C.D. Smith
11 May 1974

EPIDEMIOLOGICAL STUDY OF VINYL CHLORIDE WORKERS

Final Report

May 3, 1974

Submitted to

The Manufacturing Chemists Association
1825 Connecticut Avenue N. W.
Washington, D. C. 20009

by

Tabershaw/Cooper Associates, Inc.
2180 Milvia Street
Berkeley, California 94704

ACKNOWLEDGMENT

The research which forms the basis for this report was supported by the voluntary contributions of the following named chemical companies engaged in the synthesis and/or the polymerization of vinyl chloride, and administered in their behalf by the Manufacturing Chemists Association

Air Products and Chemicals, Inc.

Allied Chemical Corporation

Borden Chemical Division of Borden, Inc.

Continental Oil Company

Diamond Shamrock Company

The Dow Chemical Company

Ethyl Corporation

Exxon Chemical Company

Firestone Plastics Company

B. F. Goodrich Chemical Company

The Goodyear Tire and Rubber Company

Monsanto Company

Olin Corporation

PPG Industries, Inc.

Shell Chemical Company

Stauffer Chemical Company

Tenneco Chemicals, Inc.

Union Carbide Corporation

UNIROYAL, Inc.

TABLE OF CONTENTS

LIST OF TABLES

FIGURE

ATTACHMENTS

EPIDEMIOLOGICAL STUDY OF VINYL CHLORIDE WORKERS

Final Report - May 2, 1974

I. SUMMARY

Eight thousand three hundred and eighty four men who had had at least one year of occupational exposure to vinyl chloride before December 31, 1972, were followed in a historical prospective mortality study. The major findings of the study are:

1. The overall mortality of the study population was approximately 75 percent of what would be expected in a comparable population of U. S. males.
2. No cause of death showed a statistically significant excess over what would be expected in a comparable U. S. male population.
3. No new deaths identified as angiosarcoma of the liver were found.
4. All previously known Angiosarcoma deaths in the study population during the study period were found as part of the routine study procedure.
5. Cancers of the liver (primarily angiosarcoma), respiratory system, brain, and cancers of unknown primary site, as well as lymphosarcoma, occurred more often than expected in those members of the study population with the greatest exposure. Even though the excesses were not statistically significant, the findings warrant further study.
6. Other cancers occurred at lower levels than in the general male population, with the exception of cancers of the buccal cavity and pharynx. There was an excess of these

cancers, which however was inversely related to exposure. The explanation for this finding is not apparent.

II. INTRODUCTION

The study was begun by Tabershaw/Cooper, Associates, Inc. on June 15, 1973, under contract from the Manufacturing Chemists Association. The original eight month period of the contract was later extended by two months, in consideration of logistical problems arising from the number of plants which participated in the study.

The objectives of the study are:

(1) To compare the mortality of individuals who have worked in vinyl chloride plants with that of the general population;

(2) To compare mortality patterns within the population of vinyl chloride workers, based upon estimated occupational exposure; and

(3) To compare mortality among vinyl chloride workers with the mortality of other groups which TCA, Inc. has studied.

The study took the form of a historical prospective mortality study. The study population consisted of individuals who had worked for at least one year in a job involving exposure to vinyl chloride before December 31, 1972, and included retired and terminated as well as active workers. For each such worker the date of birth and an employment history were obtained, and the vital status of the worker as of December 31, 1972, was ascertained. For those found to have died, those death certificates that were available were obtained and the cause of death determined. The observed mortality was compared with that of the United States male population.

III. SELECTION OF STUDY PLANTS

The Manufacturing Chemists Association identified 43 United States plants belonging to 19 companies, which either produced vinyl chloride or used it in the production of polyvinyl chloride. The name and address of a responsible liaison person at each company and plant was provided to TCA, Inc. (Attachment A).

A brief questionnaire was sent to each plant to obtain an estimate of the exposed population and to find out how far back in time personnel records could be obtained, and to determine whether job histories were available for retired and terminated workers (Attachment B).

All the plants responded. On the basis of the replies, four plants were dropped from the study because they had begun operation after 1968. It was felt that exposures in such plants would be too recent and too brief to provide useful data. Study of one other plant was deferred because it had stopped production of vinyl chloride seven years ago, and appeared to present difficulties in identifying an exposed population.

In addition, three more plants were excluded from analysis because, when data analysis began, it was found that information from these plants was defective in one of several respects (e.g., no exposure data, no job histories available for terminated employees, etc.).

The present report therefore deals with 35 domestic plants involved in either VCM or PVC production, which provided a study population of 8384 individuals.

IV. DATA COLLECTION

In each plant, data were collected for each worker stated by the plant management to have been employed for at least one year in a job involving exposure to vinyl chloride. In some plants, particularly those producing the monomer, this determination could be made on the basis of job titles. Usually, however, exposure was a function of both job title and the location of the job in the plant, so that the assessment of exposure had to be made on a case-by-case basis by plant officials.

Data were collected for as far back in time as complete records were kept. In most cases this covered the entire history of the plant. In others, records were kept for a fixed period such as a decade. In a few, records were kept for different periods, depending on whether the worker had died on the job or had left employment.

In the latter case it was necessary to exercise care in defining the period for which study data were to be collected. For example, if a plant kept records for five years on those who left employment, but kept them indefinitely for those who died on the job, no termination could be included in the study unless it occurred within the last five years. Data from the earlier period would be biased for the purpose of this study, since a worker had to die to be in the records for the earlier period. The policy therefore was to determine how long each kind of record was kept (quit, discharged, retired and alive, retired and deceased, died while employed) and to define the study period as the shortest of these.

One problem which arose in the PVC plants resulted from the fact that a worker could transfer within the plant from exposed to non-exposed jobs. Therefore, even though the number of workers in exposed jobs at a given

time may have been a small proportion of the total work force, it was necessary to search every worker's record to find those who might in the past have had over a year's experience in exposed jobs. Several of the PVC plants undertook to conduct this search themselves.

As a result, plant data were collected in two ways. In some plants, a TCA, Inc., team visited the establishment, copied the records designated by management as representing exposed workers, and obtained sufficiently detailed exposure estimates to produce a job history. In others, the plants were provided by TCA, Inc. with standard forms which they filled out for workers whom they identified as exposed. Three basic forms were used, depending on whether the worker was known to be alive, known to be dead, or was of unknown status. In all three cases a work history with estimated exposure was recorded, the differences being in the ancillary data required. Attachment C shows such a form for workers known to be dead.

Approximately 5000 of the study population came from plants visited by TCA, Inc., while the remainder came from plants which had filled out the forms themselves.

V. THE MEASUREMENT OF EXPOSURE

The job history forms (Attachment C) were originally designed in the expectation that vinyl chloride exposure on each job could be quantified in parts per million. This proved generally to be impossible. However, industrial hygiene and safety personnel in each plant were able to identity certain jobs and locations as involving the highest exposures in the plant, and to classify other exposures as medium or low relative to the "high" represented by the jobs with the greatest exposure. Therefore, the attempt

to obtain objective exposure data was abandoned, and each exposed job in the worker's history was scored 1, 2, or 3 to indicate low, medium or high estimated exposure.

This gross classification has two major failings, as a result of the subjective nature of the estimates. The first is that the scores represent estimated relative exposure within a given plant. It is therefore possible that, in objective terms, a "high" score in one plant corresponds to a "medium" or even "low" score in another. The second is that exposures in most plants have tended to decrease over time, so that a worker with long service may have had jobs in the remote past which involved "low" exposure relative to other jobs at that time, but which might be "high" in comparison with current exposures in the same job. This subjective classification is therefore of questionable validity in characterizing the exposure of a given worker. For epidemiological purposes, however, those who have high scores can reasonably be expected, on the average, to have had the greatest exposure, while those with low scores will have had the least, even though the true exposure in each group may vary considerably from person to person.

The estimated exposure history of each worker was summarized by calculating An Exposure Index (EI). This was done by multiplying the number of months on each job by the exposure score, totalling these overall exposed jobs, and dividing by the total number of months of exposure.

VI. FOLLOW-UP OF STUDY POPULATION

A follow-up procedure was instituted for those who had left employment and whose vital status could not be determined at the local plant. A portion of the terminations was traced by mail, using a form letter sent to the last

known address. Attachment D shows such a letter. If the letter was returned as undeliverable, the procedure shown in Figure 1 was used to obtain a current address.

The remainder of the terminations was traced through a retail credit bureau. The latter was also done for a portion of those who could not be found by the mail follow-up.

Approximately 5500 of the study population came from plants in which direct mail follow up was employed. In the remainder of the population follow-ups was by retail credit bureau. There was no significant difference between the success rates of the two methodologies.

Table 1 shows the vital status of the study population as of December 31, 1972. Follow-up is 85 percent complete, although follow-up data were still being received at the time the files were closed for this report. Of the 352 workers known to be dead, death certificates have been received for all but 24.

The mortality calculations in the succeeding sections of this report are based only on those workers who were successfully traced, which is equivalent to assuming that mortality among those not found is the same as among those who were found. In order to examine the reasonableness of this assumption, the differences between those found and not found are examined briefly as part of the following section.

