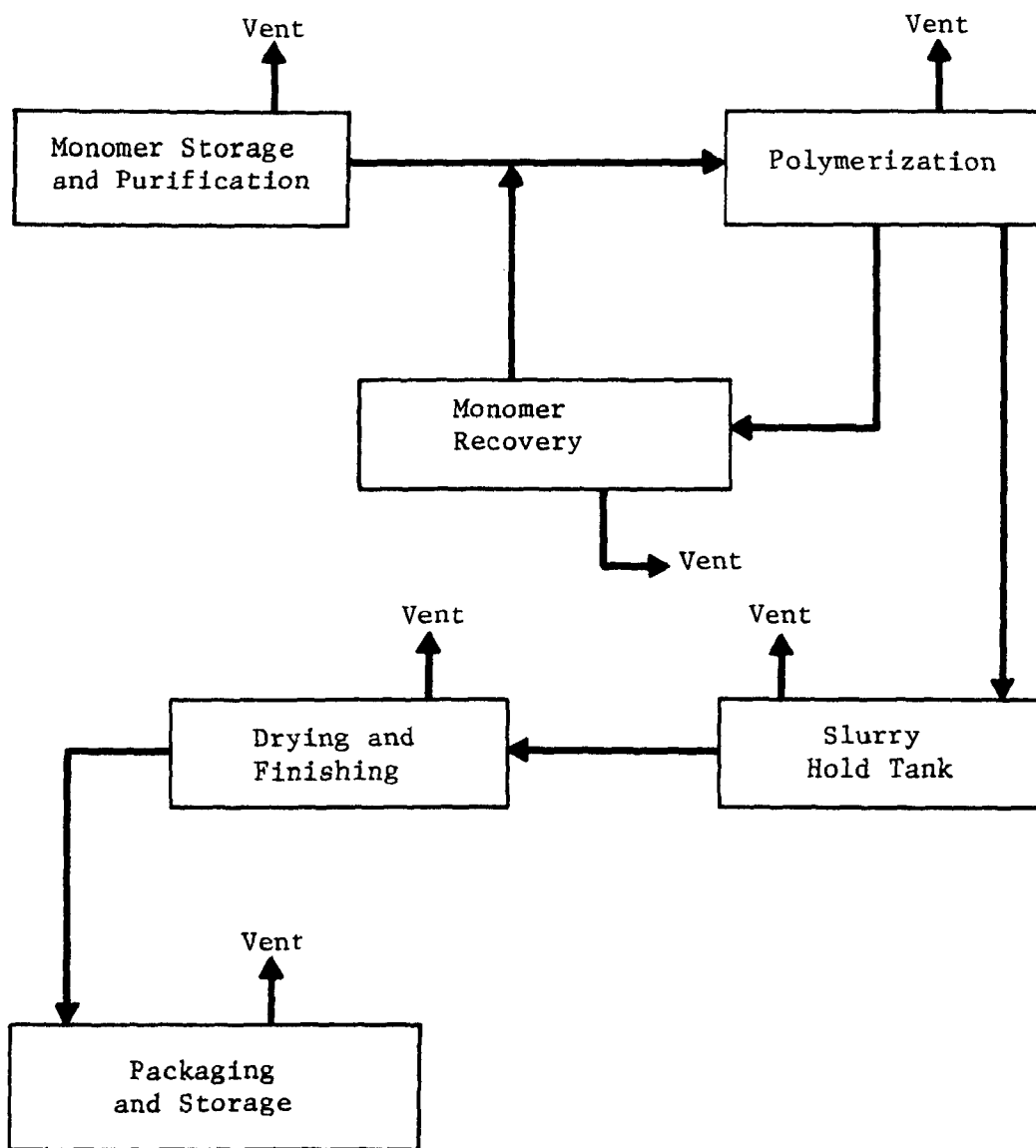


Source: Strasser, J. P., Dow Chemical, U.S.A., personal communication.

phenol inhibited vinylidene chloride, however, the phenol must first

be removed by alkali extraction or distillation (3,4).

Good discussions of the chemistry and properties of polymers con-

taining vinylidene chloride can be obtained in (3,7,15,16).

Vinylidene chloride, in the presence of air or oxygen, without

inhibitor, forms a complex peroxide and polyvinyl chloride at tempera-

tures as low as $-40^\circ$C. The peroxide compound $H_2 C \overset{\diagdown}{\underset{O}{\diagup}} C \overset{\diagup Cl}{\diagdown Cl}$ is

highly explosive. Any dry composition containing more than 15 percent

peroxide detonates from a slight mechanical chock or heat (3).

Reaction products formed with ozone are especially dangerous. The

decomposition end products of vinylidene chloride peroxides are

formaldehyde, phosgene and hydrochloric acid. The presence of a

sharp acrid odor thus indicates oxygen exposure and the possibility

of peroxides (5).

The presence of peroxides can be confirmed by the liberation of

iodine from a slightly acidified dilute potassium iodide solution.

Any formation of insoluble polymer also indicates peroxide formation

(3).

Vinylidene chloride which contains peroxides may be purified by

washing it several times with either 10 percent sodium hydroxide at

$25^\circ$C or with a fresh 5 percent sodium bisulfite solution (6).

Vinylidene chloride is quite reactive toward the hydroxyl group

and oxygen radical (half life of <1 day), but not $CO_2$ (half life of

about 3 years). Hence, any releases of vinylidene chloride to the

environment would not persist long prior to oxidation. The resulting

products are phosgene and formaldehyde (17).

Under appropriate conditions, vinylidene chloride also reacts

with hydrogen bromide (18), di-sodium cysteinate (19), sodium n-

propylthiolate (20), and hydrogen chloride (16). The only reaction

likely to be of potential environmental significance is that with

hydrogen chloride producing methyl chloroform.

B. EFFECTS

1. Humans

No epidemiological or occupational data are available for human exposures to environments in which the major contaminant was vinylidene chloride alone. Epidemiological data has been reported for workers exposed to vinyl chloride and traces of vinylidene chloride in polyvinyl chloride manufacturing plants (21), but this would not be the only place workers are exposed to vinylidene chloride. No data has been released concerning medical histories of workers exposed to vinylidene chloride in vinylidene chloride monomer plants or in facilities where vinylidene chloride homopolymers or copolymers (Saran B, Saran C, and Saran F) are produced. Health records of these workers could be extremely helpful in drawing conclusions concerning the effects of vinylidene chloride on humans.

Workers in polyvinyl chloride manufacturing plants have a slight exposure (approximately 5 ppm) to vinylidene chloride (21). Concentrations of vinyl chloride at these plants ranged up to 300 ppm (21). Peak exposures of certain individuals to vinyl chloride may often have exceeded 1,000 ppm in the production facility (22). According to data obtained from industry and the National Institute of Occupational Safety and Health (NIOSH), workers at a polyvinyl chloride facility who developed angiosarcoma of the liver were exposed to both vinyl and vinylidene chloride (23). Vinyl chloride and vinylidene chloride are structurally related compounds. Both are chlorinated

19

thenes subject to free radical polymerization, Vinylidene chloride
is more reactive in free radical polymerization than vinyl chloride,

A retrospective study (21) revealed several statistically sig-
nificant clinical changes from chronic exposure to vinyl chloride and
smaller amounts of vinylidene chloride, A time-weighted doze-response
relationship was shown for some indices of hepatic injury, Concentra-
tions of vinyl chloride were much higher than vinylidene chloride due
to the much larger quantities of vinyl chloride used and its greater
volatility,

Some industrial workers exposed to vinyl chloride levels above
200 pppm TWA* and low levels of vinylidene chloride had an apparent
increase in overall lamignancy rate (24), Angiosarcoma and partial
cirrhosis of the liver have been found in workers exposed to vinyl
chloride-vinylidene chloride atmospheres (25),

For many years, it had been suggested that vinylidene chloride
concentrations in workroom air be less than 25 ppm (9), More recently
a threshold limit of 10 ppm has been reecommended (6), The American
Conference of Governmental Industrial Hygienists (6) believes the 10
ppm level is low enough to prevent liver and kidney injury,

vinylidene chloride has significantly greater acute toxicity
than vinyl chloride (26), Acute exposure to high concentrations of

*TWA: estimated time-weighted average concentrtions for an eight
hour day,

20

vinylidene chloride would be expected to result primarily in central nervous system depression and an associated symptom of drunkenness which would progress to unconsciousness (9). These symptoms would be produced rapidly at concentrations on the order of 4,000 ppm. If the exposure is of short duration, complete recovery from this anesthetic effect may be expected.

