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April 13, 1988

Ms. Georgi Jones
Director, Office of External Affairs
Agency for Toxic Substances and Disease Registry
Chamblee 28 South
1600 Clifton Road
Atlanta, Georgia 30333

Reference: Toxicological Profile for Vinyl Chloride
Docket Control Number ATSDR-2

Dear Ms. Jones:

Enclosed is five (5) copies of comments from the Vinyl Institute on the Toxicological Profile for Vinyl Chloride, Docket Control Number ATSDR-2.

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COMMENTS ON EPA TOXICOLOGICAL PROFILE ON VCM

GENERAL COMMENTS

As described in Section 2.3.1.. SARA directs EPA to prepare toxicology profiles that will include an examination of the data, a determination of adequacy, and, where appropriate, an identification of needed toxicology testing. Although the draft Toxicological Profile for VCM (1988) is intended to meet this requirement, it falls far short of both the letter and spirit of the Act in many ways.

1) This document fails to incorporate key data.

2) It fails to provide the critical review necessary to draw a conclusion.

3) Because there is no identification of toxicological testing needs, we assume that all the data is considered adequate. The graphs appear to indicate the data is adequate. However, there is no clear statement in the test to this point and the detail of discussion of this subject is not adequate to draw any conclusions.

4) Referencing of data in tables and texts is inadequate.

5) There are some instances where reference books and computerized databases are utilized instead of the original references. The problem with this procedure is that it does not allow critical review and it presents the opportunity for perpetuating errors and misstatements. We kow for a fact that this has been a frequent problem or real concern for at least one of the reference books used.

6) The detail of information provided generally is insufficient to provide any confidence in the NOAEL, LOAEL, Minimal Risk levels or conclusions or a clear understanding of how they arrived at these. To this end we would recommend the use of NAS guidelines for use of safety factors (NAS, 1982).

7) The authors' use of a number of terms is questionable. The term FEL is misused on page 42, paragraph 1. Increase in DNA synthesis is not a frank effect. Also the descriptor "minimal" (page 44 General Discussion) is a poor choice. Standardized terms such as "Adequate" (or "Clear"), "Some" (or "Limited"), "Equivocal), "No Evidence" and "Inadequate" should be used because their meanings transcend the subject of carcinogenicity and are broadly applicable. Futhermore, it would be better if adequacy was assessed in these terms.

SPECIFIC COMMENTS

CONSUMER EXPOSURE IN CARS (Section 1.2, page 1, paragraph 1; Section 7.2.4, page 71, paragraph 3)

GM study needs to be referenced and discussed here

PLASTIC PIPE LEACHING DRINKING WATER EXPOSURE (Section 1.2, page 1, paragraph 2; Section 1.3, page 2; Section 2.2.3.2, page 18, paragraph 2; Section 2.3.3.3, page 24, paragraph 2; Section 7.2.4, page 71, paragraph 2)

Polymer Engineering and Science article on "Prediction of Vinyl Chloride Monomer Migration from Rigid PVC Pipe" needs to be referenced and discussed here.

CARCINOGENICITY AND OTHER DISEASE (Section 1.4, page 2, paragraph 1; Section 2.2.1.1, page 14, paragraph 1; Section 4.3.6.4)

The discussion of the types of disease and cancer caused by VCM is very misleading. The document states that "lower concentrations" cause "vinyl chloride disease" but fail to note that these effects are not of concern at present workplace and ambient concentrations. Furthermore, this section suggests that VCM can cause cancer of the "liver, brain, lung and possibly other organs." IARC Supplement 4 (1982) clearly states that "Vinyl chloride causes angiosarcoma of the liver" and distinguishes other tumor with lesser weight of evidence being "associated with".

A recent assessment of the data by the world-renowned epidemiologist Sir Richard Doll (1987) further supports this distinction. He notes that the evidence that VCM causes liver cancer, specifically angiosarcoma, is strong. However, the combined data on respiratory cancer fail to support an association of VCM and lung cancer. In the case of brain and lymphatic and hemopoietic cancers he indicates that the excesses are not statistically significant and that there is nothing to suggest that they are occupationally related.

EFFECTS ON OFFSPRING/DEVELOPMENTAL (Section 1.4, page 2, paragraph 1; Section 2.2.11, pages 13-14; Section 4.3.3.1; Section 4.4)

The profile on VCM fails to note that there is no evidence that VCM causes birth defects or reproductive effects but that it's only a concern from the point of view of potential transplacental carcinogenicity. The lack of focus on this conclusion or any conclusion may be due to the lack of consideration of key documents which are described in "Potential Effects of Vinyl Chloride on Human Offspring" (1987). Also, the absence of critical review has contributed to a misleading presentation.

It is also interesting to note that the discussion of developmental toxicity on page 13 appears to avoid the fact that "VCM did not cause significant embryonal or fetal toxicity and was not teratogenic in any of the species at the concentrations tested." (John et al, 1977) Based on these results it is incongruous that the document would espouse any NOAEL, not to mention one at 50 ppm. Unfortunately, there is no information provided to support the 0.7 ppm minimal risk level or the 50 ppm NOAEL. Such absence of detail and supporting rationale is a common deficiency in this document.

CANCER RISK ESTIMATES (Section 1.6.2, page 4; Section 2.2.1.1, pages 14-15)

Estimates of cancer risk are limited to those derived by EPA in 1985. Since risk estimates have been derived by others these should be identified and discussed. This is particularly important since some of these estimates (NHCH, 1987) differ by more than 2 orders or magnitude from EPA. Furthermore, EPA has recently revealed the fact that it is now using new risk assessment procedures which have dramatically lessened risk estimates for many chemicals (Chemical Regulation Reporter, 1988).

GENOTOXICITY (Section 2.2.1.1.)