VII. DESCRIPTION OF STUDY POPULATION

Table 2 shows the distribution of those successfully traced, by year of birth and number of months of exposure. The median birth year was 1931 (compared with 1920 for those not found) and the median duration of employment in an exposed job was 80 months (44 months in those not found). The

shortest exposures occur in both the oldest men, most of whoseworking life was over when vinyl chloride came into general use, and the youngest men, whose date of hire was closer to the closing date of the study.

Although nearly half of the study population had less than 60 months of exposed employment, there were nevertheless 855 workers with 20 years or more exposure, and 1641 with 15 years or more.

Table 3 shows the distribution by duration of exposure and the year in which exposure began. The median year in which exposure began was 1962 (versus 1953 for those not found). Notice that almost half the study population first entered exposed employment in the decade 1960-69. This reflects the fact that a great many plants first began vinyl chloride operations in that decade and means that these workers have not been followed for any substantial length of time after their exposure occurred.

Table 4 shows the relationship between duration of exposure and Exposure Index (EI). The median EI is 1.44 (1.75 for those not found). There does not appear to be a close relationship between the EI and the duration of exposure, that is, workers with a higher EI do not differ substantially in duration of exposure from those with a lower EI. One implication is that in assessing the relationship between mortality and exposure, both duration and level of exposure should be examined separately, as well as in combination.

The data shown above for those not found indicates that they were born (and began their exposure) about 10 years before the group on which follow-up was complete, and had about half the duration of employment with a slightly higher EI. Typically, their employment in an exposed job ended before 1960. The bulk of these workers came from older plants from

which data were collected late in the study.

Although there appears to be nothing very unusual about this group in terms of work history and exposure, it is nevertheless true that their exposures took place further back in time than that of the group successfully traced. It is therefore possible that their mortality, after a substantial latent period, might show a somewhat different pattern of mortality from the traced group.

VIII. CALCULATION OF RISK OF DEATH

The usual method of describing the risk of death in a study of this kind is to express the number of deaths which actually occurred as a percentage of the number which would have been expected in a comparable male population observed over the same age and time intervals. This statistic is called the Standardized Mortality Ratio (SMR).

In order to calculate the number of expected deaths it is first necessary to calculate the number of years for which each worker was observed, and to classify these years of observation by the age intervals into which they fell and the birth date of the worker.

Table 5 shows such a calculation for the 7128 vinyl chloride workers on whom follow-up was complete. This group represents 77,846 person-years of observation. For example, the table shows that for workers born in the years 1925-29, there were 2725 person-years of observation in the age interval 30-34, and an easy calculation shows that these person-years were lived in a period whose midpoint was about 1960. Therefore, the number of deaths to be expected in this cell of the table is the number of deaths occurring in 1960 among 2725 males aged 30-34 in a comparable non-exposed population.

In the present study the U. S. male population was used as the standard of comparison, and the age-specific U. S. male death rates for the appropriate ages and years were applied to the corresponding cells of Table 5 to obtain the expected deaths.[1] This was done for all causes of death together, and for each of the 35 causes for which detailed mortality rates are published on a national basis (omitting such causes as breast cancer, congenital malformations, and diseases of infancy).

Table 6 shows observed and expected deaths, and the SMR, for each of these causes for the total study group. In calculating the SMR's for specific causes, the 24 deaths for which no certificates were found were assumed to have the same cause distribution as those for which certificates were available.

In the standard population, each SMR would be equal to 100. Therefore, the statistical significance of the deviation of each SMR in the study population from the expected value of 100 was tested. A single asterisk indicates those SMR's which differed significantly from 100 at the 5 percent level, that is, which had a probability of .05 or less of occurring by chance. A double asterisk indicates those which were significant at the 1 percent level. SMR's based on fewer than 5 observed cases were not tested for significance.[2] The practical interpretation of these statistical statements will be examined below.

---

[1] Each diagonal in Table 5 corresponds to experience centered around a given year. For the first three diagonals (from left to right) 1950 rates were used; for the next three, 1955, 1959 and 1965 rates were used; for the last two, 1967 rates were used.

[2] Large-sample tests of significance were derived from: Chiang, C. L., "Standard error of the age-adjusted death rate," Vital Statistics Special Reports, 47 (1961) pp 275-285.

Table 7 shows the same SMR's for workers with an Exposure Index below 1.5 versus those at 1.5 or above. The dividing point of 1.5 represents a level halfway between "low" and "medium."

Table 8 shows similar results for workers with less than 5 years exposure versus those with 5 years or more.

In order to examine the possible interaction between duration and level of exposure, the study population was divided into 4 groups on the basis of both EI (low vs high) and duration of exposure (short vs long) using the same dichotomization as Tables 7 and 8.

Table 9 shows the results for short versus long exposure in the low EI group, and Table 10 shows the same comparison in the high EI group.

In each of the above tables, deaths for which certificates had not been received were assumed to be distributed as a uniform percentage of all causes. The cause specific SMR's were therefore adjusted upward by a percentage which varied in each subgroup. Table II shows the distribution of uncertified deaths in each group, and the resulting percentage by which the cause specific SMR's were increased.

IX. ANALYSIS

The overall mortality of the study population is statistically significantly lower than that of the U. S. male population. There were 352 observed deaths compared with 467 expected, for an SMR of 75.

Table 6 shows that no specific cause of death was statistically significantly greater than expected. Several, particularly heart disease, accidents and "other diseases" not detailed in the tables, were significantly below their expected values.

When the study population is divided according to length and duration of exposure (Tables 7 and 8) and combinations of these measurements (Tables 9 and 10) three major patterns emerge.

For malignant neoplasms as a whole, the SMR increases with increasing exposure, whether measured by level, duration, or both. In the high exposure group with 5 years or more exposure (Table 10) there are 36 observed cases and 26.11 expected.

For cardiovascular - renal diseases as a group, there are also increases in the SMR with increasing exposure, but the number of observed cases remain less than expected, the differences being statistically significant in all groups except the high exposure, long duration group in Table 11.

For all other causes, there are no consistent relationships with exposure.

Within the malignant neoplasms, the largest (although not statistically) significant SMR is in cancers of the buccal cavity and pharynx, with 5 observed, 2.84 expected, and an SMR of 189. However, Tables 7 to 10 show that all these cases have Exposure Indexes below 1.5, and 4 out of the 5 have less than 5 years exposure.

Cancer of the digestive system shows no excess in the study population as a whole. However, in those workers with Exposure Indexes of 1.5 or higher, there are 12 observed cases where 9.14 are expected (table 7). In the subgroup of the above workers with 5 years or more exposure, there are 11 observed cases and 7.47 expected.

Respiratory cancer shows a slight excess in the total group, and a similar pattern for different exposure categories, with 13 observed versus 10.28 expected when the Exposure Index is 1.5 or higher, and 12 observed versus 8.50 expected when, in addition, the duration of exposure is 5 years

or more.

Malignant neoplasms of other and unspecified sites show an excess in the total group, and an increase with both level and duration of exposure (Tables 7 and 8). The relationship with exposure is more pronounced, since those with exposures of less than 5 years have fewer cases than expected.

The lymphosarcomas, although occurring at about the expected rate when the whole group is considered, are concentrated almost entirely in the high exposure long duration group. In that category there are 4 cases observed and 1.84 expected.

Cancers of the genital and urinary organs, and leukemia, have fewer cases than expected. The number of cases is too small to examine any trends.

X. DISCUSSION

The favorable overall mortality of the study population is a phenomenon commonly observed in working populations. Studies of several occupational groups have shown that, even if there is an occupational hazard which greatly increases the risk of death from a particular cause, the overall mortality may well be favorable. Table 12 shows the SMR's obtained in a number of recent studies of other occupational groups. Although for technical reasons these SMR's are not strictly comparable, the overall mortality in the present study is clearly favorable.

SMR's which are higher than expected may be worthy of attention even if they are not statistically significant. This is especially true in the present study since the number of deaths from many causes is quite small, and even a relatively high SMR may not reach statistical significance.

If, in addition, a particular cause shows a consistent pattern of increase with exposure or estimated exposure, the findings are particularly

interesting.

By these criteria, mortality from digestive cancer, respiratory cancer, cancer of other and unspecified sites, and lymphosarcoma, appear to be related to exposure as estimated in this study.

In view of the association between vinyl chloride exposure and angiosarcoma of the liver, the digestive cancers were examined further to see what contribution angiosarcoma made to the observed mortality pattern.

Of the 19 digestive cancers, 7 were liver cancers, of which two were angiosarcomas according to the death certificate. However, among angiosarcoma deaths in vinyl chloride workers identified by other investigators, there were 6 which occurred in the present study population during the study period. They were all found in the course of the study, but 4 were listed on the death certificate as due to causes other than angiosarcoma. Table 13 shows these deaths with the death certificate cause.

If there had been no angiosarcomas, Table 13 shows that the total number of digestive cancers in the high exposure group would have dropped to 8, and the number with high exposure and 5 years or more exposure would have dropped to 2, so that the mortality pattern in this cause group is due entirely to angiosarcoma.

The other cause group worth further investigation is cancer of other and unspecified sites, both because it is a heterogeneous category and because it seems, unlike the other cancers, to be more related to duration than to level of exposures.