Liquid vinylidene chloride was irritating to the skin after direct contact of only a few minutes. The phenolic inhibitor content of liquid vinylidene chloride may be responsible. Where leaks occur, vinylidene chloride will evaporate and the phenolic inhibitor may accumulate until it reaches a concentration capable of causing local burns.

Acute exposure to vinylidene chloride may cause moderate eye irritation. It affects human corneal mucosa at concentrations of 0.1 mg/l (4 ppm) (27).

An accidental acute exposure of two Germans to an aqueous dispersion of vinylidene chloride has been reported (28). Within 8 to 30 hours of exposure, both patients developed sensory disturbances in the trigeminal area of the face, mouth and tongue. The functional disorders involved mainly the interneuronal system and were apparently caused by irreversible lesions of the trigeminal nerve.

Based on animal experiments, chronic exposures to low levels of vinylidene chloride would be expected to result primarily in liver and kidney damage (29). Vinylidene chloride is a potent hepatotoxin

(23). The hepatotoxicity of vinylidene chloride is, qualitatively and quantitatively, roughly comparable to that of carbon tetrachloride, although the mechanism of of hepatotoxicity appears to be different (30). Because there is so little information on toxicology of vinylidene chloride and because vinylidene chloride appears to be toxicologically similar to carbon tetrachloride, industry has often relied on carbon tetrachloride data for guidance in handling vinylidene chloride. Vinylidene chloride, however, has a much higher vapor pressure than carbon tetrachloride and is hence more volatile.

Due to its structural similarity to vinyl chloride and to some indications of carcinogenicity in laboratory animals, vinylidene chloride may be suspected of being a human carcinogen. Professor Cesare Maltoni, who in 1972 linked vinyl chloride with angiosarcoma, is suspicious of all compounds with a carbon-chloride link (31). Despite these suspicions, there has so far been no evidence of a relationship between vinylidene chloride and human cancer.

2. Animals

Acute Toxicity

Vinylidene chloride is toxic to laboratory animals through inhalation or oral exposure and can be fatal at sufficiently high dose levels. The acute toxicity of vinylidene chloride is similar to carbon tetrachloride and significantly greater than the acute toxicity of vinyl chloride.

Mortality in Sprague-Dawley rats exposed to vinylidene chloride was observed by Siegel et al. (32) during both a four hour inhalation exposure and a subsequent two week observation period. The $LC_{50}$* was determined to be 6,350 ppm. Starved rats are much more sensitive to vinylidene chloride toxicity than fed rats. Jaeger et al. (33) found that the 24 hour $LC_{50}$ for Holtzman rats fed ad libitum was 15,000 ppm while the 24 hour $LC_{50}$ for 18 hour (overnight) fasted rats was 600 ppm (see Figure 3). The minimum lethal concentration was 200 ppm for fasted rats and 10,000 ppm for fed rats.

The 24-hour oral $LD_{50}$ for vinylidene chloride in rats is approximately 1,550 mg per kg body weight (34). Adrenalectomy 10-14 days prior to administration of vinylidene chloride reduced the 14 hour oral $LD_{50}$ to 84 mg/kg.

### Chronic Inhalation Exposure

Human exposure to vinylidene chloride is expected to occur mainly through inhalation. Animal studies have shown that toxic symptoms can result from sublethal exposures to vinylidene chloride.

For example, rats exposed to a vapor concentration of 200 ppm for 20 exposures of six hours each developed a slight nasal irritation (35). Rats exposed to 500 ppm vinylidene chloride showed symptoms of nasal irritation and retarded weight gain. Histological examination revealed liver cell degeneration (35).

*$LC_{50}$: mean lethal concentration.

**FIGURE 3**
**EFFECT OF FASTING ON 24 HOUR MORTALITY FOLLOWING**
**A 4 HOUR VINYLIDENE CHLORIDE EXPOSURE**
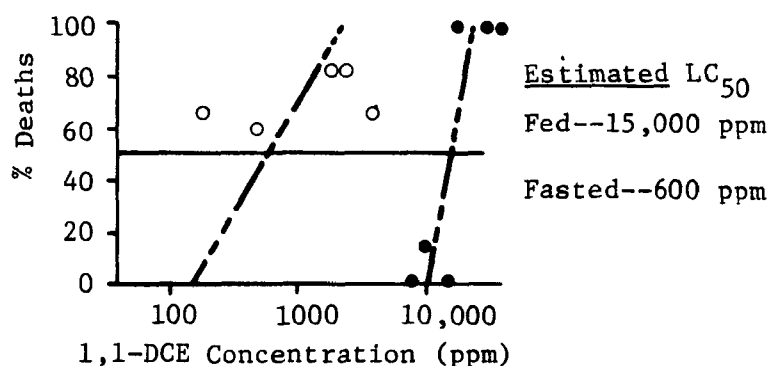


Each point represents the percent of animals dead after exposure to vinylidene chloride with group sizes of five or six. The broken lines represent an approximation of the dose-response curve. The vinylidene chloride concentration is plotted logarithmically.

Source: Jaeger, R. J., R. B. Conolly, and S. D. Murphy, _Experimental and Molecular Pathology_ 20:187-198, 1974.

Rabbits, rats, dogs, monkeys and guinea pigs (29) were exposed to repeated daily eight hour exposures of 395 mg/m$^3$ (100 ppm) for five days per wek over a period of six weeks. No animals died. The rabbits and monkeys lost weight and one rat had bloody urine.

During continuous 90 day exposure to 189 mg/m$^3$ (67 ppm), seven out of 15 guinea pigs died between the fourth and ninth day of exposure and three out of nine monkeys dies on days 26, 60 and 64. Dogs and monkeys lost weight. Rats gained less than controls. Gross examination revealed mottled livers. The hepatic changes in dogs, monkeys and rats and the renal changes in rats were considered to be a direct result of vinylidene chloride exposure. Histological examination showed morphological changes in both liver and kidney of rats and livers only of dogs and monkeys. One adrenal gland from a dog contained a cortical adenoma. There were also nonspecific inflammatory changes in lungs of a majority of animals. Activity of microsomal enzymes (liver alkaline phosphatase and serum glutamic-pyruvic transaminase) were increased in rats and guinea pigs (29).

During continuous 90 day exposure to 101 mg/m$^3$ (25 ppm), three out of 15 guinea pigs died between the third and sixth exposure days and two out of three monkeys on days 39 and 47. At an exposure level of 61 mg/m$^3$ (15 ppm), three out of 15 guinea pigs died on the third and fourth days of exposure. At an exposure level of 20 mg/m$^3$ (5 ppm), two out of 45 rats, two out of 45 guinea pigs and one out of 21 monkeys died. The dogs lost weight and rats gained less than controls.