The ability of VC to induce mutations in human cells was examined in three separate, independent studies conducted by Funes-Craviato et.al. (1975), Ducatman et. al. (1975), and Purchase et. al. (1975) which all examined workplace exposure to VC. All three studies suffer from a number of flaws which includes lack of exposure data, inappropriate statistics, and no descriptions of how control groups were selected. The studies show a slight association between potential VC exposure and chromosomal breaks in lymphocytes. Two of the studies, Funes-Craviato Set. al. (1975), Ducatman et. al. (1975), actually found that the number of chromosomal aberrations were often lower among workers as length of employment increased. The last observation is significant since it suggests that metabolism and/or repair become active after extended periods of exposure. Because of all the information that is missing from these studies, it is hard to develop any conclusions on the relationship between VC exposure and chromosomal aberrations.

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Workplace exposure and chromosomal aberrations among lymphocytes of male workers was also examined by Leonard et. al. (1977). The workers studied were employed in a VC facility in Belgium where there was no record of exposure levels. The authors assumed that employment during early exposure years was initially 500 ppm which was eventually reduced to 10 ppm. Controls who were employed in the laboratory of the same facility and were assumed to be exposed to less than 1 ppm. An additional 10 controls were obtained from outside the factory and assumed to never have been exposed. The average of the number of aberrations per 100 cells was approximately the same for each group. Even though some specific chromosomal aberrations were found in the exposed group, the significance of the finding is questionable since each of those workers received regular radiologic examinations which would produce the same observations. Consequently, this study does not support the premise that VC causes mutations in human subjects.

Fleig and Thiess (1978) studied aberrations of lymphocyte chromosomes among workers at a BASF plant in Germany. The actual plant exposures have not been documented in this study, but they were reported to be comparable to the German workplace standard which was around 1,000 ppm in 1945 and decreased to 150 ppm in 1973. The subjects chosen in this study had overt signs of VC toxicity which included angiosarcoma of the liver, and the controls were carefully matched and took into account smoking, alcohol, vaccinations, and other medical problems or treatment. They reported that they could find no differences between the cases and controls with the exception of one case that was receiving chemotherapy for cancer. Their data was not able to demonstrate an increase in chromosome aberrations as a result of high level VC exposure in the workplace.

Vinyl chloride has been examined in a number of different laboratory studies with mice, rabbits, and rats. Studies were designed to examine mutational effects (changes in DNA) as well as teratogenic/reproductive effects (birth defects, fertility, miscarriages, etc.).

An early study done by Anderson et. al. (1976) examined VC mutagenicity in fertile male mice using the DOMINANT LETHAL ASSAY. Their study exposed mice to VC for 6 hours per day for 5 days to concentrations of 3,000, 10,000, and 30,000 ppm. They were not able to find any mutagenic effects at any of the stages of spermatogenesis. To assure that their test system was functional, this study employed two known mutagens as positive controls (cyclophosphamide and ethly methane sulphonate) which did in fact give a positive response in their study. Therefore, the lack of mutagenic response from VC is indicative that VC is non-mutagenic rather than their test system lacks sensitivity.

The dominant lethal mutation study was later repeated by Himeno et. al. (1983) in CD-1 mice under two different exposure conditions. One group of mice was exposed for four hours per day during five consecutive days to 10,000 ppm of VC and the second group of mice was exposed for four hours per day, five days per week over a 10 week period to 5,000 ppm VC. This study, like the earlier study of Anderson et. al. (1976), was not able to demonstrate any dominant lethal type of mutations as a result of VC exposure, and this independently supports the observation that VC is non-mutagenic.

John et. al. (1981) examined the effects of VC exposure on the fetal development of mice, rabbits, and rats. All animals were exposed to 500 ppm VC for seven hours per day during the critical development

phase (organogenesis) for each species (days 6 to 15 for mice/rats and days 6 to 18 for rabbits). Some additional rats and rabbits were exposed to 2,500 ppm VC, and some additional mice were exposed to 50 to 500 ppm VC plus ethanol in their drinking water. At the high VC concentrations, there were some maternal deaths in mice, and some fetal body weights were lower for mice and rats treated with 500 ppm VC. Other than a depression in fetal weight in the high dose groups, there were no other apparent fetal changes. Quite surprisingly, fetal weights were unchanged from rats exposed to 2,500 ppm VC, and natural abortions in these particular rats were lower than the unexposed controls.

A similar study on teratogenicity was also carried out by John et. al. (1977) using mice, rabbits, and rats. Dose levels and exposures were similar to the previous study, but there was more emphasis placed upon concurrent ethanol treatment. The high dose of VC caused maternal toxicity in all species in addition to some embryonic death. The ethanol co-administration exaggerated all aspects of maternal toxicity in addition to decreased litter size, fetal weight, and fetal resorption. A number of skeletal defects were found with the co-administration of ethanol, but VC by itself was not teratogenic in mice, rabbits and rats. If anything the study emphasized the risks associated with ethanol consumption during pregnancy rather than indicating VC related problems.

Ungvary et. al. (1978) carried out a detailed study of VC exposure upon rats. In the first part of their experiments, the exposed rats on the 18th day of pregnancy to either 2,000, 7,000, or 12,000 ppm VC for 2.5 hours. They then detected and measured VC in maternal

blood, amniotic fluid, and fetal tissue which demonstrated that VC could be transferred to the fetus from the mother. In additional experiments they exposed rats to 1,500 ppm VC during different periods of pregnancy, and they were unable to produce teratological effects in the offspring. These results demonstrated that lack of teratological response in VC exposed rats were NOT due to the inability of VC to be transferred from the mother to the developing fetus.

All the animal studies cited above are remarkable in two important ways. First, some of the exposure levels of VC employed in the experiments were on the order of tens of thousands of ppm. Such exposures would only be encountered in the workplace under rare, isolated circumstances, and environmental exposure to such levels are not possible except in rare circumstances. Second, even though very high exposure levels were used in the animal studies, no mutations or birth defects could be detected. Substances known to give positive responses under similar conditions behaved as expected, so any lack of response to VC in the animal test systems could not be attributed to failure of the test systems. Rather, the more likely conclusion is that VC does not produce the anticipated adverse effects.