Table 14 shows a list of the specific causes included in this category, which is essentially brain cancer and generalized cancer with primary site

unknown. About 40 percent of the observed deaths were due to brain cancer. In the general male population, about 22 percent of this category is due to brain cancer, so that not only is the mortality from this cause excessive, but brain cancer is overrepresented.

The possibility exists based on the lack of specificity of some of the listed causes that some of the brain cancers were not primary, but metastases from another unidentified site such as the lung.

The cancers of the buccal cavity and pharynx are difficult to explain because of their occurrence in the low exposure - short exposure group. It is possible that this is a chance occurrence, or that exposures to other substances were involved.

XI. POSSIBLE BIASES IN THE CALCULATION OF RISK

There are three areas in which bias could have entered the calculation of the above SMR's. The first is the choice of the U. S. male population as a standard. The second is the absence of 15 percent of the population who were not found. The third is the discovery, as the study ended, of a group of 1500 workers whose exposures occurred up to 35 years ago, and who are not included in the study group.

The choice of the U. S. male population was not entirely appropriate. The study population was concentrated almost entirely in the Eastern half of the United States, whose normal mortality is slightly higher than that of the whole country. This implies that the correct expected values should have been slightly higher than the ones which were used, and the corresponding SMR's slightly lower. This has no practical significance except for those malignancies in which the SMR's were above 100. However, regional differences in mortality from those causes is such that these SMR's would have been at most 1.5 to 3 percent lower, with no substantive difference in the results.

The workers who could not be traced may conceivably contain a disproportionate number of deaths, which could increase the overall SMR. It appears unlikely that the distribution of causes would be substantially different. However, without actual data in hand, the possibility cannot be ruled out that either some new causes of death might become significant, or that some of the relationships found in this study might be diluted.

The same comments apply even more to the group of 1500 workers discovered too late to be included in the study group, since they would provide data in precisely the area (very long exposure and long latency) in which the present study is relatively weak.

References:

Archer, VE, Wagoner, JK, Lundin, FE, Jr: Cancer mortality among uranium mill workers. J Occup Med 15:11-14, 1973.

Enterline, PE, DeCoufle, P, Henderson, V: Mortality in relation to occupational exposure in the asbestos industry. J Occup Med 14:897-903, 1972.

Enterline, PE, Kendrick, MA: Asbestos-dust exposures at various levels and mortality. Arch Environ Health 15:181-186, 1967.

Hammond, EC, Selikoff, IJ: Relation of cigarette smoking to risk of death of asbestos-associated disease among insulation workers in the United States. Symposium on Biological effects of Asbestos, Lyon, October 2-5, 1972, paper #46.

Lloyd, JW, Ciocco, A: Long-term mortality study of steelworkers; methodology. J Occup Med 11:229-310, 1969.

Lundin, FE, Jr., Lloyd, JW, Smith, EM, Archer, VE, Holaday, DE: Mortality of uranium miners in relation to radiation exposure, hard-rock mining and cigarette smoking--1950 through September 1967. Health Physics 16:571-578, 1969.

Redmond, CK, Ciocco, A, Lloyd, JW, Rush, HW: Long-term mortality study of steelworkers--VI. Mortality from Malignant Neoplasms among coke oven workers. J Occup Med 14:621-629, 1972.

Tabershaw, IR, Cooper, WC, Balzer, JL: A Labor-management occupational health service in a construction industry. Arch Environ Health 21:784-788, 1970.

Table 1

Follow-up Status of 8384 Vinyl Chloride Workers

	Total	Alive	Dead	Unknown	Death Certificates	
					Rec'd.	Not Rec'd.
Number	8384	6776	352	1256	328	24
Percent	100.0	80.8	4.2	15.0	92.7	7.3

April 15, 1974

Table 2

Distribution of Months in Exposed Employment by Birth Year,
for 7128 Vinyl Chloride Workers with Completed Follow-up

Year of birth	Total	Months of exposure							Unknown
		<60	60-119	120-179	180-239	240-299	300-359	360-419	
1875-79	1	1							
1880-84	5		2	2	1				
1885-89	7	1	3	3					
1890-94	21	3	7	9	2				
1895-99	38	10	4	9	12	2			1
1900-04	89	9	13	21	22	17	6		1
1905-09	214	33	27	35	37	43	34	3	2
1910-14	404	79	62	58	72	73	43	15	2
1915-19	648	145	96	80	120	105	84	13	5
1920-24	897	229	191	98	143	160	64	8	4
1925-29	940	276	214	118	169	139	20		4
1930-34	877	287	252	128	181	26			3
1935-39	986	454	378	120	27				7
1940-44	1169	664	481	16					8
1945-49	700	632	66						2
1950-54	132	132							
Total	7128	2955	1796	697	786	565	25	39	39

April 15, 1974

Table 3

Distribution of Months in Exposed Employment by Year
in Which Exposure Began, for 7128 Vinyl Chloride Workers with Completed Follow-up

Year Exp. Started	Total	Months of Exposure							
		<60	60-119	120-179	180-239	240-299	300-359	360-419	Unknown
1930-39	35	2	4	1	4	6	13	5	
1940-49	1048	135	93	119	151	277	237	34	2
1950-59	1962	389	257	383	631	282			20
1960-69	3368	1714	1442	195					17
1970-71	715	715							
Total	7128	2955	1796	698	786	565	250	39	39

April 15, 1974

Table 4

Distribution by Months of Exposed Employment and
Exposure Index
for 7128 Vinyl Chloride Workers with Complete Follow-up

Exposure Index	Total	Months of exposure							
		<60	60-119	120-179	180-239	240-299	300-359	360-419	Unknown
1.0-1.4	4032	1715	1125	402	406	274	92	18	
1.5+	3057	1240	671	295	380	291	159	21	
Unknown	39								39
Total	7128	2955	1796	697	786	565	251	39	39

May 2, 1974

Table 5

Person Years of Observation by Year of Birth and Age for Vinyl Chloride Workers

Age	Totals	Year of Birth															
		1875 1879	880 884	1885 1889	1890 1894	1895 1899	1900 1904	1905 1909	1910 1914	1915 1919	1920 1924	1925 1929	1930 1934	1935 1939	1940 1944	1945 1949	1950 1954
Total	77846	13	92	124	317	705	1662	4055	6710	11038	14305	13807	9408	7239	6174	2023	174
15-19	134										1	11	12	5	53	21	31
20-24	4070									39	180	572	440	475	920	1301	143
25-29	12076								32	335	1090	1882	1642	2173	4221	701	
30-34	13861							14	264	1054	2183	2725	2863	3778	980		
35-39	13550						3	221	697	1685	2742	3688	3706	808			
40-44	12031					5	70	524	1028	2044	3502	4113	745				
45-49	9527					56	241	710	1226	2631	3847	816					
50-54	6368				35	135	334	762	1574	2768	760						
55-59	3632			22	88	155	363	386	1636	482							
60-64	1684		14	35	77	142	341	322	253								
65-69	632	2	25	31	62	126	270	116									
70-74	202	5	23	20	43	71	40										
75-79	58	5	15	11	12	15											
80-84	16	1	10	5													
85-89	5		5														

April 15, 1974

BLE 6

OBSERVED DEATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS

Cause of death with I.C.D. number	obs/exp	SMR[1]
All causes	352/467.28	75**
Tuberculosis (001-019)	1/5.71	19
Tuberculosis of respiratory system (001-008)	1/5.34	20
Malignant neoplasms (140-205)	79/77.16	110
Malignant neoplasms, buccal cavity and pharynx (140-148)	5/2.84	189
Malignant neoplasms, digestive organs and peritoneum (150-159)	19/21.67	94
Malignant neoplasms, respiratory system (160-164)	25/23.93	112
Malignant neoplasms, genital organs (170-179)	3/3.55	91
Malignant neoplasms, urinary organs (180-181)	1/3.60	30
Malignant neoplasms, other and unspecified sites (190-199)	17/11.75	155
Leukemia and aleukemia (204)	3/3.77	85
Lymphosarcoma, lymphatic and hematopoietic tissues (200-203, 205)	6/6.06	106
Diabetes mellitus (260)	7/6.31	120
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	155/207.46	80**
Vascular lesions affecting CNS (330-334)	13/24.48	57**
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	5/6.87	78
Arteriosclerotic heart disease (420)	121/137.33	95
Nonrheumatic endocarditis (421, 422)	1/6.58	16
Hypertensive heart disease (440-443)	3/9.33	35
Other hypertensive disease (444-447)	3/2.60	124
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/4.27	0
Influenza and pneumonia (480-493)	5/9.96	54*
Ulcer of stomach and duodenum (540,541)	2/3.83	56
Appendicitis (550-553)	0/0.66	0
Hernia and intestinal obstruction (560, 561, 570)	1/1.51	71
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/1.31	82
Cirrhosis of liver (581)	3/15.60	21
Hyperplasia of prostate (610)	0/0.39	0
Symptoms, senility and ill-defined conditions (780-795)	1/7.34	15
All other diseases (residual)	21/45.78	49**
Motor vehicle accidents (810-835)	17/32.70	56**
Other accidents (800-802, 840-962)	18/30.64	63**
Suicide (963, 970-979)	16/16.83	102
Homicide (964, 980-985)	1/11.98	9
Number of workers	7128	
Person-years	77846	

[1]SMR's adjusted for deaths with cause unknown. See Table 11.
*Significant at 5% level.
**Significant at 1% level.

