DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
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October 3, 1977

Mr. Paul Bureau
Personnel Manager
Monsanto Plastics and Resins Co.
730 Worcester Street
Indian Orchard, Ma. 01151

Dear Mr. Bureau:

Enclosed please find a copy of the NIOSH preliminary report of the epidemiologic study done on decedents from the Indian Orchard Plant during the period 1950 - 1976. We ask that you review these results and send us your criticism as soon as possible. We will make every effort to consider your criticisms in preparing our final report.

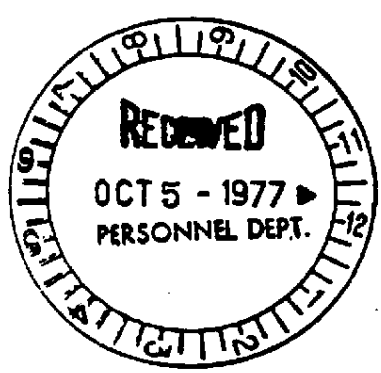
Sincerely yours,

Robert Spirtas

Robert Spirtas, Dr. P.H.
Chief, Illness Effects Section
Surveillance Branch
Division of Surveillance, Hazard Evaluations, and Field Studies

Enclosure

cc: Dr. Nessell
Mr. Schriver



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A PROPORTIONAL MORTALITY ANALYSIS OF A CHEMICAL PLANT IN MASSACHUSETTS

PREPARED BY

THE NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

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SUMMARY

A mortality analysis of 465 deaths among males formerly employed at a chemical-producing plant demonstrated significantly elevated proportions for cancer of the digestive organs and peritoneum and cancer of the genito-urinary tract. The deaths represented those employees who were eligible for death benefits during the period 1950-1976.

Mortality excesses remained elevated when specific digestive sites were examined individually. Prostatic cancer was significantly elevated in addition to the general genito-urinary category.

Occupational factors are suspected in association with the observed excess of digestive cancers, since the effects of foreign birth do not seem to account for all the excess observed. Moreover, when U.S. born workers are examined separately, digestive cancers remain elevated in proportion to total deaths.

BACKGROUND

In 1975, the National Institute for Occupational Safety and Health (NIOSH) was informed of the occurrence of 86 deaths among former workers of a chemical-producing plant. Analysis of the 86 deaths indicated an excess of cancer, particularly of the pancreas. Among the 86 deaths, 21 (24.4%) were attributed to cancer, four of which were due to cancer of the pancreas. In comparing the mortality experience of the plant with that observed for United States males in 1972, 15 deaths due to all cancers and less than one death due to cancer of the pancreas could have been expected among the 86 observed deaths. Upon reviewing this information, NIOSH initiated further study of the mortality experience among plant employees.

The plant, located in Massachusetts, has been operating since 1901. It currently employs about 1500 workers. The majority of active employees at the plant have been working over 10 years and therefore comprise a relatively stable working population. In 1901, the plant started the production of cellulose nitrate and cellulose acetate which ended in the late fifties and sixties. In 1938, the plant began the production of polyvinyl butyral and phenol-formaldehyde. A few years later, the plant began the production of formalin, formaldehydes, amino plastics, and polystyrene. Production of polystyrene was expanded in the mid-fifties and early sixties and included processes such as polymerization and compounding. Between 1948 and 1955, various polyvinyl chloride products were handled and manufactured. In 1968, styrene alcohol products were introduced, and in 1972, barrier resin production

began at the plant. In addition, various other chemicals have been manufactured at both the pilot and main plant.

Review of the recent literature for health effects related to the manufacture of these chemicals has revealed no established association with cancer excesses with the exception of vinyl chloride products.[1-18] Production of vinyl chloride and its polymers has been most frequently associated with the increased occurrence of angiosarcoma of the liver. However, increased risk for other cancers of the digestive system, cancers of the respiratory system, brain, and lymphomas has also been observed for workers exposed to these compounds.[5]

It should be recognized that a large number of chemicals are used in the manufacture of the major products made at the plant under study. Although some specific chemicals have already been associated with health effects, it is very difficult to pinpoint etiologic agents because workers frequently have exposures to numerous substances over their working lifetime.[16-18]

METHODS

The plant was asked to identify all deaths among former male employees for the interval 1950 to April, 1976. Women were excluded since such a small number of female deaths occurred. The plant was able to identify 484 deaths which occurred during the designated interval. Death certificates were acquired for 465 (96%) of the identified deaths. The majority of the deaths occurred either among active employees (who were currently working at the time of their deaths) or among retirees (who were eligible for pension or death benefits). Only deaths of employees for which plant records were maintained were identified. Deaths occurring among employees who terminated employment or who retired and were ineligible for death benefits were essentially not identified. Therefore, the 465 deaths studied are probably more representative of long-term employees than of those who left after a short period of employment.

Additional data was collected on deaths identified by the union and other sources. Since these deaths were not identified by any one method, we excluded them from the PMR analysis but utilized them to verify company-identified deaths. No unusual mortality patterns were observed among the additional deaths that would substantially change the results of the PMR analysis.