25

Gross examination revealed mottled livers in about one third of the
animals in all species. Histological examinations revealed no
specific inflammatory changes in lungs of all species and in kidneys
and livers of monkeys. Elevations in liver alkaline phosphatase and
serum glutamic-pyruvic transaminase activities that had been found in
the 189 mg/m$^3$ exposure could not be demonstrated at 20 mg/m$^3$.

### Liver

The liver is the prime target organ of vinylidene chloride
in most laboratory mammals and, apparently, in humans. Hepatotoxicity
has also been observed in perfused livers.

Hepatic injury occurs quite rapidly after inhalation exposure.
Hemorrhage and necrosis were observed in liver sections of fasted
rats within two to four hours of exposure to 2000 ppm vinylidene
chloride (33). Oral doses of 400 mg/kg body weight were hepatotoxic
to rats (36).

Morphological changes in livers of animals (dogs, monkeys and
rats) exposed to vinylidene chloride included fatty metamorphosis,
focal necrosis, hemosiderin deposition, lymphocytic infiltration,
bile duct proliferation, fibrosis and pseudo-lobule formation (29).

Jaeger et al., (33) observed morphological changes in livers
of fasted rats. They noted that these changes were distinctive and
did not resemble those which occur following vinyl chloride exposure.
Early changes do not involve the endoplasmic reticulum. Striking
changes in the nuclear mitochondria, and/or are plasma membrane

suggest that one or all three of these organelles are primarily affected by vinylidene chloride or its toxic metabolites.

Symptoms of liver injury caused by both oral and inhalation exposures to vinylidene chloride have been observed during a series of toxicology tests being conducted by the Manufacturing Chemists Association (3). Results of a 90 day study incorporating vinylidene chloride in the drinking water of rats showed minimal liver injury at a vinylidene chloride concentration of 200 ppm. This concentration was equivalent to an average daily dose of 19 mg per kg body weight for males and 16 mg per kg body weight for females. The liver injury, as revealed during microscopic pathology examinations, was in the form of increased vacuolation in the cytoplasm of the hepatocytes. Inhalation exposure of rats to 25 to 75 ppm of vinylidene chloride for 30 or 90 days also produced minimal liver injury. The incidence of liver injury was dose-related and increased with duration of exposure (37).

The appearance of large quantities of neutral fat within the liver is an indication of hepatotoxicity. Jeager et al., (30) observed an increase in the total amount of hepatic fat in rats as the dosage of vinylidene chloride was increased. At 800 mg/kg body weight, the amount of lipid present was almost doubled.

Perfused livers of fasted rats became pale and swollen and the flow rate of perfusate through the liver decreased when the liver was exposed to vinylidene chloride at a concentration of 20,000 ppm in the gas phase (33).

Vinylidene chloride can disrupt normal microsomal enzyme activity.
These enzyme activity alterations are the most sensitive indicators
of vinylidene chloride injury to the liver. Twenty hours after
administration of single doses of 500 mg vinylidene chloride per kg
body weight, male rats showed 11-fold increases in liver alkaline
phosphatase activity and 13-fold increases in liver tyrosine trans-
aminase activity (34). Both of these are stress responsive enzymes
(30). After 44 hours, plasma alanine transaminase reached a maximum
activity of 19 times controls and plasma alkaline phosphatase activity
showed a maximum 5-fold increase. Liver glucose 6-phosphatase
activity reached maximum depression (52 percent of controls) after 44
hours and then increased. These effects resemble the biochemical
effects of carbon tetrachloride (34).

Elevated serum alanine--ketoglutarate transaminase (AKT) precedes
hepatic necrosis and death. In rat liver perfusion experiments
increased AKT concentrations in perfusate correlate well with grossly
observable liver injury and are often used as indicators of hepato-
toxicity (33). Oral administration of vinylidene chloride (100 to
800 mg/kg body weight) caused dose-dependent liver injury as measured
by increased serum AKT activity (30).

Glutathione, a sulfhydryl-containing tripeptide, has been postu-
lated as a site of detoxification of vinylidene chloride. Animals
with a diminished hepatic glutathione concentration are significantly
more susceptible to vinylidene chloride (37). In both intact liver

28

and isolated perfused rat liver, normally insensitive livers were rendered susceptible to vinylidene chloride toxicity after glutathione depletion (33).

Glutathione concentrations are lowered during fasting and during the night. Animals are more susceptible to vinylidene chloride under these conditions (37). Vinylidene chloride was uniformly hepatoxic and fatal to 18 hour fasted rats at concentrations which were not fatal to rats fed ad libitum. Fasted rats have a very sharp vinylidene chloride dose response curve (see Figure 3), 100 ppm is a no-effect concentration, while 150 ppm causes a significant elevation of serum AKT and at 200 ppm deaths are observed. In fed rats, 2,000 ppm produced some injury but no deaths (33).

Fed animals are more sensitive to the hepatotoxic and lethal actions of vinylidene chloride if exposures are conducted at night. At this time hepatic glutathione concentrations are at or near their circadian* minimum (33, 38). Enhancement of vinylidene chloride toxicity due to short-term fasting or the circadian periodicity of glutathione are important factors to consider in reproducibility of toxicity testing or in industrial hygiene.

Renal damage has been observed following vinylidene chloride exposure. Inflammation of the kidney has been observed in rabbits, rats, dogs, monkeys and guinea pigs (29). Sections of kidney from rats showed nuclear hypertrophy of the tubular epithelium (29).

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*Circadian: pertaining to rhythmic biological cycles recurring at approximately 24 hour intervals.

These renal changes were considered to be a direct result of vinylidene chloride exposure.

Although lung damage has been observed during prolonged inhalation exposures (29), the lungs are not particularly sensitive to vinylidene chloride. Carlson and Fuller (43) observed no lung damage in rats exposed to vinylidene chloride vapor for one hour.

### Central Nervous System Depression

Vinylidene chloride is a central nervous system depressant (34). At 0.5 to 45 mg/l (12 to 1125 ppm), vinylidene chloride vapor was slightly narcotic to mice, rats, rabbits and guinea pigs (27). High concentrations of vinylidene chloride can elicit a strong narcotic effect leading to unconsciousness (9).

Two to four hours after administration of 400 mg per kg body weight oral dose of vinylidene chloride Jaeger and Murphy (36) observed prolonged pentobarbital-induced sleeping time. The prolonged sleeping time was accompanied by elevated serum corticosterone levels but hepatic injury was not observed at his time. These observations imply that central nervous system effects rather than disturbances of liver metabolism may have been involved in the prolongation of sleeping time.

### Mutagenicity

Vinylidene chloride has been shown to be mutagenic in microbial systems. Bartsch et al., (39) examined the mutageniccity of vinylidene chloride in Salmonella typhimuriam strains, using a

30

tissue-mediated assay which has been found effectiove in detecting

the mutagenicity of various carcinogens. Liver, kidney, and lung

fractions from mice efficiently converted vinylidene chloride into

mutagenic metabolites in vitro (39). Conversion was greatest in the

liver. With liver and kidney fractions, a much lower mutagenic

response was observed in rats than in mice. When the concentration

of vinylidene chloride in air was raised from 2 to 20 percent, an

increased mutagenic effect was noted only with mouse kidney and lung

fractions.

As has been shown for vinyl chloride, these results demonstrate

that the mutagenic effect of vinylidene chloride is mediated by

microsomal enzymes from various organs in the presence of an HADPH-

generating system and oxygen. These enzymes of the microsomal

mixed-function oxidase system probably convert vinylidene chloride to

alkylating intermediates.