FDA REGULATIONS (Section 1.7, page 6; Section 2.2.3.1, page 18)

Statement of FDA regulations of food packaging is inaccurate. The FDA has recently proposed to amend the prior sanctions and regulations regarding vinyl food packaging to limit VCM to 5 to 50 ppb, not ppm. Also, there is no current official "action level" or "regulatory level" for VCM but there are manufacturer's voluntary guidelines.

EFFECTS ON OFFSPRING/REPRODUCTIVE (Section 2.2.1.1., page 14)

No reference is provided to indicate what animal data is being discussed. We assume this is Bi et al (1985). The authors fail either here or on page 50 to ask whether these observations are consistent with observations in other long-term studies. In fact, a review of the data indicates it is not (Maltoni and Mehlman, 1984).

More importantly, it should be noted that histopathologic organ changes are evidence of systemic changes even if they are found in reproductive tissues. Although observations of toxic effects in reproductive tissues would lead one to question whether the reproductive capacity of an animal might be compromised, it does not provide any evidence that a chemical is a reproductive toxin. In the case of VCM, the data indicates that VCM does not affect reproduction.

BIOLOGICAL MONITORING (Section 2.2.2, page 16, paragraph 3)

The authors note observations of elevated urinary coproporphyrin as a common finding associated with VCM-related liver disease and suggest it might be useful in population monitoring. What is not pointed out, however, is that while it is indicative of liver disease, it is not specific to VCM or chemical induced liver disease.

ADEQUACY OF DATA (Section 2.3.2.1, page 20)

Item 2 should note that while a potency factor or potency factors can be derived, the precision of those estimates is unknown.

The health effect and exposure route referred in Item 4 are unclear.

INTERACTIONS (Section 4.4)

The document fails to discuss the interaction of VCM and alcohol (i.e. metabolism and cancer in workers).

REFERENCES

1. Drinking Water and Health, Vol. 4, National Academy Press, Washington, D.C., page 153.

2. Maltoni, C. and Mehlman, M.A. Experimental Research on Vinyl Chloride Carcinogenesis, Vol. II, Princeton Scientific Publishers, Inc., Princeton, New Jersey, 1984.

3. "Prediction of Vinyl Chloride Monomer Migration from Rigid PVC Pipe," A. R. Berens and C. A. Daniels, Polymer Engineering and Science, V16, 8, August, 1976.

Prediction of Vinyl Chloride Monomer Migration from Rigid PVC Pipe

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Data on the solubility and diffusion of vinyl chloride monomer (VCM) in PVC resin powders have been combined with published solutions of Fick's diffusion equation to yield predictions of the amount and rate of loss of residual VCM (RVCM) from rigid PVC pipe under storage and service conditions. The principal factors controlling VCM migration are the initial VCM content, thickness of the PVC section, temperature, and the age of the PVC product. Analytic solutions are presented for RVCM loss from freshly extruded pipe (uniform VCM concentration) into either the storage environment or the pipe contents. From these solutions, estimates are made for the real-world situation of closed-system service following variable storage periods. The validity of this approach for rigid PVC pipe in water-service is supported by reasonable agreement between its predictions and experimental laboratory data on the VCM content of water stored in PVC pipes. Both the predictive model and experimental data indicate that PVC pipe containing ≤1 mg/kg (1 part per million) residual VCM will result in VCM concentrations in water of less than 0.002 mg/kg under any expected service conditions.

INTRODUCTION

A subject of continuing concern since the discovery of the potential toxic hazard of vinyl chloride monomer (VCM) has been the migration of residual VCM from finished PVC products into the environment or into liquids transported in PVC vessels. Many efforts have been made to investigate this problem by direct analysis for VCM in media in contact with PVC vessels. Early efforts in 1973 were questionable because of the imprecise analytical methods then available. As analytical procedures have been improved, the residual VCM levels in commercial PVC products have been sharply reduced, so the direct analysis for VCM migrating from today's PVC products remains a very difficult problem. Recent data (1) show that sensitivity in the thousandths of a milligram per kilogram range is needed to analyze for VCM in water contained in PVC pipe even at the residual VCM level of 20 mg per kilogram. A reliable model for predicting the amount and rate of VCM migration from available basic transport data and theory thus would be very useful.

Our approach to the development of a predictive model has been to combine the solubility and diffusion data we have obtained for VCM in uncompounded PVC resin powders (2, 3) with the solutions of the Fickean diffusion equations given by Crank (4). This report summarizes the assumptions and approximations involved in applying these diffusion equations to the VCM migration problem and illustrates the possible calculations with some numerical examples. The predictions of our model are compared with experimental data on VCM contents of water stored in PVC pipe.

CALCULATIONS OF VCM MIGRATION

Background and Assumptions

On the basic assumption that migration of VCM thru PVC is controlled by the diffusion of VCM in the PVC phase, this process may be treated by well-known theory. The basis of classical diffusion theory is the simple differential equation known as Fick's First law,

$$F = -D \frac{\partial c}{\partial x} \qquad (1)$$

which states that the amount of diffusing substance crossing a unit plane area in unit time, F, is proportional to

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the concentration gradient across the plane, $\frac{\partial c}{\partial x}$. The proportionality constant, D, is called the diffusion coefficient. The minus sign indicates that diffusion always occurs toward the region of lower concentration, i.e., "downhill". Net diffusive transport ceases when the concentration gradient becomes zero, i.e., when the concentration becomes uniform.

The use of Fick's law to calculate useful quantities, such as the rate at which a diffusing substance escapes from a solid object, or the concentration profile within the object, involves some very complicated mathematics. Exact mathematical solutions are generally possible only for geometrically simple shapes and for certain specified initial and boundary conditions. Many of the useful solutions are presented in Crank's text (4), and fortunately it seems that some of the most important problems in VCM migration from PVC can be handled with a few of these equations.

Our application of these equations and our diffusion data to VCM migration from PVC products involves several assumptions:

(1) The diffusion of VCM in PVC obeys Fick's law.