May 2, 1974

OBSERVED DEATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS, BY ESTIMATED LEVEL OF EXPOSURE

Cause of death with I.C.D. number	EI <1.5 obs/exp	EI <1.5 SMR[1]	EI ≥1.5 obs/exp	EI ≥1.5 SMR[1]
All causes	188/270.33	70**	157/195.68	80**
Tuberculosis (001-019)	0/3.38	0	0/2.33	0
Tuberculosis of respiratory system (001-008)	0/3.16	0	0/2.18	0
Malignant neoplasms (140-205)	37/44.28	90	41/32.67	134
Malignant neoplasms, buccal cavity and pharynx (140-148)	5/1.62	330	0/1.21	0
Malignant neoplasms, digestive organs and peritoneum (150-159)	7/12.50	60*	12/9.14	141
Malignant neoplasms, respiratory system (160-164)	11/13.56	86	13/10.28	135
Malignant neoplasms, genital organs (170-179)	2/2.30	93	1/1.43	75
Malignant neoplasms, urinary organs (180-181)	1/2.07	51	0/1.52	0
Malignant neoplasms, other and unspecified sites (190-199)	9/6.57	146	8/4.52	190
Leukemia and aleukemia (204)	1/2.18	49	2/1.57	136
Lymphosarcoma, lymphatic and hematopoietic tissues (200-203, 205)	1/3.48	31	5/2.54	212
Diabetes mellitus (260)	5/3.65	146	2/2.65	81
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	84/120.11	75**	69/86.99	85*
Vascular lesions affecting CNS (330-334)	7/14.42	52**	6/10.06	64
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	3/3.98	80	2/2.85	75
Arteriosclerotic heart disease (420)	68/78.94	92	51/58.05	95
Nonrheumatic endocarditis (421, 422)	0/4.20	0	1/2.89	38
Hypertensive heart disease (440-443)	1/5.48	19	2/3.86	56
Other hypertensive disease (444-447)	1/1.52	70	2/1.07	201
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/2.50	0	0/1.77	0
Influenza and pneumonia (480-493)	5/5.80	92	0/4.13	0
Ulcer of stomach and duodenum (540, 541)	1/2.21	48	1/1.60	68
Appendicitis (550-553)	0/0.39	0	0/0.27	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.88	0	1/0.63	171
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	0/0.76	0	1/0.55	196
Cirrhosis of liver (581)	2/8.90	23	1/6.64	16
Hyperplasia of prostate (610)	0/0.25	0	0/0.14	0
Symptoms, senility and ill-defined conditions (780-795)	0/4.22	0	1/3.09	34
All other diseases (residual)	14/21.90	68*	6/15.89	41**
Motor vehicle accidents (810-835)	8/19.08	45**	9/13.46	72
Other accidents (800-802, 840-962)	11/17.83	66*	6/12.67	50**
Suicide (963, 970-979)	9/9.73	98	7/7.02	107
Homicide (964, 980-985)	0/6.98	0	1/4.94	21
Number of workers	4032		3057	
Person-years	45354		32108	

[1]SMR's adjusted for deaths with cause unknown. See Table 11.
*Significant at 5% level.
**Significant at 1% level.

May 2, 1974

OBSERVED DEATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS
BY DURATION OF EXPOSED EMPLOYMENT

Cause of death with I.C.D. number	<60 months obs/exp	<60 months SMR[1]	≥60 months obs/exp	≥60 months SMR[1]
All causes	94/140.53	67**	251/329.30	76**
Tuberculosis (001-019)	0/2.23	0	0/3.55	0
Tuberculosis of respiratory system (001-008)	0/2.07	0	0/3.33	0
Malignant neoplasms (140-205)	13/19.96	78	65/57.61	116
Malignant neoplasms, buccal cavity and pharynx (140-148)	4/0.70	688	1/2.16	47
Malignant neoplasms, digestive organs and peritoneum (150-159)	2/5.26	46	17/16.56	106
Malignant neoplasms, respiratory system (160-164)	3/5.52	65	21/18.51	116
Malignant neoplasms, genital organs (170-179)	0/0.99	0	3/2.76	112
Malignant neoplasms, urinary organs (180-181)	0/0.83	0	1/2.79	37
Malignant neoplasms, other and unspecified sites (190-199)	2/3.40	71	15/8.23	187
Leukemia and aleukemia (204)	1/1.25	96	2/2.53	81
Lymphosarcoma, lymphatic and hematopoietic tissues (200-203, 205)	1/2.01	60	5/4.07	126
Diabetes mellitus (260)	2/1.80	134	5/4.54	113
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	28/51.45	65**	125/157.39	81**
Vascular lesions affecting CNS (330-334)	4/5.97	81	9/18.71	49**
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	2/2.39	101	3/4.53	68
Arteriosclerotic heart disease (420)	21/32.54	78*	98/105.39	96
Nonrheumatic endocarditis (421, 422)	0/1.79	0	1/5.35	20
Hypertensive heart disease (440-443)	1/2.42	49	2/7.01	30
Other hypertensive disease (444-447)	0/0.79	0	3/1.82	170
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/1.55	0	0/2.76	0
Influenza and pneumonia (480-493)	3/2.93	123	2/7.09	29
Ulcer of stomach and duodenum (540,541)	1/1.07	112	1/2.78	37
Appendicitis (550-553)	0/0.24	0	0/0.43	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.42	0	1/1.10	93
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/0.40	301	0/0.92	0
Cirrhosis of liver (581)	1/4.49	26	2/11.18	18
Hyperplasia of prostate (610)	0/0.07	0	0/0.33	0
Symptoms, senility and ill-defined conditions (780-795)	1/2.28	53	0/5.09	0
All other diseases (residual)	4/11.97	40	16/26.34	63*
Motor vehicle accidents (810-835)	10/16.14	75	7/16.65	43**
Other accidents (800-802, 840-962)	7/12.94	65*	10/17.82	58*
Suicide (963, 970-979)	6/6.34	114	10/10.28	100
Homicide (964, 980-985)	1/5.80	20	0/6.20	0
Number of workers	2955		4134	
Person-years	34201		43240	

[1]SMR's adjusted for deaths with cause unknown. See Table 11.
*Significant at 5% level.
**Significant at 1% level.

May 2, 1974

TABLE 9

OBSERVED ATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS WITH EXPOSURE INDICES BELOW 1.5, BY DURATION OF EXPOSED EMPLOYMENT

Cause of death with I.). number	<60 months exposure obs/exp	<60 months exposure SMR[1]	≥60 months exposure obs/exp	≥60 months exposure SMR[1]
All causes	56/89.23	63**	132/181.28	73**
Tuberculosis (001-019)	0/1.41	0	0/1.97	0
Tuberculosis of res atory system (001-008)	0/1.31	0	0/1.85	0
Malignant neoplasms (1 205)	8/12.86	73	29/31.46	95
Malignant neoplasms uccal cavity and pharynx (140-148)	4/0.45	1036	1/1.17	88
Malignant neoplasms igestive organs and peritoneum (150-159)	1/3.43	34	6/9.08	68
Malignant neoplasms espiratory system (160-164)	2/3.58	65	9/10.00	93
Malignant neoplasms enital organs (170-179)	0/0.68	0	2/1.62	127
Malignant neoplasms rinary organs (180-181)	0/0.55	0	1/1.53	67
Malignant neoplasms ther and unspecified sites (190-199)	1/0.97	120	8/4.43	187
Leukemia and aleuke (204)	0/0.79	0	1/1.40	73
Lymphosarcoma, lymp ic and hematopoietic tissues (200-203,205)	0/1.26	0	1/2.23	46
Diabetes mellitus (260	2/1.15	203	3/2.50	124
Major cardiovascular a renal diseases (330-334, 400-468, 592-594)	21/33.38	73**	63/86.82	75**
Vascular lesions af ting CNS (330-334)	2/3.93	59	5/10.51	49*
Rheumatic fever & c nic rheumatic heart dis. (400-402, 410-416)	2/1.50	155	1/2.49	41
Arteriosclerotic he disease (420)	16/21.14	88	52/57.87	93
Nonrheumatic endoca tis (421, 422)	0/1.18	0	0/3.02	0
Hypertensive heart ease (440-443)	1/1.58	74	0/3.90	0
Other hypertensive ease (444-447)	0/0.50	0	1/1.02	101
Chronic & unspecifi nephritis & renal sclerosis (592-594)	0/0.97	0	0/1.53	0
Influenza and pneumoni 480-493)	3/1.86	188	2/3.95	53
Ulcer of stomach and d enum (540, 541)	0/0.68	0	1/1.53	67
Appendicitis (550-553)	0/0.15	0	0/0.24	0
Hernia and intestinal truction (560, 561, 570)	0/0.27	0	0/0.61	0
Gastritis, duodenitis, teritis and colitis (543, 571, 572)	0/0.26	0	0/0.51	0
Cirrhosis of liver (58	1/2.80	42	1/6.09	16
Hyperplasia of prostat 610)	0/0.05	0	0/0.20	0
Symptoms, senility and l-defined conditions (780-795)	0/1.43	0	0/2.79	0
All other diseases (res ual)	4/7.45	63	10/12.92	79
Motor vehicle accidents 810-835)	3/9.87	35	5/9.22	56
Other accidents (800-8 840-962)	3/7.98	44	8/9.85	83
Suicide (963, 970-979)	3/4.08	86	6/5.66	109
Homicide (964, 980-985)	0/3.55	0	0/3.43	0
Number of workers	1715		2317	
Person-years	21418		23920	

[1]SMR's adjusted for de s with cause unknown. See table 11.
*Significant at 5% leve
**Significant at 1% lev .