Addition of sulfur-containing compounds to in vitro mutagenicity

assays containing mouse-liver fractions reduced the mutagenic effects

of vinylidene chloride (39). These results indicate that the mutagenic

vinylidene chloride metabolites are trapped by nucleophilic sulfur

groups, thus competing for binding to bacterial DNA. This observation

parallels the findings of Jaeger et al., (33) that the toxicity of

vinylidene chloride in rats is correlated with hepatic glutathione

concentration, and that cysteine has a protective effect.

## Carcinogenicity

Most chemical carcinogens have now been found to be mutagens, when assayed in one of the mutagenicity tests that combine microbial or mammalian cell systems as genetic targets with an in vitro or in vitro metabolic activation system (40). Therefore, there is a high probability that chemicals found to be mutagenic in the Salmonella test will then to be carcinogens. Although mutagenicity in microorganisms cannot be assumed to indicate carcinogenicity in humans, mutagenicity tests are currently used to select environmental chemicals for carcinogenicity tests. Since vinylidene chloride has been found to be mutagenic in Salmonella typhimurium, using a tissue mediated assay, the need for investigating its carcinogenicity is indicated.

Professor P.L. Viola of Regina Elena Institute for Cancer Research in Rome has reported that vinylidene chloride may be carcinogenic in high concentrations via inhalation (41). Viola, one of the first researchers to associate vinyl chloride with liver tumors in animals, allegedly has found liver tumors in animals exposed to vinylidene chloride (39,41).

### Synergistic and Antagonistic Interactions

Because vinyl chloride and vinylidene chloride monomers are chemically similar and are used simultaneously in the manufacture of copolymers, the question of possible toxic interactions between these must be considered. The interaction between these compounds

32

appears to be time dependent. Data suggest that the vinyl chloride monomer may protect against vinylidene chloride hepatotoxicity when given simultaneously, but prior administration of vinyl chloride diminished liver glutathione concentration, enhancing the degree of liver injury (37).

Vinyl chloride monomer is not acutely hepatotoxic in normal rats. Modest doses of vinyl chloride given simultaneously with vinylidene chloride afford significant protection against vinylidene chloride-induced hepatic injury.

The combined exposure to vinyl chloride monomer (1,056 ppm) and dichloroethylene (195 ppm) in a molar ratio of 5 to 1, resulted in complete protection of fasted rats. At a lower molar ratio (3 to 1) the protection was still apparent. Only at an equimolar concentration did injury occur to two out of the five fasted, exposed rats. (42).

Simultaneous exposure of fasted rats to a six-fold excess of vinyl chloride monomer (12,093 ppm) completely prevented elevation of serum AKT or serum sorbitol dehydrogenase (SDH) associated with approximately 2,000 ppm of dichloroethylene. This antagonism of vinyl chloride to the hepatotoxic effect of vinylidene chloride suggests a competitive interaction between these compounds (37).

On the other hand, pre-exposure to vinyl chloride can deplete glutathione concentrations in the liver, enhancing vinylidene chloride toxicity. Jaeger et al., (42) observed enhanced early acute hepatotoxic response to vinylidene chloride in fed rats which had been previously

33

appears to be time dependent. Data suggest that the vinyl chloride

monomer may protect against vinylidene chloride hepatotoxicity when

given simultaneously, but prior administration of vinyl chloride

diminished liver glutathione concentration, enhancing the degree of

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suggests a competitive interaction between these compounds (37).

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glutathione concentrations in the liver, enhancing vinylidene chloride

toxicity. Jaeger et al., (42) observed enhanced early acute hepatotoxic

response to vinylidene chloride in fed rats which had been previously

exposed to vinyl chloride. This synergism is consistent with a
hypothesis of a common pathway involving glutathione for both vinyl
chloride and vinylidene chloride toxicity or metabolism (37).

Administration of the hepatic microsomal enzyme-inducing agents
phenobarbital and 3-methylcholanthrene to rats increased the lethality
of vinylidene chloride inhalation (43). Rats were able to tolerate
very high concentrations of vinylidene chloride for one hour.
However, when animals were pretreated with either phenobarbital or
3-methylcholanthrene and exposed to vinylidene chloride for one hour,
most animals died either during exposure or shortly thereafter.
Surprisingly, the microsomal enzyme inhibitors SKF 525A and Lilly
18947 also increased vinylidene chloride lethality as evidenced by
shortened survival time during continuous inhalation (43). These
data lend credence to the results of Jenkins et al., (34) and Carlson
and Fuller (43) concerning the effects of phenobarbital may be due to
differences in time parameters, routes of administration or indicators
of toxicity.

The findings of Carlson and Fuller (43) that microsomal enzyme
inhibitors increse vinylidene chloride lethality, the findings of
Jenkins et al., (34) that microsomal enzyme inducers decrease vinyli-
dene chloride toxicity and the observation of Conney (44) that female
rats, which have lower microsomal enzyme activities than male rats,
were more susceptible to vinylidene chloride toxicity suggest that
metabolism of vinylidene chloride by microsomal enzymes results in
products which are less toxic than the parent compound.

## Current Research

The Manufacturing Chemists Association, in cooperation with Dow Chemical Corporation (U.S.A.) is administering a research program to be conducted by an independent laboratory on the health aspects of exposure to vinylidene chloride. Their research proposal outlines five major research projects: 1) a toxicological study on the effects of vinylidene chloride included in the drinking water of rats for 90 days and two years; 2) a toxicological study of vinylidene chloride in peanut oil fed to dogs for 90 days; 3) a 90 day and two year vapor inhalation study of vinylidene chloride on rats; 4) a study of the effects of maternally inhaled or ingested vinylidene chloride on rat and rabbit embryonal and fetal development; 5) a study of absorption, distribution, metabolism and ex cretion of ingested and inhaled vinylidene chloride in rats (47). The third study mentioned above would parallel Dr. Viola's work in Rome which has been the only report published concerning a possible carcinogenic effect of vinylidene chloride (17). Only the first two 90 day studies have been completed to date with no evidence of carcinogenicity of vinylidene chloride reported (48).

Research is also in progress by S. D. Murphy and R. Jaeger on the toxic interactions between vinyl and vinylidene chloride. The quality of the interaction appears to be related to the temporal relationships of exposure to the two compounds. They are investigating the biochemical bases for observed effects on intact animals exposed to industrial chemicals (49).

36

E. S. Reynolds and S. Szabo are investigating the sequential morphologic, enzymatic and compositional changes in cellular membrane systems in cells following vinylidene chloride poisoning to determine their cause and interrelationships (50). The lesions produced by vinylidene chloride on liver parenchymal cells appear similar to the lesions produced by the free radical reactions of carbon tetrachloride. Their proposed researches will attempt to provide a clearer understanding of the chemical and cellular injury associated with vinylidene chloride and how these events can be detected, prevented, or reversed in man.

C. AMBIENT CONCENTRATIONS, POPULATION AT RISK AND MEASUREMENT TECHNOLOGY

There are four normal sources of vinylidene chloride monomer emissions in the normal production process (see Figure 4 for their locations). These process vents plus spills incurred during loading of transport vehicles would constitute most of the vinylidene chloride monomer emissions associated with its production. Vinylidene chloride is also released during the production of saran resins and saran latex as well as during 1,1,1-trichloroethane production. Releases in these polymer production facilities are summarized in Table II. Total annual releases from the polymeric uses of vinylidene chloride are approximately the same in amount as releases from the production of vinylidene chloride monomer. However, the former sources occur at many different sites (see Section D) while only three plants produce vinylidene chloride. Thus, site specific release of polymer plants is actually far less than that for vinylidene chloride monomer production. These data are presented in Table II (estimated emissions are prior to control device application).