(2) The diffusion coefficient is independent of VCM concentration.

(3) The value of the diffusion coefficient in rigid PVC products is the same as we have determined for pure PVC resins.

(4) The diffusion coefficient is independent of the medium surrounding the PVC; i.e., values we have determined by vapor sorption/desorption also apply to migration into a liquid phase.

Assumptions (1) and (2) have been demonstrated to be very satisfactory approximations at quite low RVCM concentrations by our work on PVC powders (3). More limited experiments on thin, rigid PVC films also support assumption (3), although some variation of D might be expected for varying amounts and types of compounding additives. The use of assumption (4) should be considered a tentatively useful approximation; we shall see that it does seem justified by experimental data on VCM migration into water from PVC pipe.

To describe the diffusion of RVCM from PVC products through Crank's equations, three situations have been considered:

Case I: RVCM loss during storage of a freshly-manufactured PVC product.

Case II: RVCM loss from freshly formed PVC products into a closed medium.

Case III: RVCM loss into a closed medium from previously aged PVC products.

In both Cases I and II, the initial RVCM concentration is assumed to be uniform through the thickness of the PVC product; this is probably a valid assumption only at the time of extrusion, as the surface concentration of RVCM will quickly decrease upon exposure to a low-VCM environment. Cases I and II differ in the time-dependence of the surface concentration: In Case I, the surface concentration of VCM remains essentially zero, as any RVCM escaping is carried away in the environment; this case may represent storage or service in a continuously renewed environment, such as flowing water. In Case II, the VCM concentration in the medium builds up with time, and consequently so does the VCM concentration in the surface of the PVC. Case III is the general situation in a real-world application: VCM loss into a closed medium follows a variable storage period and thus proceeds from a product in which the surface VCM concentration is already depleted. We will see that the aging period between manufacture and closed-system service is quite important in determining the rate of VCM migration into the contents of a PVC pipe.

Now let us consider the details and some numerical examples of each of these three cases.

Case I—VCM Loss During Storage

Consider a freshly extruded PVC product, quickly cooled to ambient temperature and stored in an atmosphere of essentially zero VCM content. The initial RVCM concentration in the product is C_0 and may be assumed to be uniform through the product. At the surface, equilibrium is quickly established with the environment and the RVCM concentration is zero. We assume that VCM leaving the PVC is carried away (e.g., good air circulation) so that the surface concentration remains zero. We want to calculate (a) the amount of VCM which leaves the PVC and (b) the concentration profile within the PVC, both as functions of time, temperature, and sample thickness.

Crank gives solutions to this problem for several simple geometries—plane sheets, solid and hollow cylinders and solid spheres. The predictions for hollow cylinders are virtually identical to those for plane sheets, provided the wall thickness is less than the inside diameter. Thus for all practical PVC products (pipes, bottles, sheets, films), we need consider only the mathematical solutions for plane sheets. Equations for the amount of VCM escaping from the sheet may be written in terms of M, the fraction of the original VCM which escapes in time t. The general expression, valid at all times, is

$$M = 1 - \sum_{n=0}^{\infty} \frac{8}{(2n+1)^2 \pi^2} e^{-Dt(2n+1)^2 \pi^2 / L^2} \qquad (2)$$

where n is the series of integers (0, 1, 2—), D the diffusion coefficient, and L the sheet thickness. For the late stages of the process ($M > \sim 0.6$), terms beyond $n = 0$ become insignificant, and *Eq* 2 becomes

$$M = 1 - \frac{8}{\pi^2} e^{-D\pi^2 t/L^2} \qquad (3)$$

For $M < \sim 0.6$, a very good approximation is given by

$$M = 4 \left(\frac{D}{\pi L^2} \right)^{\frac{1}{2}} t^{\frac{1}{2}} \qquad (4)$$

Thus the initial loss of VCM is proportional to the square root of the storage time after extrusion. For numerical calculations, we need only the sheet thickness and the diffusion coefficient values. From our measurements on PVC resins (3), the value of D at various temperatures is given by

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$$D = 3.7 \exp \frac{-17{,}000}{RT} \qquad (5)$$

for D in cm²/sec, T in °K, and R = 1.987 cal/mol°K.

Equations 3, 4 and 5 permit prediction of the fractional loss of RVCM from rigid PVC products for different sheet thicknesses, times and storage temperatures. Examples of numerical results are given in *Figs. 1* and 2 as plots of M vs $t^{\frac{1}{2}}$. *Figure 1* shows such RVCM-loss curves for several sheet thicknesses at 30°C, where $D = 2 \times 10^{-12}$ cm²/sec = 1.73×10^{-7} cm²/day, and *Fig. 2*, for a 1 mm sheet thickness at several temperatures.

The concentration of RVCM remaining at time t at various distances from the sheet surface (i.e., the concentration profiles) can also be calculated from the same parameters. Crank gives the general solution as

$$\frac{C - C_o}{C_1 - C_o} = \sum_{n=0}^{\infty} (-1)^n \, erfc\left[\frac{(2n+1)\,l - x}{2(Dt)^{\frac{1}{2}}}\right] + \sum_{n=0}^{\infty} (-1)^n erfc\left[\frac{(2n+1)\,l + x}{2(Dt)^{\frac{1}{2}}}\right] \qquad (6)$$

where C is the concentration at time t at distance x from the center of the sheet, C_o is the initial (uniform) concentration, C_1 is the constant concentration at the surface (zero in our case), and l is the half-thickness of the sheet; "*erfc*" stands for error function complement, defined as

$$erfc\, z = 1 - erf\, z \qquad (7)$$

The error function, "*erf*", also called the probability integral, is tabulated in standard mathematical tables. For times short enough that the concentration at the center of the sheet does not decrease significantly below C_o, only the first term of *Eq 6* is necessary; then, using *Eq 7*, we have simply

$$\frac{C}{C_o} = erf\left[\frac{l - x}{2(Dt)^{\frac{1}{2}}}\right] \qquad (8)$$

Using *Eq 8*, or *Eq 6* where necessary, we have calculated concentration profiles at 30°C ($D = 1.73 \times 10^{-7}$ cm²/day) for a PVC sheet ⅛ in. thick (or pipe with ⅛ in. wall). The results at various times are plotted in *Fig. 3*. Note that the VCM lost in the first month comes only from the 100 microns of PVC near the surface. It takes over 20 years in this case for the VCM concentration near the center of the sheet to decrease appreciably.