May 2, 1974

TABLE 10

OBSERVED DEA S/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS WIT EXPOSURE INDICES OF 1.5 OR GREATER, BY DURATION OF EXPOSED EMPLOYMENT

Cause of death with I.C.D number	<60 months exposure obs/exp	<60 months exposure SMR[1]	≥60 months exposure obs/exp	≥60 months exposure SMR[1]
All causes	38/47.93	79	119/147.81	81*
Tuberculosis (001-019)	0/0.76	0	0/1.57	0
Tuberculosis of respor ory system (001-008)	0/0.71	0	0/1.48	0
Malignant neoplasms (140- 5)	5/6.57	96	36/26.11	141
Malignant neoplasms, b cal cavity and pharynx (140-148)	0/0.23	0	0/0.99	0
Malignant neoplasms, d estive organs and peritoneum (150-159)	1/1.67	76	11/7.47	151
Malignant neoplasms, r piratory system (160-164)	1/1.79	71	12/8.50	144
Malignant neoplasms, g ital organs (170-179)	0/0.29	0	1/1.41	73
Malignant neoplasms, u nary organs (180-181)	0/0.26	0	0/1.26	0
Malignant neoplasms, o er and unspecified sites (190-199)	1/1.18	107	7/3.51	204
Leukemia and aleukemia 204)	1/0.44	288	1/1.13	90
Lymphosarcoma, lymphat and hematopoietic tissues (200-203, 205)	1/0.71	178	4/1.84	222
Diabetes mellitus (260)	0/0.61	0	2/2.04	100
Major cardiovascular and nal diseases (330-334, 400-468, 592-594)	7/16.54	54**	62/70.46	90
Vascular lesions affec ng CNS (330-334)	2/1.87	135	4/8.19	50
Rheumatic fever & chro c rheumatic heart dis. (400-402, 410-416)	0/0.82	0	2/2.04	100
Arteriosclerotic heart isease (420)	5/10.41	61*	46/47.65	98
Nonrheumatic endocardi s (421, 422)	0/0.57	0	1/2.32	44
Hypertensive heart dis se (440-443)	0/0.76	0	2/3.10	66
Other hypertensive dis se (444-447)	0/0.27	0	2/0.81	253
Chronic & unspecified n obritis & renal sclerosis (592-594)	0/0.54	0	0/1.23	0
Influenza and pneumonia ( )-493)	0/0.99	0	0/3.13	0
Ulcer of stomach and duod m (540, 541)	1/0.35	362	0/1.25	0
Appendicitis (550-553)	0/0.08	0	0/0.19	0
Hernia and intestinal obs ction (560, 561, 570)	0/0.14	0	1/0.49	209
Gastritis, duodenitis, ent ritis and colitis (543, 571, 572)	1/0.14	904	0/0.41	0
Cirrhosis of liver (581)	0/1.56	0	1/5.08	20
Hyperplasia of prostate ( ))	0/0.01	0	0/0.13	0
Symptoms, senility and ill lefined conditions (780-795)	1/0.80	158	0/2.30	0
All other diseases (resid l)	0/4.02	0	6/11.88	51*
Motor vehicle accidents ( )-835)	7/6.05	146	2/7.43	28
Other accidents (800-802, 0-962)	4/4.73	107	2/7.96	26
Suicide (963, 970-979)	3/2.40	158	4/4.62	88
Homicide (964, 980-985)	1/2.18	58	0/2.76	0
Number of workers	1240		1817	
Person-years	12828		19305	

[1]SMR's adjusted for deaths with cause unknown. See Table 11.
*Significant at 5% level.
**Significant at 1% level.

May 2, 1974

Table 11

Distribution of Deaths by Exposure Category
and by Whether Cause of Death Was Determined,
and Correction Factors Used for SMR Calculations

		Exposure Categories							
	Total*	Exposure Index		Total Exposure Time in Months		Exposure Index and Total Exposure in Months			
		<1.5	≥1.5	<60	≥60	<1.5 & <60	<1.5 & ≥60	≥1.5 & <60	≥1.5 & ≥60
All causes	352	188	157	94	251	56	132	38	119
Cause known	328	176	146	78	244	48	128	30	116
Cause unknown	24	12	11	16	7	8	4	8	3
Percent increase in SMR's	7.3	6.8	7.5	20.5	2.8	16.6	3.1	26.6	2.5

*Deaths in exposure categories do not add up to deaths in total category because 7 deaths had unknown exposure data, and one of those was of unknown cause.

May 2, 1974

Table 12

Results of Follow-up in Several Occupational Groups

Study Population	Number Observed	Years of Observation	SMR All causes	Reference
Asbestos building product workers	12,402	1951-1963	89	Enterline & Kendrick, 1967
Asbestos friction material workers	7,510	"	89	Enterline & Kendrick, 1967
Asbestos textile plant workers	1,843	"	121	Enterline & Kendrick, 1967
Cotton textile workers	6,281	"	88	Enterline & Kendrick, 1967
White underground uranium miners	3,414	1950-1967	159	Lundin et al, 1969
Steelworkers	59,072	1953-1961	82	Lloyd & Ciocco, 1969
Asbestos insulation workers > 20 years	250	1946-1965	140	Tabershaw et al, 1970
Coke oven workers	4,661	1951-1966	98	Richmond et al, 1972
Asbestos maintenance service, etc.	438	1941-1969	124	Enterline et al, 1972
Asbestos insulation workers > 20 years	370	1963-1971	198	Hammond & Selikoff, 1972
Asbestos insulation workers	17,800	1967-1971	136	Hammond & Selikoff, 1972
Uranium mill workers	662	1950-1967	99	Archer et al, 1973
Lead smelter workers	2,352	1946-1970	107	TCA, Inc., unpublished
Lead battery plant workers	4,680	1946-1970	99	TCA, Inc., unpublished
Petroleum refinery workers	18,916	1962-1971	68	TCA, Inc., unpublished

May 3, 1974

Table 13

Angiosarcoma Deaths in VCM Epidemiology Study Population

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
4250	54	203	Cirrhosis of liver (sev. weeks)	yes
4255	60	281	Bleeding from hepatoma (3 days) (Laennecs cirrhosis)	yes
5765	45	167	Angiosarcoma rt. lobe of liver (10 mos.)	yes
7303	38	174	Liver failure (1 mo.) Cancer of liver, primary (15 mo.)	-
7376	52	238	Cardiac tampanade and massive l ft hemothorax (mins.) Widely metastatic angiosarcoma of liver (4 mos.)	-
7385	43	214	Hepatic failure Primary carcinoma liver	yes

April 15, 1974

Table 14

Other Malignancies (190-199, I.C.D.) in VCM Epidemiology Study Population

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
2744	38	186	Widespread metastatic melanoma (1 yr.) Malignant melanoma of back	yes
3700	48	50	Malignant melanoma with widespread metastases	no
2096	67	69	Brain tumor (carcinoma) (8 mos.)	-
3946	43	64	Meningitis and pneumonitis (5 wks.) Ependymoma, fourth ventricle-brain (2 mos. post-op craniotomy)	no
4433	54	81	Astrocytoma, malignant-left cerebral hemisphere (1 yr.)	yes
4800	61	51	Carcinoma of the brain Arteriosclerosis Pneumonia due to static congestion	no
7279	57	271	Brain tumor, glioblastoma multiforme (18 mos.)	-
7300	44	211	Brain tumor, malignant (2 mos.)	-
7371	58	249	Brain tumor, malignant	no
7306	53	216	Thyroid carcinoma with metastases	no
5769	52	239	Carcinomatosis Carcinoma vertebral body	yes

Table 14
Other Malig ncies (cont.)