Threshold Limit Values (TLV) are set by the American Conference of Governmental Industrial Hygienists for airborne concentrations of hazardous chemicals. Since 1971, the TLV for vinylidene chloride has been reduced from 25 ppm for an 8-hour working exposure to 10 ppm (6). Sweden sets its exposure levels at a recommended 1 ppm exposure with a maximum of 5 ppm weighted average for 15 minute periods (26). Dow Chemical Corporation has stated that the average exposure of a

38

**FIGURE 4**
**VINYLIDENE MONOMER PRODUCTION**
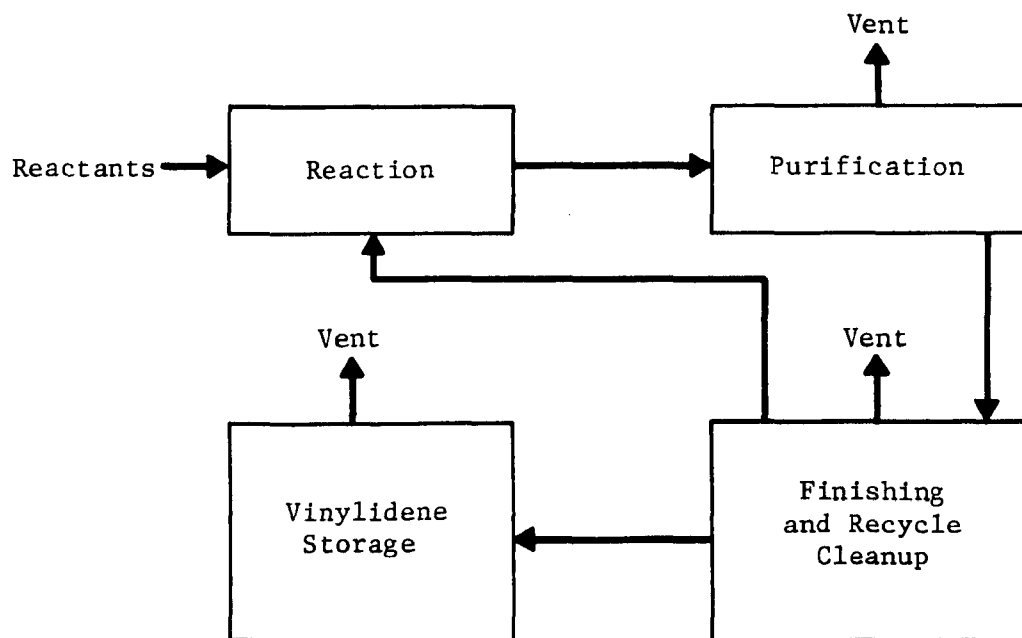


Source:   Strasser, J. P., Dow Chemical, U.S.A., personal communication.

TABLE II

ESTIMATED ANNUAL EMISSIONS OF VINYLIDENE CHLORIDE IN THE U.S.A.

|  |  |  | lbs/yr |
|---|---|---|---|
| A. | Monomer Synthesis | | 3,355,000* |
|  |  | | 611,000** |
| B. | Polymer Synthesis | | |
|  |  | Total | 679,000 |
|  | 1. Latex for Burner Coatings | | 120,000 |
|  | 2. Latex for Miscellaneous Coatings | | 150,000 |
|  | 3. Synthetic Fibres | | 160,000 |
|  | 4. Coating Resin for Cellophane | | 182,000 |
|  | 5. Extrusion Resin (Emulsion) | | 27,000 |
|  | 6. Extrusion Resin (Suspension) | | 40,000 |
| C. | Fabrication Polymer Processing | | |
|  |  | Total | 30,400 |
|  | 1. Coating Cellophane | | 1,600 |
|  | 2. Coating Plastics, Paper and Glassine | | 16,400 |
|  | 3. Extrusion | | 400 |
|  | 4. Miscellaneous Coating | | 12,000 |
|  |  | TOTAL | 4,064,000 lbs/yr. |

Sources: Strasser, J. P., Dow Chemical, U.S.A., personal communication.

Little, A. D., personal communication.

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*Emissions using an existing control technology.

**Emissions reflecting new control technology at PPG plant by late 1975.

production facility employee to vinylidene chloride monomer rarely if

ever reaches the TLV of 10 ppm (14). During those operations where

higher exposure is possible, appropriate protective gear is worn.

Determination of the risk to the general population is difficult.

If a cumulative effect exists, however, then the hazard may be

significant and should be measured. Ambient levels of vinylidene

chloride may be estimated in the vicinity of a theoretical plant,

releasing vinylidene chloride through ventilation and exhaust of

internal air. Atmospheric dispersion from this plant would distribute

the gaseous emissions downwind, as affected by the turbulent diffusion

of the air. Hypothetical plant conditions can be used as inputs to a

Gaussian plume equation* from Turner's Workbook of Atmospheric

Dispersion Estimates (49). The basic diffusion equation should be

---

*Ground level downwind concentrations resulting from a point source
 are predicted by the following equation:

$$X(x,0,0;H) = \frac{Q}{\pi \sigma_y \sigma_z u} \exp \left[ -1/2 \left( \frac{H}{\sigma_z} \right) \right]^2$$

where Q = uniform emission rate (grams/sec)
      u = mean wind speed affecting the plume (meters/sec)
      H = effective stack height, that is sum of stack height plus
          plume rise (meters)
      $\sigma_y$ = horizontal dispersion coefficient evaluated in terms of
          downwind distance to the point for which the concentration
      $\sigma_z$ = vertical dispersion coefficient evaluated in terms of down-
          wind distance to the point for which the concentration is
          being computed (meters)
      $X(x,0,0;H)$ = the ground level concentration along the plume axis
          in $gm/m^3$.

41

modified, however, due to the effect of the plant itself on the flow of air. Mechanical turbulence in the wake of a building tends to produce aerodynamic downwash, resulting in the fairly rapid diffusion of a gaseous emission down to the ground. This region of disturbed flow extends downwind a distance equal to several times the height of the building. While this situation does not result in a Gaussian distributed plume, it is proposed that a modification of the usual formula still can be employed. This is because the turbulent mixing in the wake of the building is assumed to be distributed uniformly in the vertical direction, analogous to the situation where a plume is trapped below an inversion layer. A limited vertical mixing height can be estimated using the equation:

$$\chi(x,0,0;H) = \frac{Q}{\pi \sigma_y u \ (.8L)}$$

where L is the limit of the mixing depth. In the case of an isolated rectangular building it is assumed that L equals 1.5 times the building height.

In addition there is a horizontal wind turbulence which is assumed to result in an initial horizontal plume spread equal to the width of the building normal to the wind direction. This is analogous to an area source emission where the area equals the building top. This can be modeled using a further modification of the basic Gaussian diffusion equation. A virtual point source is assumed upwind from the building at distance where the plume spread (for the given stability conditions)

42

would equal the crosswind width of the building. The modified diffusion equation is therefore:

$$X(x,0,0;H) = \frac{Q}{\pi \sigma_y' u \ (1.2h)}$$

where h is the building height and $\sigma_y'$ is based on the downwind distance to the receptor point plus the upwind distance to the virtual point source.*

A downwind distance of 500 meters from the emission source has been chosen as the hypothetical measurement point. This value was chosen to represent a point within the perimeter of each production facility. The values obtained for $X$ at this point for each of the three vinylidene chloride production facilities are given in Table III. The following equation is used:

$$X(mg/m^3) = 1.04852 \times 10^{-6} , \quad (lbs/yr)$$

where Y is the total annual emissions of vinylidene chloride.