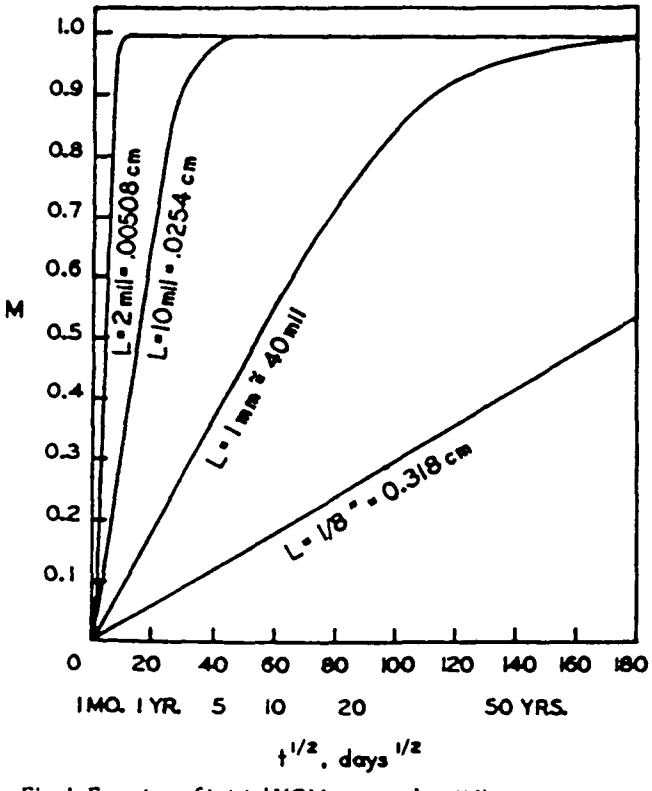

Fig. 1. Fraction of initial VCM content lost (M) vs square root of time ($t^{\frac{1}{2}}$) for PVC sheets of various thicknesses (L), calculated for 30°C ($D = 1.73 \times 10^{-7}$ cm²/day), Case I.

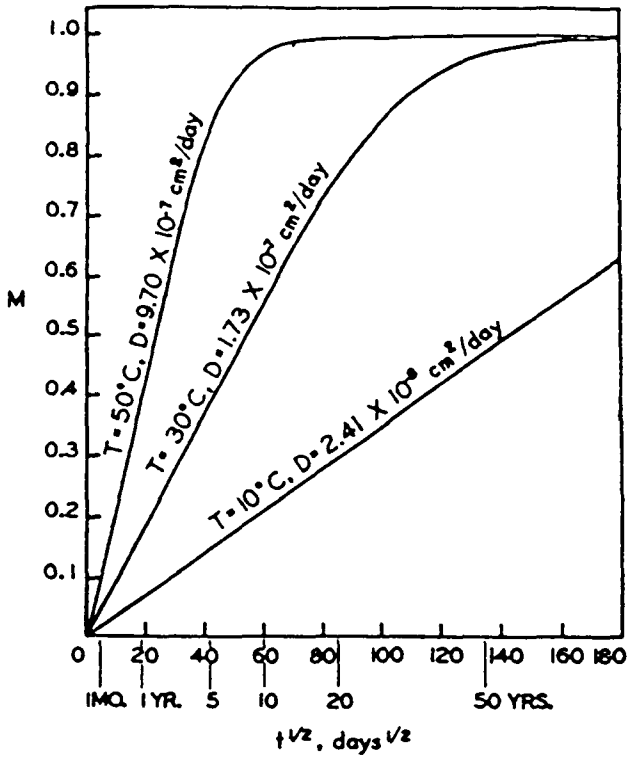

Fig. 2. Fraction of initial VCM content lost (M) vs square root of time ($t^{\frac{1}{2}}$) for PVC sheet 1 mm thick, calculated for various temperatures, Case I.

CASE II—VCM MIGRATION FROM FRESHLY/FORMED PVC PIPE INTO CONTENTS

The Case I calculations may be applied whenever VCM leaving the PVC product is carried away by the environment (storage in circulating air, water-pipe service with flowing water, etc.), so that the surface concentration of RVCM remains essentially zero. For PVC pipes in ordinary service, the outer surface is generally exposed to a low-VCM environment, i.e., a Case I situation. On the inside of pipes with stagnant contents, on the other hand, VCM leaving the PVC builds up in concentration in the contents. Consequently, the inside surface concentration of RVCM in the PVC, assumed to remain in equilibrium with the contents, also increases with time. At very long times, RVCM in the inner half of the

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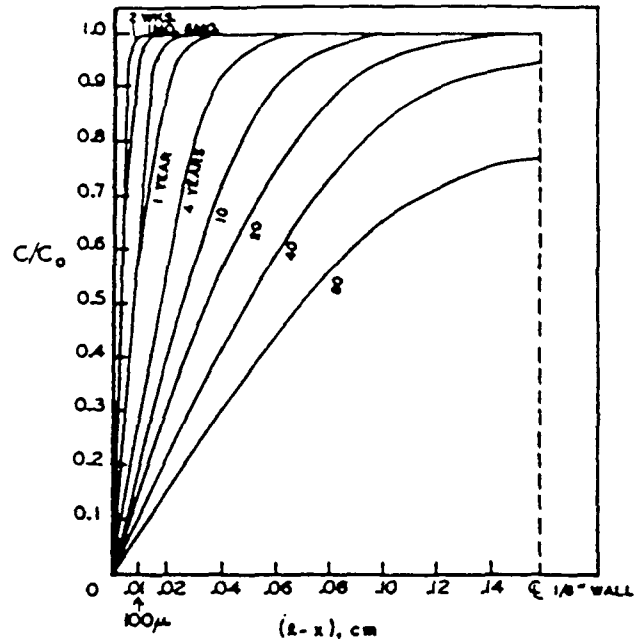

Fig. 3. Relative VCM concentration (C/C_o) vs depth below sheet surface ($l - x$) at various times, calculated for ⅛ in. thick PVC sheet at 30°C ($D = 1.73 \times 10^{-7}$ cm²/sec). Case I.