Study No.	A	at Death	Mos. Exposed	Cause as Given	Autopsy
5246		67	201	Liposarcoma with metastasis (3 mos.)	yes
2850		64	193	Carcinomatosis, primary region not known (Past history, rheumatoid arthritis, myocardial infarction)	no
4778		54	300	Cardiac arrest (1 hr.) Respiratory arrest and cerebral hypoxia (2 days) Widespread metastatic carcinoma (2 mos.)	-
4786		61	318	Generalized metastasis undifferentiated (7 mos.) Squamous cell carcinoma, primary site undetermined	yes
5783		69	166	Metastatic carcinoma of abdomen Critical site undetermined (Pulmonary emphysema)	-
7301		55	133	Carcinomatosis (4 mos.) Primary site undetermined	no

April 15, 1974

Table 15

Malignant Neoplasms of Buccal Cavity and Pharynx (140-148, I.C.D.)
in VCM Epidemiology Study Population

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
3003	31	49	Carcinoma of lip with metastasis to lung and neck (5 yrs.)	no
3431	56	30	Pulmonary edema (50 min.) Pulmonary metastases (Carcinoma, epidermoid, tongue and mandible)	no
3001	57	71	Metastatic squamous cell ca. - primary lesion palate	yes
7365	54	40	Atelectasis due to metastasis to mediastinum (4 mos.) Multiple malignant metastases to brain, liver & mediastinum (1 yr.) Adeno-carcinoma of nasal pharynx (2 yrs.)	yes
3354	63	26	Massive hemorrhage into tracheo-tree Status post laryngo-pharyngectomy, left radial neck dissection Well-differentiated keratinizing squamous cell carcinoma of left pyriform sinus (mos.)	yes

April 15, 1974

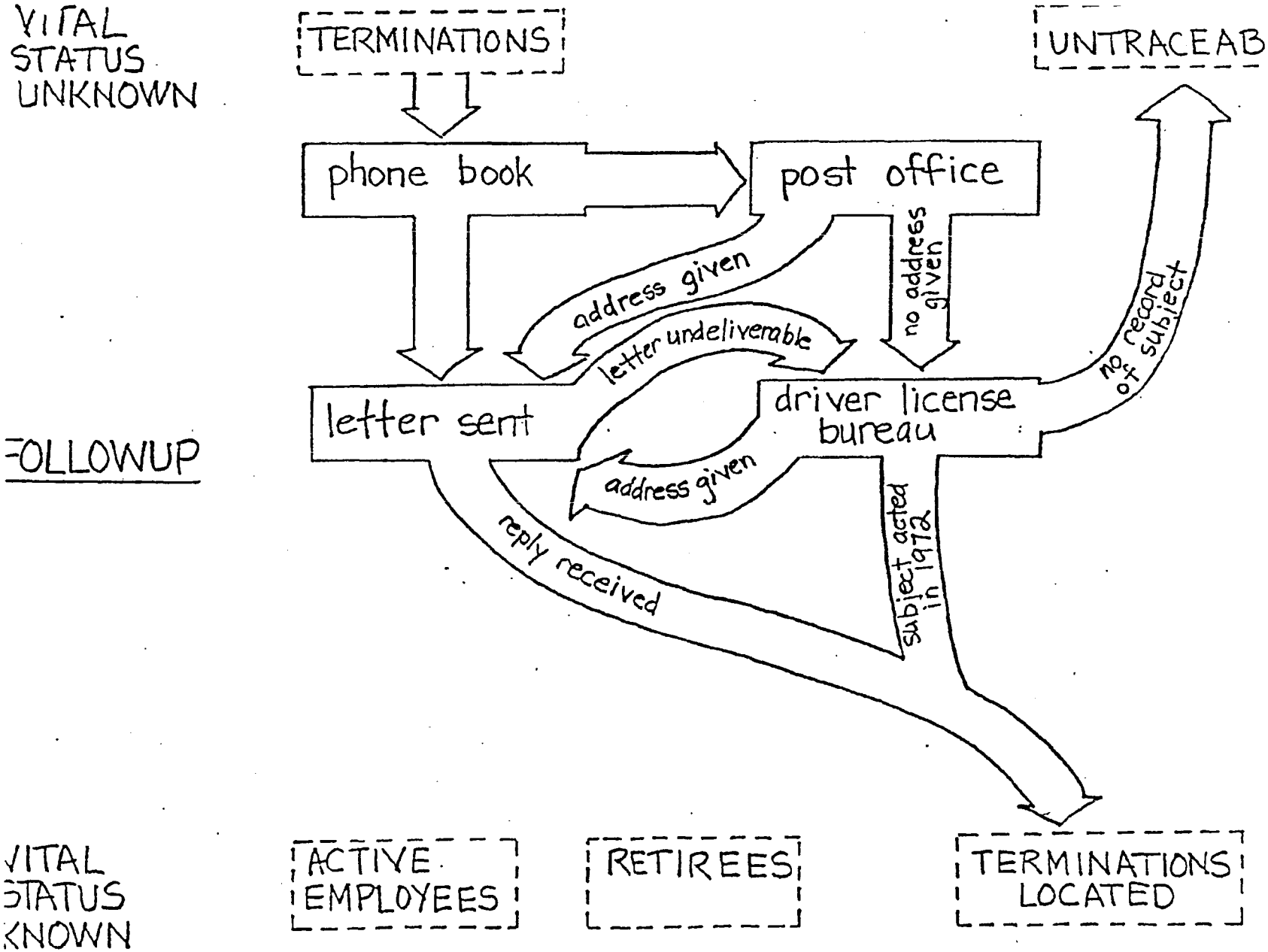

FIGURE 1

Manufacturing Chemists Association VCM Epidemiology Study

Parent Corporation _____

Plant Designation _____

Address _____

_____

Telephone ( ) - ext.

TABERSHAW-COOPER ASSOCIATES' CONTACT FOR SUBJECT STUDY:

Name _____

Title _____

Mailing Address _____

_____

Street Address (please identify with sufficient precision to serve as a taxi-driver's instruction)

_____

_____

Telephone ( ) _____

Signed _____

Name _____

Title _____

Company _____

Address _____

_____

Telephone ( ) ext.

ATTACHMENT A

Tabershaw-Cooper Associates, Inc.
2180 Milvia St., Suite 104
Berkeley, California 94704

Manufacturing Chemists Association VCM Epidemiological Study

Plant No.

1. When did this plant begin work involving vinyl chloride? _____

2. What is the approximate size of the current work force, exclusive of secretarial and office personnel? _____

3. Approximately how many of these are employed in jobs involving some exposure to vinyl chloride? _____

4. Please answer the appropriate one of the following questions about your retirement plan.

   a. No retirement plan _____

   b. Voluntary plan, begun in _____

   c. Required of all employees, begun in _____

   d. Now required, formerly voluntary.
      Voluntary plan begun in _____

      Required plan begun in _____

   Remarks: _____

   _____

   _____

5. Please answer the applicable questions below about retention of records. How long are personnel records kept

   a. for those who retired but were not covered by a retirement plan? _____

   b. for those who retired under a retirement plan and have died? _____

   c. for those who quit or were discharged? _____

   d. for those who died while employed? _____

ATTACHMENT B

6. Do the personnel records which are retained contain a record of job titles and dates of job title changes for the individual concerned? _____

7. Please list the job titles in the current work force which involve some exposure to vinyl chloride.

ATTACHMENT B (cont.)

VCM Epidemiological Study - Individual Work History

Form B - Workers known to have died (please attach copy of death certificate)

1. Date of birth _____

2. Date hired _____

3. Date of death _____

4. Place of death _____

Status at time of death:

5. Employed _____

6. Retired (give date) _____

7. Terminated (give date) _____

8. If death certificate is not available, please give name of worker so that death certificate can be obtained. No contact will be made with relatives or attending physician.

_____

Job history with estimated exposure

Job no.	Title		Check estimated exposure in ppm <50	50-199	200-500	>500
1	_____	from _____				
		to _____				
2	_____	from _____				
		to _____				
3	_____	from _____				
		to _____				
4	_____	from _____				
		to _____				
5	_____	from _____				
		to _____				
6	_____	from _____				
		to _____				
7	_____	from _____				
		to _____				

ATTACHMENT C

tabershaw/cooper associates inc.
2180 Milvia Street, Berkeley, California 94704. Telephone (415) 845-3355

Dear

We are a research firm concerned with the health of workers in industry in the United States. We have been asked to study certain occupations in order to determine the health of men who have at one time or another worked in these occupations.

To accomplish this we need to know if those men we are interested in were alive on December 31, 1972. For any of the men who died on or before that date, we need to know the place and date of death. With these facts, we can compare the health statistics of our men with the general population.

To assist us, can you provide information regarding the person whose name appears below? A stamped, addressed return envelope is enclosed for your reply.

Needless to say, any information you give us is strictly confidential.

Thanks for your cooperation and assistance with this project.

Sincerely,

W. Clark Cooper, M.D.

Name: Date of birth:

Was above named man alive as of 12/31/72? _____

If deceased prior to 12/31/72:
Date of death _____

Place of death (city and state)

_____

_____

ATTACHMENT D

— T. L. Carey / Valley Forge.

Air Products and Chemicals, Inc. PUT IT IN WRITING

Date 5 MAY 1975

To J. D. KRAMER Subject VCl ABATEMENT REPORT

From G. B. H. SPEED

( ) Take Action ( ) For Your Approval ( ) For Your Info ( ) Discuss With Me
( ) For Your Comments ( ) As Requested ( ) File ( ) Note & Return

The following is the status of the VCl Abatement projects:

1. REGULATED AREA

The fencing of the regulated area is complete except for three personnel gates. Shipment of these gates has slipped and are rescheduled for installation in June.

2. PERSONNEL MONITORING

Personnel in the Resin Plant were monitored on April 9, 14, and 22, 1975. Of the 39 operators monitored, nine had exposures exceeding 1 PPM as follows:

Castleberry	3.1 PPM
Timmons (Utility)	2.6 PPM
Henson (Utility)	4.5 PPM
Copeland	1.8 PPM
Draffen (Utility)	7.1 PPM
MacDonald	3.1 PPM
Coleman (Utility)	1.4 PPM
Hurst (Utility)	1.3 PPM
Overbey	2.2 PPM

All utility operators in the above list spent some time in reactors and were wearing fresh air masks.