For the purposes of the following calculations it is assumed that the improvements predicted at the PPG plant at Lake Charies, Louisiana are installed and function as predicted in Table III. An individual standing on the ground 500 meters downwind would receive a 1 hour dose ranging from 0.16 mg/m$^3$ at PPG, Lake Charies

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*Additional assumptions; (1) vinylidene chloride monomer is non-reactive; (2) atmospheric stability class is neutral; (3) wind speed is 6 meters/sec.; (4) plants are in constant operation except for 1-2 weeks/year for maintenance; and (5) the building height is 50 feet (15.25m) and width is 100 feet (30.48m).

43

TABLE III

EMISSIONS OF VINYLIDENE CHLORIDE

| Producer | Location | Total annual emmisions (lbs/yr) | $\chi$mg /m$^3$ | Emmission in lbs/100lbs VDC monomer |
|---|---|---|---|---|
| PPG | Lake Charles, La. | 2,920,000* | 3.06 | 1.72 - 1.67 |
|  |  | 175,000** | .1 83 | .10 |
| Dow Chemical | Freeport Texas | 289,000 | .3 03 |  |
|  |  |  |  | .51 - .48 |
|  | Plaquemine, La. | 146,000 | .1 53 |  |
|  | TOTAL | 3,355,000* |  |  |
|  |  | 610,000** |  |  |

Source: Little, A. D., personal communication.

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*Emissions using an existing control technology.

**Emissions reflecting new control technology at PPG plant by late 1975.

to 0.31 mg/m$^3$ at Dow, Freeport. The 24 hour exposures would range from 3.72 mg/m$^3$ to 7.32 mg/m$^3$. The maximum time-weighted average during an eight hour work day determined by the American Conference of Governmental Industrial Hygienists is 40 mg/m$^3$ (6). When the reactivity of vinylidene chloride monomer is also taken into account (i.e., that it remains in its original form in the atmosphere for only a short time prior to oxidation), the potential hazard from emissions of monomer production is reduced. Except during adverse atmospheric conditions, the exposure levels of the nearby resident population is probably not significant. If the original PPG emissions level is assumed, the maximum eight-hour allowed dose of 40 mg/m$^3$ would be reached at 500 meters in 13.1 hours and a possible daily exposure over 24 hours would reach twice the allowed daily exposure. Therefore, the planned controls are essential to reduce vinylidene chloride emissions to acceptable levels.

Vinylidene chloride is not generally measured in the ambient atmosphere but several methods of measurement are available. Samples may be collected by absorption on silica gel, condensation in a cold trap or as grab samples in vacuum bottles. These samples may then be analyzed by infrared spectrometry or mass spectrometry (9). Development of gas-liquid chromatography has made it possible to rapidly detect vinylidene chloride monomer at the ppm level (55). Once the samples are obtained, care must be taken to avoid oxidation and peroxide formation.

Vinylidene chloride requires very careful handling since not only is it toxic, but there is a concurrent fire and explosion risk.

The liquid monomer is irritating to the skin after a direct contact of only a few minutes. The presence of an inhibitor may well be partially responsible for this irritation since vinylidene chloride monomer would vaporize rapidly leaving MEHQ or phenol in increasing concentrations. Phenol, in high concentrations, is especially dangerous since, in addition to causing burns, it may be readily absorbed through the skin in lethal quantities (4). Hence, when handling vinylidene chloride protective clothing (impervious gloves, aprons, shoes) should be worn. If contact does occur, all contaminated clothing, including shoes should be removed immediately followed by a thorough washing with soap and water. Contaminated clothing should be thoroughly aerated and cleaned before reuse (5).

To prevent peroxide formation, large-scale equipment used intermittently in conjunction with vinylidene chloride handling should be left filled with water during shutdown periods. Piping used for loading and unloading is generally purged with nitrogen then flushed with water. Storage of vinylidene chloride monomer must be carried out in darkness, under a nitrogen blanket at about 10 psi pressure and less than -10°C. Empty tank cars or storage tanks must be kept pressurized and checked regularly for leaks.

A more thorough discussion of handling and storage precautions is available from Dow Chemical Corporation in a manual supplied to their users (4).

D. SOURCES

Vinylidene chloride was first reported by Reynault in 1838.
He described it as a "strange new fluid." The compound, according to
him, had a slight odor of garlic and became cloudy on standing (16).
It was regarded as a laboratory curiosity until the late 2920s.

In 1922, B. T. Brooks announced that halogenated ethylenes other
than vinyl chloride and vinyl bromide showed tendencies toward
polymerization (4). This led to a stimulated interest in vinylidene
chloride and the development by Ralph Willey and coworkers at the Dow
Chemical Company from 1932-1939 of the copolymerization and plasticiza-
tion techniques. The commercialization of polymers containing
vinylidene chloride under the trade name Saran began in 1940 (5).
The constant and increasing use of Saran is a result of its unique
properties of resistance to chemical and physical degradation, low
water absorption, and transparency (4).

The current domestic production level of vinylidene chloride is
around 260 million pounds per year (51). It is difficult to validate
this figure. The industry does not have to report their production
to the Federal Trade Commission since there are only two manufacturing
companies (the law requires that if three or more companies exist
which produce the same chemical, the production figures must be
reported to the Federal Government). They also refused to disclose
exact production figures for the purpose of this study.

According to SRI (17), 90 percent of the vinylidene chloride produced in the United States was domestically consumed, and the remaining 10 percent exported.

Demand for Saran copolymers has increased steadily since their development. Total annual world production of vinylidene chloride in 1967 was 100-150 million pounds. This annual production figure has approximately doubled in the last 10 years and there is a predicted growth rate of 5 to 10 percent per year for the next 5 years (16). Vinyl chloride monomer is readily available as a raw material since its use in the building industry has declined recently due to a reduction in housing starts.

Two companies produce vinylidene chloride at three geographic locations in the United States situated along the Gulf Coast (52). Dow Chemical Corporation, the major producer, has two facilities. They have been marketing vinylidene chloride monomer since 1939. PPG has only been marketing vinylidene chloride monomers for approximately six years (5). Figure 5 and Table IV provide estimates of production sites and corresponding levels. PPG uses 85 percent of its vinylidene chloride monomers for production of 1,1,1-trichloroethane and sells the remainder to industry. They are not involved directly in the production of polymers or copolymers. Dow Chemical Corporation uses about 80 percent of its production of vinylidene monomer internally to produce Saran polymers and markets the remaining monomer (14).

There are also several foreign producers of vinylidene chloride. In Japan vinylidene chloride is produced by Kureha Chemical Industries

# FIGURE 5
## GEOGRAPHIC DISTRIBUTION OF VINYLIDENE CHLORIDE PRODUCTION FACILITIES



● = Plant Location

TABLE IV

U.S. VINYLIDENE CHLORIDE MANUFACTURERS (1974)

| Producer | Location | Estimated Production (million lbs) |
|---|---|---|
| Dow Chemical, U.S.A. | Freeport, Texas<br>Plaquemine, Louisiana* | 90-95 |
| PPG Industries, Inc. | Lake Charles, Louisiana | 170-175 |
| | TOTAL PRODUCTION | 260-270 |

Source:  Arthur D. Little, Inc.

*Dow Chemical Corporation plans to expand the capacity of this plant during 1965.

and Asahi-Dow Chemical Company for the production of Saran-type

resins. In Germany Badischi-Anilin Soda-Fabrik (BASF) A. G. and

Huls produce vinylidene chloride (16).

A number of companies, among them BASF-Wyandotte Corp. B. F.

Goodrich, Dow, duPont, W. R. Grace and Co., Morton Chemicals and A. E.

Stanley use vinylidene chloride monomer to produce polyvinylidene

chloride (PVDC) laytex resins. In 1973, 21.26 million pounds of PVDC

laytex resins were produced (53).