PVC wall and in the contents will diffuse outward to the environment. This net outward diffusion of VCM will only occur after the VCM concentration at the midline of the wall falls below its initial value; the time at which this occurs can be estimated from concentration profiles such as those in *Fig. 3*. Since this effect occurs at such long times for pipes of normal wall thickness, we have not considered it in our model, but instead have applied Case I and Case II calculations independently to the outer and inner halves of the PVC walls, respectively.

The rate and amount of VCM entering the pipe contents may be calculated through equations given by Crank. The maximum amount of VCM which will enter the contents is that required to establish equilibrium between PVC and contents, and is governed by the partition coefficient and the ratio of volumes of PVC and contents. The partition coefficient, K, is defined as the ratio of RVCM concentration in the PVC to that in the contents at equilibrium (both expressed in the same units, e.g., g/liter). The volume of PVC supplying VCM to the container contents, V_{PVC}, is one-half the total PVC volume, as RVCM in the outer half diffuses outward in the situation we are considering. It can be shown that the maximum VCM concentration, $C_{m,max}$, (ppm by weight) in the liquid contents of a PVC pipe originally containing C_o ppm VCM, is

$$C_{m,max} = \frac{C_o d_{PVC}}{(K + \frac{V_m}{V_{PVC}})d_m} \qquad (9)$$

where d_{PVC} and d_m are densities of the PVC and contents, and V_m is the volume of the contents.

From our data (2) on VCM solubility as a function of VCM pressure over PVC and water, we have estimated a value of $K = 49$ for the distribution coefficient of VCM between PVC and water at 30°C. It should be noted that this estimate of K assumes that the solubility of VCM in PVC is not affected by contact with water. It also involves a somewhat arbitrary selection of a value for VCM solubility in PVC, since we have shown (2) that this system shows non-ideal and history-dependent solubility. Using this value of K, *Eq 9* predicts that the maximum VCM concentration in water in a 1 in. I.D., ⅛ in. wall, PVC pipe containing 1 mg/kg residual VCM will be 0.027 mg/kg.

The rate of VCM desorption into the pipe contents may be obtained from another of Crank's equations:

$$M = \alpha[1 - e^{T\alpha^2}\, erfc(T/\alpha^2)^{\frac{1}{2}}] \qquad (10)$$

where $T = Dt/l^2$, M is the fraction of the original RVCM desorbed at time t, and $\alpha = V_m/KV_{PVC}$. To illustrate, we have applied *Eq 10* to the 1 in. I.D., ⅛ in. wall, PVC pipe filled with standing water. *Figure 4* shows the results, with scales showing both M, the fraction of original RVCM desorbed, and the VCM concentration in the water per original ppm RVCM. Also shown in *Fig. 4* is the M vs $t^{\frac{1}{2}}$ plot for Case I. Note that the initial rate of VCM desorption is the same for both Cases I and II, but the buildup of VCM in the water in Case II causes the desorption to slow down as equilibrium is approached.

Case III—VCM Migration in Closed-System Service from Previously Aged PVC Products

When a PVC product is put into closed-system service some time after manufacture, the RVCM distribution through the PVC at the time of filling and closing the container will not be uniform, as was assumed in the Case II calculations. Rather, RVCM will already be depleted near the surface, and VCM migration into the pipe contents will start from a VCM distribution as calculated in Case I (e.g., Figure 3). An analytical solution for this situation has been obtained by Daniels and Proctor (5), but a useful and simpler estimate of the rate

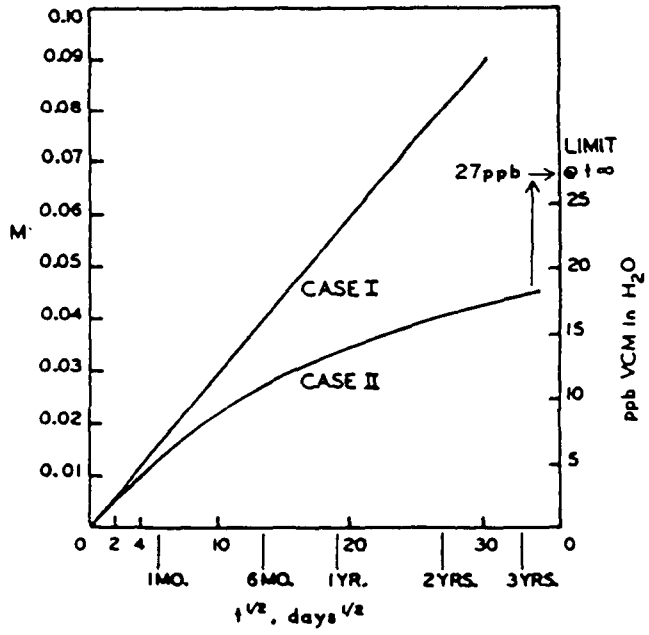

Fig. 4. Fraction of original VCM content lost (M) vs square root of time ($t^{\frac{1}{2}}$) for 1 in. I.D., ⅛ in. wall PVC pipe, calculated for 30°C ($D = 1.73 \times 10^{-7}$ cm²/day), Case I and Case II with water in pipe. Right-hand scale gives ppb VCM in water per ppm initial VCM in pipe.