The Compound Plant operators were monitored on 27 March 1975 and all results were less than 0.1 PPM. These operators are on a quarterly monitoring schedule.

3. SOLVENT CLEANING

A CE has been submitted for solvent storage, piping, etc. for Calvert City.

4. STRIPPER PROJECT

A CE has been submitted for the addition of three post strippers and automation.

5. FRSH AIR HAVEN

The control panel has been enclosed to use as a fresh air haven. This project is complete and will not be included in future reports.

RECEIVED
MAY 9 - 1975
T. L. CAREY

Air Products and Chemicals, INC.

INTEROFFICE MEMORANDUM

RECEIVED

Date 26 February 1975

Subject EPA Meeting - Durham, N. C.
25 February 1975

To T. L. Carey — R. E. JONES (Location, Organization, or Department)

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

cc: A. R. Adams, R. C. Burnham, A. J. Diglio, R. Fleming, R. E. Jones - Pensacola, J. D. Kramer - Calvert City, T. J. Medovich - S. Brunswick, E. A. Primeau - Pensacola, J. T. Sebastianelli, E. Steck - Calvert City, H. L. Watson

SUMMARY

1. The Standards Group has sent to Washington recommendations to declare vinyl chloride a hazardous air pollutant and to proceed with setting a standard under Section 112 of the Clean Air Act.

2. A meeting of NAPTAC is scheduled for 25-26 March in Washington to consider the proposed action.

3. The proposed standard will limit emissions at some sources on the basis of production rates and from others at a maximum concentration of 10 ppm. Control methods are similar to, but more strict than those discussed in my report of the 7 January visit. Surveillance methods are not fixed at this time.

4. Vinyl chloride in water will be limited to a maximum of 0.0043 lb/100 lb. PVC produced.

5. Proposed sampling methods include the headspace method for resins and the gas bag method for air.

6. Industry representatives presented their comments on the questions asked by EPA.

(320)

7. Several specific comments or pieces of information are to be prepared for use by the group, and a meeting will be held 21 March to prepare for the NAPTAC presentation.

REPORT

The special subcommittee of the EPA/SPI committee met with EPA personnel in Durham, N. C., on 25 February to discuss activities related to the proposed standards on vinyl chloride emissions. The agenda prepared by EPA and a list of participants are attached and the numbering of the agenda is followed in this report. Mr. Don Goodwin, head of the Engineering Standards and Emissions Division, chaired the meeting.

1. He opened the meeting by stating that the final "package" for NAPTAC was almost ready and that the impact and health effects sections would be complete in about two weeks. This will be presented to NAPTAC on 25-26 March in Washington. (Note the date change). The proposed regulation is expected to be published in late June, with a public hearing following within 30 days and final promulgation by the end of the year. He forbade discussion on the decision on Section 111 vs. 112, or on health effects, on the basis that this would not the proper group for those subjects. A meeting for this purpose could be arranged later, but Ruckelshaus should handle this.

2. Item no. 2 on the agenda was a report by Ms. Susan Wyatt on current monitoring nearplants. Each test site at B. F. Goodrich, Louisville; Conoco, Aberdeen; and Shell, Narco; has 15 samples taking 24-hour integrated samples by the carbon tube method. One site has a Bendix FID operating on a 2.5 minute cycle and reporting hourly averages, and all sites have a meteorology station. The 24-hour samples are taken twice a week, and they have T5 from each plant. The highest reading reported so far was 10 ppm., found between two storage tanks at Shell. Shell confirmed the high concentration but could not relate it to an unusual condition. The next highest reading was 1.42, also inside a plant. Most of the readings have been below 0.04 ppm. The 50 hourly averages had 4 above 1 ppm. This program is scheduled to continue until April, but may be extended.

   A similar program has just been started at 5 fabricators; Ford at Mt. Clements, Congoleum Nair, Reynolds at Richmond, Sylvania at Warren, Pa., and Charlotte Pipe and Foundry at Monroe, N. C. These each have 4 of the 24-hour samplers. No data are available.

   They are concerned that the recent reduction in production will affect the results of this study. We will get a copy of this test protocol.

3. The writer discussed the problems with using a material balance as the only method to control emissions, and presented the SPI proposal for a combination of process check points, ambient monitoring, emergency reports, and long term material balances, tailored for each process, as a preferred method for determining compliance. Comments by EPA personnel generally supported the opinion that material balance was not satisfactory alone. This and the other presentations generally followed the outline agreed on at the meetings in New York on 11 February and Washington 18 February.

4. Les Evans stated that his source of information had been from the Section 114 reports of last year, follow-up questionaires to about 15 companies, and visits to 12 plants (those which he considered well-controlled). He has talked to Calgon, Chemical Design, Vulcan, Houdry and IGCI.

He proposes best available control techniques in the following areas:

Fugative leaks

Seals - double mechanical or canned pumps.

Maintenance - purge with nitrogen or steam before opening any system. Institute a formal leak patrol reporting system.

Car loading - purge to control equipment, use magnetic level indicator.

Leaking Reliefs - Add rupture disks and tell-tales.

Sampling - use closed loop manifold.

He would not estimate at this time the permissible emissions in this category.

Reactor entry

With installation of a gasholder, equipment purge, either water or solvent reactor cleaning, and reformulation of recipes to reduce fouling losses from this source can be reduced to a maximum permissible rate of 0.1 lb/100 lb. of PVC.

Emergency Reactor Discharges

Additional instrumentation and control, including shortstop, plus vent connections to the gasholder and emergency power will eliminate this source, and no discharge will be permitted.

Recovery Vent

Use of solvent or carbon systems, incineration, or refrigeration

should reduce this stream to a proposed maximum of 10 ppm.

Blend Tanks and downstream

Proper stripping will be required, and plants will be allowed a loss of no more than 400 ppm. monomer in resin as measured by the difference from the stripper exit to shipped resin. Composite analyses of each grade will be used. Any monomer measured from or destroyed in exit gases could be credited.

Aqueous streams

Flow and concentration of each stream must be measured and controlled before going to an open system so that total exit levels are below 0.0043 lb./100 lb. of PVC.

He still believes that there are feasible methods of treating dilute air streams to recover monomer, but did admit that these were not demonstrated.

5. It was stated that most, but not all suspension, bulk, solution, and latex products can now be stripped to 400 ppm., dry solids basis. Dispersion resins cannot be reduced below 2000 ppm., on some types only, and with significant productivity loss. It was estimated that 2 - 2.5 years would be needed to equip all suspension plants, and 4 - 5 years for dispersion plants, to achieve these levels at full rates. Goodwin said he was not worried about productivity losses.

6. It was stated that industry did not believe that incineration, carbon absorption, or solvent adsorption would be suitable except on a very limited number of small concentrated streams.

7. It was stated that recycle of drier exhaust was not feasible because of corrosion, heater fouling, and energy costs, and that reduction of drier exhaust flow would not effect monomer emissions.

8. Mr. Bill Grimly presented the information in the attachment titled "VC Source Test Method Background". He favors the gas bag for air samples and is inclined toward the BFG Perkin Elmer headspace method for resin analyses. He was told that this latter method was not proven for slurries, copolymers, and emulsions. We asked that we not be restricted to one method, and were told that EPA would specify the standard or referee procedure.

9. Mr. Bill Hamilton presented the preliminary cost estimates for control systems given in attached Table I. These are for their "typical" 150MM lb/yr. plant using 5M gal. reactors. Net costs are based on a value of 10¢/lb. for monomer, and the figures reflect his estimate of a 5% overall increase in efficiency of the plant with controls. Objections were raised to most of his numbers.

10. Ms. Wyatt presented the attached chart of impact effects from the proposed control devices. The first four were given as small in actual impact, and she stated that solvent cleaning was undesirable because of all the associated problems. She did say these estimates are preliminary, and can be improved. Energy cost for prolonged stripping was given as 50% more than the present standard procedure at 170°F. Incineration was estimated to increase total energy consumption by 50% in a suspension plant and 400% in a dispersion plant, and still leave 10 ppm in the effluent. They admitted incineration probably was not practical, and that their data on carbon absorption are inadequate for proper evaluation. The solid waste impact is expected to be quite high.

11. It was stated that true fugative emissions amounted to no more than 10% of unaccountable monomer and that a good preventative maintenance program with intensive personnel retraining were more effective than any other single item in reducing emissions. Industry generally is well along on the EPA list in response to OSHA.

12. It was recommended that specific equipment or methods should not be required, but used as found suitable. Each of the listed types has some proper applications, but is not universally applicable.

13. It developed that this referred to pumps, filters, etc., so it was stated that, with time, arrangements can be made to return this to the recovery system.

14. Analytical methods have been exchanged with EPA.

15. Union Carbide admitted to three runaway dispersion batches in the last 6 months due to relaxed cleaning schedules, and BFG spoke of "several" lost batches at Louisville recently because of reformulation experiments aimed at reduced fouling. It was predicted that little further advance would be made in this area. Emergencies beyond the scope of realistic protective systems always will occur. Connections to common vents or a gasholder for relief valves were stated to be unsafer.