The earliest and still most widely used commercial process for

the production of vinylidene chloride is the treatment of 1,1,2-tri-

chloroethane with sodium hydroxide:

$$CH_2ClCHCl_2 + NaOH \longrightarrow CH_2 = CCl_2 + NaCl + H_2O$$
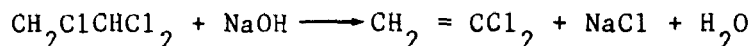
1,1,2-trichloroethane can be obtained in either or two ways: chlori-

nation of ethylene dichloride or addition of chlorine to vinyl

chloride (16). Industrially, however, large quantities of 1,1,2-

trichloroethane are generally available as a byproduct from the

preparation of other chlorinated hydrocarbons such as ehtylene

dichloride. Calcium hydroxide may be substituted for the sodium

hydroxide (16).

The reaction scheme is shown in Figure 6. All equipment is

fabricated from stainless steel except reboiler tubes which are

non-ferrous. A 10 to 20 percent by weight aqueous solution of sodium

hydroxide (or a calcium hydroxide slurry) and liquid 1,1,2-trichloro-

ethane are charged simultaneously into a heated, agitated reactor

51

where a temperature of 60-to-100°C and 15-30 psi of pressure are maintained (16). As the reaction proceeds, vinylidene chloride is distilled from the batch to prevent further cracking to acetylenic compounds. A phenolic inhibitor is generally also added at this point.

The condenser is then purged to prevent oxygen buildup and resulting peroxide formation. Water is removed after separation. The wet, inhibited vinylidene chloride is dried by azeotropic distillation and then fed to the finishing column where, under a pressure of 10-to-20 psi, the finished vinylidene chloride is withdrawn (16). More inhibitor is added prior to storage. Either phenolic or MEHQ inhibitor systems are normally employed.

The initial inhibitor, heavies and unconverted 1,1,2-trichloroethane are removed from the bottom of the finishing column and processed through the recycling tower. During recycling, waste products are removed from the bottom of the tower and the phenolic inhibitor drawn off just slightly above the bottom for recycling. The 1,1,2-trichloroethane is removed from the top of the recycling tower. After each batch is completed, a small purge of this system is also required to remove impurities which would otherwise accumulate producing chlorinated acetylenes and hence an explosion hazard (16). By the above process, the overall yield of vinylidene chloride is 96 to 98 percent.

Vinylidene chloride is transported by tank truck, tank car, and barge, with the jumbo tank car containing 20,000 gallons being the most commonly used container (16). Storage tanks should be equipped with a pressure release valve, level gauge, pressure gauge and remote shut off valves. The monomer must be kept away from sunlight, air, water and other polymerization initiators. It is generally stored at -10°C, in the absence of light under a nitrogen blanket at 10 psi pressure (5). A water spray system should be available to keep tanks cool in the event of a fire. Dikes and adequate drainage should also be available to confine and dispose of the liquid in event of the tank rupture (16). Vinylidene chloride must be stored in tanks lined with nickel, baked phenolic or glass since it may be corrosive or unstable in the presence of steel.

After extended periods of storage, vinylidene chloride monomer will often pick up iron contaminants. It is generally desirable to remove these compounds immediately prior to use.

Domestic productioon losses are estimated to release 3.355 million pounds per year of vinylidene chloride into the environment* (51). The principle production losses are through vents on the purification equipment and during recycling. There is also additional monomer loss during transportation and storage.

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*This figure assumed no control technology, and thus must be viewed
 as an upper bound.

## E. CONTROL TECHNOLOGY

Vinylidene chloride is both toxic and volatile. Care must be taken in both handling and storing material. Precautions should be taken to avoid skin contact or vapor inhalation, hence protective clothing should be worn and the use areas well ventilated. When disposing of waste vinylidene chloride (resulting from tank cleanups or spills) great care must be taken to avoid explosions and to prevent mixing with the sewer water.

Workers in industrial or commercial operations manufacturing for using vinylidene chloride run the greatest exposure risk. It is essential that control procedures be implemented so that the time weighted average concentration in the atmosphere does not exceed 10 ppm. Immediate cleanup of spills, periodic inspection of equipment, repair of damaged equipment and rapid repair of leaks must be the rule in order to minimize atmospheric contamination and accidental skin contact with vinylidene chloride.

Since vinylidene chloride is stored under pressure, it is essential that storage facilities are hermetically sealed. A melt seal is generally used because mechanical sealing methods tend to fail in long-term operations. An effective seal can be easily achieved in a twin-screw mechanism through the dynamic sealing generated by reverse elements built into the screw configurastion (54). Such a screw configuration can be readily assembled on equipment that allows interchangeable and segmented screw sections.

All emission controls for the production of vinyl chloride, and vinylidene chloride required by law are not yet due in place. The OSHA office of the State of Michigan has required chemical companies located there to maintain emissions rates for organic halogenated compounds below 40 pounds per day. Controls to accomplish this will be in place by the third quarter of 1977 at Dow Chemical Corporation's polymer production facility (14). These are more strict than those in effect at the vinylidene chloride monomer production facilities in Texas and Louisiana. The EPA still has the standards for vinyl chloride under consideration and industry plans to adopt comparable control measures for vinylidene chloride once the standards are set (14). Industry has, however, implemented many of the planned controls (14).

Several methods are available to control airborne emissions of vinylidene chloride during monomer production, storage and transportation.

● <u>Proper design of vents and vapor condensing apparatus.</u>

Vents are located on the separator, finishing columns, recycling tower and on storage containers of normal production facilities. At worst, this vented air may contain 5 percent monomer residues (14). These vents are ducted to actived charcoal absorbers (industry has not yet found a way to recycle used charcoal economically so it must be disposed of as a waste product).

A danger of peroxide formation on the surface of the charcoal exists (55). In work areas, aspirators can be used to draw contaminated air to a cyclone separator and a cloth filter used to remove particulates. The air is then drawn through a blower which exhausts the monomer away from the work area, preferably to an incinerator tower. Separated particulates resulting from the filters and scrubbers must be oxidized or incinerated to reduce any potential explosion hazard from vinylidene chloride adsorbed on the fines.

● Recovery of Vinylidene Chloride

It is possible to recover vinylidene chloride from purging gas streams used in equipment cleaning by refrigerated solvent scrubbing. The choice of solvent is largely a matter of availability. Following solvent selection, the conditions for the scrubbing are determined to minimize solvent loss with the vent gas stream. Solvents successfully used are acetone, methyl ethyl ketone, ethylene dichloride, butyl acetate and heptyl butyl ketone. The vinylidene chloride recovered is returned to the reaction system. The resulting vent gas is free of vinylidene chloride (55).

● Incineration of Organic Wastes

Incinerators, or flare stacks, are widely used to dispose of flammables and should be used as a final cleanup technique for even solvent-scrubbed vent gases (55). Use of incineration for disposal of large volumes of vinylidene chloride would require the removal and separate disposal of the resulting hydrochloric acid.

- Proper Vent Location

Within the plant, it is important to maintain constant air flow and have filtered vents at points near where spills or leaks are likely to occur. For example, the point in the production scheme where the project quality samples are taken. Since vinylidene chloride is 2.8 times heavier than air, floor vents should be used in addition to usual wall or ceiling vents.

- Proper Handling and Transport

During transport, the potential problem areas are sampling, vehicle emissions, vehicle gauging and loading line clearance. Oxygen content of empty transportation vehicles should be determined remotely prior to loading and the oxygen analyzer vented remotely to avoid personnel exposure. These should be equalizing lines so the vehicle vapor content may be displaced during loading. The final problem of loading operations is flushing the loading hose so that the hose may be disconnected without allowing exposure. Liquid in the loading hose must be flushed with nitrogen into the transportation vehicle. The loading line is then purged to an incinerator or tall stack so the vinylidene chloride vapors are removed before the hose is disconnected (57).