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of VCM migration can be made by combining results of our Case I and Case II equations. For Case I during desorption of the first 60% of the VCM, *Eq 4* shows that the amount of VCM desorbed is proportional to the square root of storage time. Differentiating *Eq 4* gives

$$\frac{dM}{dt} = 2\left(\frac{D}{\pi L^2}\right)^{\frac{1}{2}} t^{\frac{1}{2}} \qquad (11)$$

Thus the *rate* of VCM loss is inversely proportional to the square root of storage time. For Case II, we saw that the *initial* rate of VCM migration into the medium in a closed system is the same as in Case I. Thus *Eq 11* also gives the initial rate of VCM migration into the closed system when t is the storage age of the PVC product at the start of closed-system service. Applying *Eq 11* to a PVC product with ⅛ in. wall thickness at 30°C ($D = 1.73 \times 10^{-7}$ cm²/day) gives the curve shown in *Fig. 5*. We see that the rate of RVCM desorption drops very sharply in the first few weeks of storage after manufacture.

It is also possible to estimate the amount of VCM which will migrate from a PVC product during a given period of a closed-system service following various Case I storage periods. This estimation may be explained with reference to *Fig. 6*, which illustrates the VCM-loss (M) vs $t^{\frac{1}{2}}$ curves for the three cases. Case III is approximated by shifting the origin of the Case II curve to point t_1 along the Case I line, where t_1 is the age of the PVC product at the start of closed-system service. Then the VCM lost from the PVC in the time interval a during continued open-system storage would be, from *Eq 4*

$$\Delta M = 4\left(\frac{D}{\pi L^2}\right)^{\frac{1}{2}} [(t_1 + a)^{\frac{1}{2}} - t_1^{\frac{1}{2}}] \qquad (12)$$

The VCM lost, and entering the contents of a PVC container, in Case III service, will be approximately equal to ΔM for relatively short service periods, and always less than ΔM. *Equation 12* thus is useful for calculating the maximum fraction of the original RVCM which will migrate into the contents of a PVC container during closed-system service for any time period as a function of the age of the container at the time of filling and closing. *Figure 7* illustrates results calculated from *Eq 12* for 7 and 30 day service periods for a ⅛ in. wall thickness at 30°C as functions of t_1. Again we see the important effect of a few weeks prior aging in reducing the amount of VCM migration into the contents.

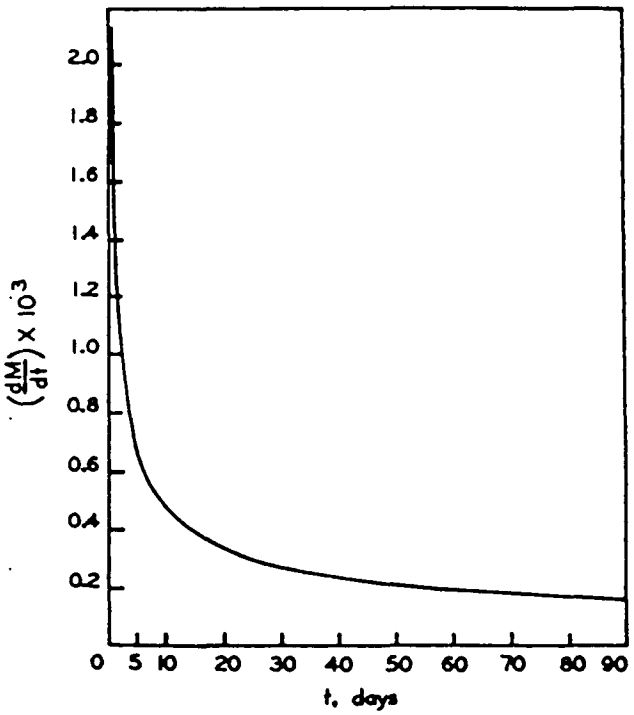

Fig. 5. Rate of VCM loss (dM/dt) vs time for ⅛ in. thick PVC sheet, calculated for 30°C, Case I.

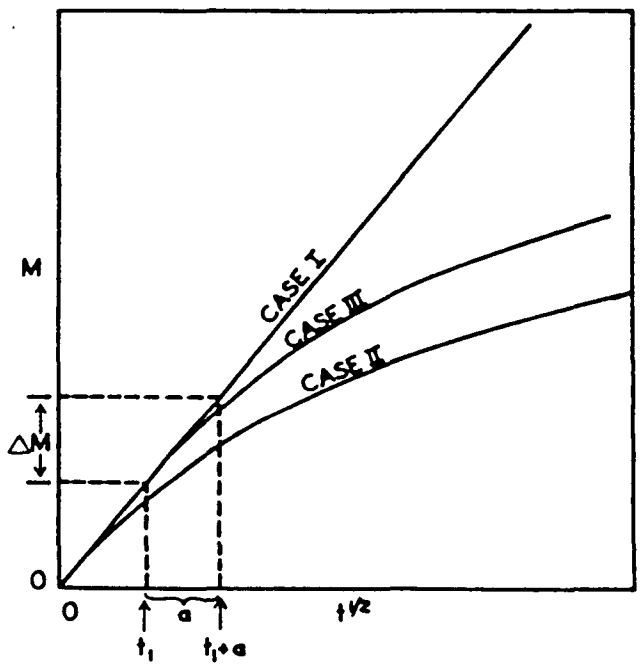

Fig. 6. Schematic comparison of fractional VCM loss (M) vs $t^{\frac{1}{2}}$ curves for Cases I, II, and III.

Comparison of Predictions with Experimental Data

The foregoing analysis clearly shows that the age of a PVC product at the start of an extraction test is an important factor in determining the amount of VCM extracted. Since this information is seldom available in reported extraction data, direct comparisons between our predictions and experimental data are possible for only a few cases.