The two representatives of monomer producers met later with Evans and Goodwin. In summary they were told that fugative emissions would be placed on the same basis as at PVC plants (as yet unspecified) and that all process vents will be limited to 10 ppm.

The industry group met later and agreed that the dispersion area required immediate attention. Representatives of this business will meet 3 March to prepare more

information for EPA and try to arrange a meeting at Durham 10 March for discussion.

Meanwhile, the committee will: try to obtain more data on the Connecticut cases, if it can be made available; prepare comments on the proposed water standard; comment on the control costs; and meet on 21 March to prepare a statement to be made at NAPTAC.

J. T. Barr

JTB:fm
Attachments

TABLE I

NET COST OF CONTROL DEVICES

Source	Device	Cost $M	Net Annual Cost $M/Yr.	Net Annual Cost ¢/Lb.
Fugative	Several	226	204	0.14
Reactor	Gasholder[1]	824	(489)	(0.33)
Recovery Vent and Slurry	Carbon bed[2]	220	(29)	(0.02)
Drier, bulk area	Improved Stripping	368	94	0.06
Total		1,637	(220)	(0.15)
Without Monomer Credit			530	0.35

[1]93M ft.^{3}, 10,000 lb. capacity.

[2]Gas flow 60 acfm, enclosed blend tanks with small nitrogen purge.

JTB
2/26/75

AGENDA

EPA and SPI Meeting - The Control of
Vinyl Chloride Emissions from Polyvinyl Chloride Plants

February 25, 1975
Durham, North Carolina

1.	PVC Regulation Work Schedule	EPA
2.	PVC Ambient Monitoring Program	EPA
3.	Recognizing that source sampling to monitor total emissions from PVC plants is difficult to impossible - Discuss techniques of using plant material balances to determine emissions. (Sampling techniques, manpower requirements, accuracy, etc.) -	SPI
4.	Current Status of PVC regulation.	EPA
5.	Technical feasibility of reducing the vinyl chloride content of the PVC resin from the stripper to 400 ppm dry basis.	SPI
6.	Compliance schedules for installation of stripping systems at existing plants. Use of incineration, solvent absorpion, and/or carbon adsorption to reduce the vinyl chloride concentration of the dryer exhaust to less than 10 ppm. (Cost, capability.)	SPI
7.	Quantify the reduction in dryer exhaust gas volume that can be achieved by using fluidized bed dryers, flash dryers, and/or by recycling the dryer exhaust after removing the water and vinyl chloride. Discuss the factors that would control the rate at which these techniques can be installed.	SPI
8.	Source sampling and analytical procedures for PVC.	EPA
9.	Preliminary cost estimates of controlling PVC plant emissions.	EPA
10.	Environmental impact of PVC plant regulation.	EPA
11.	Status of installation of control equipment to restrict fugitive emissions in response to OSHA regulations.	SPI
12.	Use of canned pumps, or double mechanical seals on pump, agitator, and compressor seals to reduce fugitive emissions.	SPI

13. Feasibility of purging all vinyl chloride to the monomer recovery system or to a carbon adsorber; incinerator or flare before opening process equipment for maintenance and inspection. SPI

14. Sampling and analysis techniques to determine the vinyl chloride content of the resin after stripping. SPI

15. Use of alarm systems and shortstop to eliminate reactor pressure relief valve discharges or to eliminate such discharges by venting the reactor contents to a gasholder. SPI

Roster 2-25-75

Meeting on Vinyl Chloride

Don R. Goodwin	Director, ESED
George W. Walsh	EPA, ESED
G.W. Daigre	Dow Chemical, Plaquemine, La.
W.W. Madden	Firestone Plastics Co.
J.T. Barr	Air Products & Chemicals
Jos. E. Hadley	SPI General Counsel
R.W. Laundrie	General Tire
W.C. Holbrook	B.F. Goodrich Chem. Co.
D.A. Scholes	Hooker Chemicals & Plastics
R.N. Wheeler	Union Carbide Corp.
L.B. Evans	EPA, ESED
Chuck Kleeberg	EPA, ESED
Bill Hamilton	EPA, SASD
Bill Vatavuk	EPA, SASD
Susan Wyatt	EPA, ESED/SDB
Jack Farmer	EPA, ESED/SDB
Bill Grimley	EPA, ESED/EMB
Albert J. Beveridge	Ruckelshaus, Beveridge & Fairbanks
Gary H. Baise	Washington, D.C.
Jim Durham	EPA, ESED/ISB
Stan Cuffe	EPA, ESED/ISB

QUALIFIED SECONDARY ENVIRONMENTAL
IMPACTS OF INDIVIDUAL CONTROL SYSTEMS

CONTROL SYSTEMS	SECONDARY ENVIRONMENTAL IMPACTS					
	Air Impact	Water Impact	Solid Waste Impact	Noise Impact	Radiation Impact	Energy Impact
Fugitive Emission Controls						+
Gasholder/Water Purge System		+				+
Safety Valve Controls						
Reactor Solvent Cleaning	+	+				+
Improved Slurry Stripping		+				+
Solvent Absorption	+					+
Incineration (Without Scrubber)	+					+
Incineration (With Scrubber)	+	+				+
Carbon Adsorption		+	+			+

small ↑ ?

VC SOURCE TEST METHOD BACKGROUND

For stack sampling purposes, vinyl chloride (CH_2 = CHCl, molecular weight 62.50) must be assumed to exist in conjunction with other hydrocarbons, both chlorinated and otherwise. Accordingly, the accepted method for VC (vinyl chloride) analysis has consisted of first separating the VC from other hydrocarbons, followed by measuring the quantity of VC with a flame ionization detector. However, between various groups concerned with measuring VC, non-uniformity in procedures was found to exist in the following areas: (1) sample collection, (2) introduction of sample to gas chromatograph, (3) chromatographic column and associated operating parameters, and (4) chromatograph calibration.

Two of the approaches for VC sample collection involved an evacuated flask for grab samples and tubes containing activated charcoal for an integrated sample. Since it was established that a two-hour average concentration was desired, the integrated sample approach offered a greater advantage over the grab sample approach because emission fluctuations due to process variations would be automatically averaged. In addition, the integrated approach naturally minimizes the number of samples that need to be analyzed. Upon investigating the activated charcoal sampling tubes, it was found that they were basically designed for sampling ambient concentration levels of VC. Since source effluent concentrations are expected to be higher, there was the uncertainty involved with predicting sample breakthrough or when the sample should be terminated. In addition, the amount of charcoal that would be required for some sources would be excessive for a two-hour sampling period.

In view of these deficiencies, it was decided that collection of the sample in Tedlar bags might be a better approach. To check the flexibility of this approach a study of VC stability, or deterioration in Tedlar bags in the presence of various process-associated gases was undertaken.[1] The study showed no significant deterioration of VC over a period of 48 hours. Consequently the integrated bag technique was deemed suitable.

Introduction of a collected gas sample to a gas chromatograph can be done either through use of a gas-tight syringe or an automated sample loop. The latter approach was selected since it has less possibility of leakage and provides a more reproducible sample volume.

Several columns, used by the various groups, are mentioned in the literature as being suitable for the separation of VC from other gases; most notable among them have been Carbopak A[2] and Chromosorb 102.[3] Carbopak A is the more sensitive of the two; however, the VC elution time is not as great as with the Chromosorb 102. For this reason Chromosorb 102 was selected for further study. A program was undertaken to establish whether various hydrocarbons that were known to be associated with VC in stack emissions interferred with the VC peak from the Chromosorb column. The study revealed no such problems.[4] Furthermore, analysis of actual source samples of the peak reported as VC with this column by two qualitative techniques, spectroscopy and electron capture, indicated the peak was only VC.[5] It should be noted that selection of Chromosorb 102 does not mean that Carbopak A or some other column(s) may not work equally well.

Calibration has been accomplished by two techniques, the most common being the dilution of 99+% VC with nitrogen into a series of lower concentrations in Tedlar bags. The second technique utilizes a set of cylinder standards. Both techniques have been found to produce acceptable results and will be included in the reference method.

Based on the study of VC stability in Tedlar bags, possible interferences by various process associated gases, and calibration methods, and as a result of a field study and tests conducted at a source of vinyl chloride, Method 104 was prepared for determining compliance with new source performance standards. This method is essentially the same as used for the data gathering process for setting the standards. Leak tests for the flexible bags and calibration procedures to insure accuracy, precision, and reliability are also specified.

REFERENCES

1. "Evaluation of a Collection and Analytical Procedure for Vinyl Chloride in Air," by G.D. Clayton and Associates. December 13, 1974. EPA Contract No. 68-02-1408, Task Order No. 2. EPA Report No. 75-VCL-1.

2. "Vinyl Chloride Monitoring Near the B.F. Goodrich Chemical Company in Louisville, Kentucky." Region IV, U.S. Environmental Protection Agency, Surveillance and Analysis Division, Athens, Georgia. June 24, 1974.

3. "The Evaluation of Airborne Concentrations of Vinyl Chloride," Richard R. Keenan, G.D. Clayton & Associates, 1974.

4. Same as 1

5. EPA Report No. 75-VCL-2