During filling of a storage tank, there is also the problem of displaced inerts disposal. Two methods are available to avoid having to vent the tank to the atmosphere. One is use of a refrigerated vent recovery system. The second involves the compression of the

storage vent stream to feed it to the plant process where vinylidene chloride is recovered and the inerts, mostly nitrogen, are removed with the production waste (56).

Currently, new tank car sizes are being limited to 25,500 gallons. The car must be insulated or have heavy shields to reduce the possibility of tank punctures during derailments. Major industry use of a poorly-designed 38,000-gallon car has resulted in a number of accidents and the resultant new tank car regulations (55).

It is necessary to monitor emissions of vinylidene chloride during distribution operations. A portable organic vapor analyzer with a chromatographic column attachment can be used (55). Each loaded tank vehicle should be thoroughly checked for leaks with a similar instrument prior to shipment.

Pipelines offer the ultimate reduction in vinylidene chloride emissions. However, pipeline systems carrying suspected carcinogens will be subject to a new stringent set of rules requiring labeling, surface marking of underground pipes and emergency crews on standby (55).

● Use of Proper Cleaning Techniques

The procedure for clearing a production equipment section for replacement involves first purging of that section of the line with high pressure nitrogen. Inerts used in the purge should then be vented to a high stack or incinerator (56). Safety precautions such as protective clothing and breathing masks followed by proper cleanup

procedures should be employed. In the same way, empty tank cars and other transportation vehicles should be purged first with inert gas and then the inert gas removed by exposure to the atmosphere. Before refilling tanks, the reverse purging process must be followed to remove the air.

Some loss of vinylidene chloride occurs with the waste water from the production process. Waste water is discharged from reactor, separator, and recycling tower (see Figure 6). Waste water is also generated from tank washings and cleaning up of spills. Vinylidene chloride is insoluble in water and thus vaporizes quickly. If exposed to air, these discharges could produce toxic vapors and explosive peroxides. Some control measures are available to minimize these vinylidene chloride emissions.

● Water Purification by Activated Charcoal

Waste water can be purified by passage over an activated charcoal slurry. The need for disposal of the spent charcoal and the potential oxidation problem were discussed previously in atmospheric control. After passage over the bed of activated charcoal, the effluent may then be altered by catalytic or thermo-conversion into less harmful and more easily contained substances (56).

● Water Purification by Steam Stripping

Waste water resulting from spills or leaks should be rapidly vacuumed and sent to a steam stripper for temperature distillation. From this, the monomer can be recovered by vapor condensation or compression and recycled. (14).

59

**FIGURE 6**
**PRODUCTION AND PURIFICATION OF VINYLIDENE CHLORIDE**
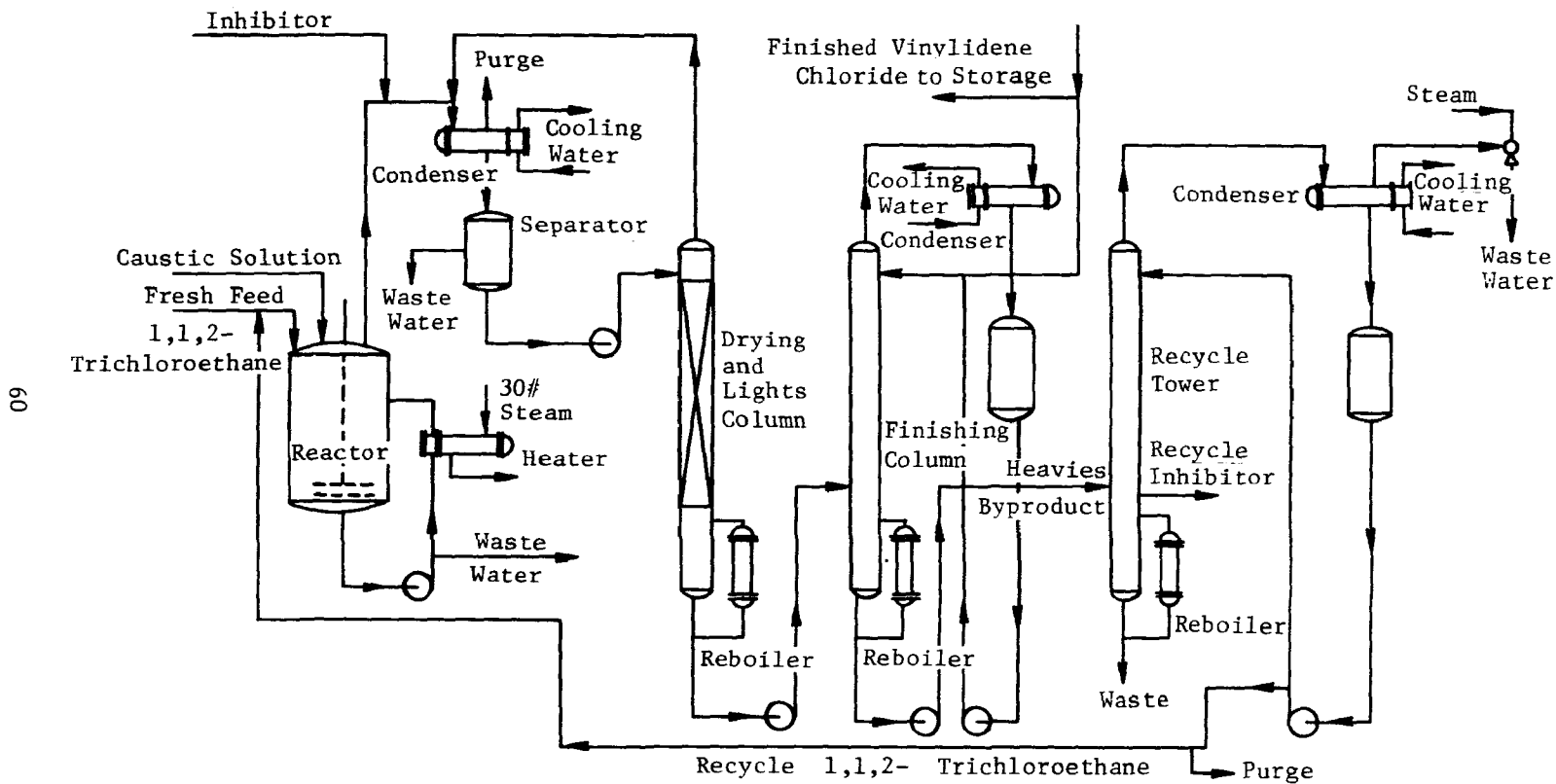


Source: Shelton, L. G., D. E. Hamilton, and R. H. Fisackerly, "Vinyl and Vinylidene Chloride" in
<u>Vinyland Diene</u> Monomers, E. C. Leonard, ed., Vol. XXIV, Part 3, 1971, pp. 1205-1282.

- ## Use of Waste Water in Cooling System

The purified waste water can be cycled to the condensers for use as a coolant to condense vinylidene chloride from the vapor to the liquid phase (58).

- ## Acid Neutralization

Hydrochloric acid, resulting from incineration of organic chlorides, mixed with process water will need to be neutralized or otherwise disposed. Neutralization of this waste water can be accomplished by passage over a limestone slurry bed (57).

The industry is adopting control procedures for vinylidene chloride which parallel those for vinyl chloride (14). The rationale for this is that the two compounds are chemically similar in their properties, and except for during monomer production, the two are generally present together for various polymerization reactions.

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