For the data recently obtained by O'Mara and DeCapita (1) on the VCM content of water stored in 1 in. I.D. ⅛ in. wall PVC pipes at 23°C, the *approximate* age of the pipe samples, between extrusion and start of the extraction test, was known. A "headspace" GC analytical method was used to provide sensitivity to low VCM levels in the water (on the order of 1 or 2 thousandths of a milligram per kilogram). We have calculated the VCM content expected in the water, using *Eq 12* with the parameters appropriate to the experimental conditions; D was obtained from *Eq 5*. *Table 1* compares the calculated and experimental results. The agreement must be considered quite satisfactory, especially in view of a) our application of D values obtained from resin powders to pipe compounds, b) the somewhat uncertain age and

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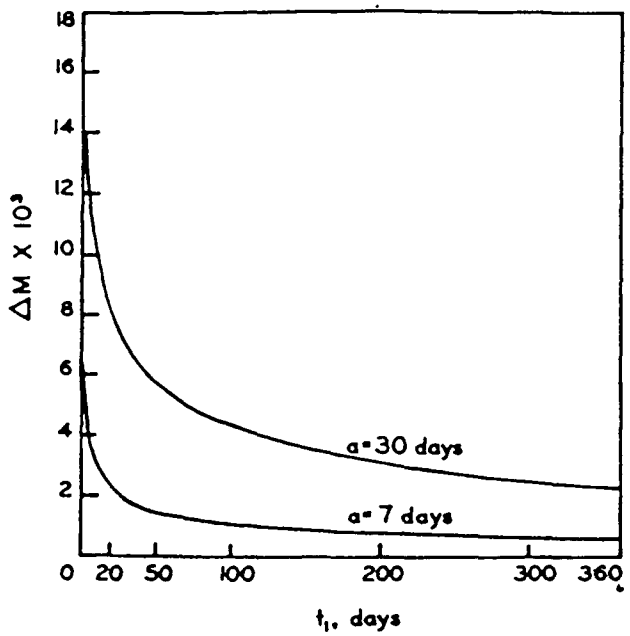

Fig. 7. Fraction of original VCM content entering pipe contents (ΔM) for 7 and 30-day extraction periods vs pipe age at start of extraction (t_1), calculated for ⅛ in. wall thickness and 30°C, Case III.

storage conditions of the pipe samples, and c) the difficulty of analysis of water for extremely low levels of VCM.

DISCUSSION

While further experimental verification would be desirable, it appears that the simple approach discussed above is quite adequate for describing and predicting the migration of RVCM from PVC pipe into water. The reasonable agreement between predicted and observed migration data for this application of rigid PVC supports the premises that, a) the diffusion coefficient determined for pure PVC resins is applicable to rigid PVC pipe compounds, and b) contact of PVC with water produces little change in the diffusivity of VCM compared to that measured by vapor sorption/desorption techniques. Further, for such relatively thick walled products as pipes, the diffusion into the environment from the outer half of the wall and into the contents from the inner half may be treated as independent processes over normal service lifetimes.

Our simple predictive model may thus be used with some confidence for estimating the concentrations of VCM in water which might arise during actual service of PVC water pipe systems. To illustrate, *Eq 12* has been used to calculate the VCM concentrations resulting from various stagnant exposure times of water in PVC pipes of 1 mg/kg original VCM content and varied diameter, warehouse age before installation and service age. Some results of such calculations are given in *Table 2* for 2, 6 and 8 in. SDR21 pipes in service at room temperature (~23°C). While these data are presented as though they represent stagnant water situations, from known use conditions (flow rates, pipe dimensions and resultant residence time), these calculations can be shown to model dynamic flowing systems.

The figures in *Table 2* clearly show that the highest VCM concentrations would be found in new installations of recently manufactured small diameter PVC pipe after long stagnation periods. Yet even for these most extreme conditions, the predicted VCM-in-water concentrations are well below the level of 0.002 mg VCM/kg H_2O when the original residual VCM content of the pipe is 1 mg/kg or less. In actual installations, stagnation times of more than a few days are rarely encountered. A typical residence time for water in PVC pipes is believed to be about 2 days (6-8) and in this situation, the predicted VCM-in-water concentration falls in the parts-per-trillion range. Thus we may conclude that PVC pipe containing ≤1 mg/kg residual VCM will result in VCM concentrations of less than 0.002 mg VCM/kg H_2O under any expected service conditions, and, therefore, non-detectable by present analytical methods.

Table 1. Comparison of Predicted and Experimental Extraction Results, Water-Filled 1 in. I.D. ⅛ in. Wall PVC Pipe Samples, 23°C

RVCM in pipe, mg/kg	Pipe age, t	Extraction Time, a,days	VCM in water, (mg/kg) Experimental	Calculated
292	~6 mo.	3	0.021	0.0257
292	~6 mo.	7	0.0414	0.0598
292	~6 mo.	14	0.113	0.118
177	~6 mo.	3	0.0173	0.0156
177	~6 mo.	7	0.0335	0.0362
177	~6 mo.	14	0.056	0.0717
22	~6 mo.	3	0.0006	0.0019
22	~6 mo.	7	0.0022	0.0045
22	~6 mo.	14	0.0046	0.0089
29	~1 yr.	14	0.0105	0.0084

Table 2. Calculated VCM in H_2O Concentrations for Stagnant Storage of Water in PVC Pipes of Varied Size and Age and Original 1 mg/kg Residual VCM Content

Pipe size (SDR21)	Warehouse age, days	Service age, years	VCM in water after given storage times, mg/kg 2 days	2 weeks	1 month
2 in.	30	0(new)	.00007	.00044	.00087
	60	0	.00005	.00033	.00067
	90	0	.00004	.00027	.00056
	90	1	.0000180	.000125	.000265
	90	2	.00001	.00009	.000199
	90	5	.00001	.00006	.00013
6 in.	30	0	.00002	.00015	.00029
	60	0	.00002	.00011	.00022
	90	0	.00001	.00009	.00019
	90	1	.000006	.000042	.000088
	90	2	.000004	.00003	.000066
	90	5	.000003	.00002	.00004
8 in.	30	0	.00002	.00011	.00022
	60	0	.00001	.00008	.00016
	90	0	.00001	.00007	.00014
	90	1	.0000045	.000031	.000066
	90	2	.000003	.00002	.000050
	90	5	.000002	.00001	.00003

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