In addition, the plant classified each decedent as wage (hourly) or salary, according to the decedent's last held job at the plant. This information, along with demographic data such as race, age at death, place of birth, and

date of death were considered as independent variables in the analysis. In the statistical analysis, emphasis was placed on disease etiologies which may be associated with chemical manufacturing processes.[1-18]

Death certificates were coded by a trained nosologist for underlying cause of death, according to the rules of the Seventh Revision of the International Classification of Diseases, Adapted.[19] Mortality data from 1967 United States Vital Statistics were used for comparing the plant's mortality experience with that of U.S. males.[20] The data was analyzed by using the proportional mortality method (PMR), in which the number of observed deaths is compared to the number of expected deaths, based on a standard population.[21] The following formula was used to calculate the PMR for 21 selected causes of death. Adjustments were made for age by using age-specific values for the expected number of deaths in the total PMR calculation.

$$PMR_k = \frac{Observed_k}{Expected_k} = \frac{\sum_j t_{jk}}{\sum_j T_j \frac{s_{jk}}{S_j}}$$

Where t_{jk} = number of observed deaths from cause k in the jth age group of the test population.

T_j = number of observed deaths from all causes in the jth age group of the test population.

s_{jk} = number of deaths from cause k in the jth age group of the standard population.

S_j = number of deaths from all causes in the jth age group of the standard population.

The Chi-Square test for significance was applied to all PMR's which have five or more observed cases. Two tailed tests of significance were made on PMRs based on fewer than five cases, assuming the ratios had a Poisson distribution. All tests of significance were made at the .05 level.

RESULTS

The 465 deaths were grouped by race and classification of jobs held at the plant, resulting in the distribution of deaths as shown in Table 1. The majority of the deaths were among whites (441), 358 of which occurred among wage workers. Due to the relatively small number of black deaths (24) observed, the greatest emphasis in statistical analysis was placed on the mortality patterns of the white workers.

In grouping the total deaths by age, it was observed that the greatest number of deaths occurred in the 55-64 age group (Table 2). The frequency of deaths increased with age up to age 65 and then decreased with advancing age. Black wage workers, however, tended to die at earlier ages than their white counterparts. While both white wage and salary workers died most frequently at ages 55-64, most black wage workers died between ages 45-54.

The concentration of deaths at earlier than expected ages may indicate the effects of occupational hazards or may simply reflect biases in the reporting system. Active workers were more likely identified through the plant's death benefit program. If a retrospective cohort study had been conducted, relatively more deaths in the older age groups would have been expected.

Proportional mortality ratios were calculated for 21 causes of death for ages 20-75+, using the following age groupings: 20-34, 35-44, 45-54, 55-64, 65-74, and 75+ for 441 white and 24 black deaths separately (Tables 3 and 3.1).

Among whites, a significant excess (PMR = 1.49, χ^2 = 5.81, p <0.025) of deaths (36) were attributed to cancers of the digestive organs and peritoneum. Cancer of the genito-urinary tract was responsible for 16 deaths and resulted in an elevated PMR of 1.64 (χ^2 = 4.02, p <0.05). Each specific cancer site within digestive cancers displayed elevated PMRs, although they were not individually statistically significant values (Table 3.1). The largest proportion of the 36 digestive cancers were due to cancer of the large intestine and rectum. Cancer of the liver, however, while based on 4 cases, demonstrated the most elevated PMR of 2.77 (n.s.). Nine of the genito-urinary cancers were attributed to prostatic cancer. The calculated PMR of 1.94 (χ^2 = 4.06, p <0.05) is a significantly elevated value.

White workers did not demonstrate an overall increased risk for any of the non-malignant causes of death, although significant excesses in mortality occurred in individual age groups for both heart and digestive diseases. Heart disease occurred more than four times greater than expected in the 20-34 age group but less than expected in workers aged 65 or older. Digestive diseases were most elevated in workers aged 75 or older.

Age Trend

Because of the inherent bias of the study design, it is difficult to interpret relative differences in PMR's across age. Additional deaths may have occurred among persons aged 65 or older or among former workers who quit before retirement but remained unidentified due to pension ineligibility. On the other

hand, since deaths are being examined in a proportional manner, the deficiency may be due to the effects of competing causes of death. The unusual number of deaths in the 20-34 age group, however, does not seem to be explained by study design. The excess might have resulted simply by chance, since multiple comparisons have been made.

Individual age groups also displayed elevated PMRs for digestive cancers, although in most instances statistical significance was not attained, possibly due to small numbers. In general, however, the greatest relative excess occurred in the age group 35-44 and steadily decreased over age (Table 3.1).

Time Trend

Deaths from all causes among white males occurred with increasing frequency when grouped in five year intervals between 1950 and 1974 (Table 4). Total cancer deaths followed a similar pattern with the largest number of deaths occurring in the five year interval between 1970 and 1974. When black deaths are included in this distribution, the pattern of an increasing frequency of deaths over time is maintained.

The total 441 white deaths were divided into two approximately equivalent intervals so that any changes in mortality trends could be observed. In comparing the deaths which occurred between 1950-1964 and 1965-1976, several notable differences in mortality can be seen (Table 5). A greater proportion of deaths (268) fell into the latter interval and proportional mortality for

selected causes differed. While deaths due to all cancers increased by 2% with time, deaths due to heart disease and cerebrovascular lesions decreased. Cancers of the digestive organs and peritoneum increased from 6.9% to 8.9%. This increase was also reflected by the significantly elevated PMR of 1.62 ($\chi^2 = 5.67$, $p < 0.025$) for digestive cancers in the latter interval. Several specific digestive sites also demonstrated increased PMRs which however, were not significantely elevated, possibly due to small numbers. In addition, diseases of the respiratory system became more prevalent, while cancer of the respiratory system declined with time.

Socio-Economic Effects

The mortality experience of white workers was further examined by class of worker and by birth place, because they are recognized factors in disease etiology. Increased risks for particular cancers have been associated with socio-economic class and ethnicity.[22-27] Increased disease risks among wage employees also may suggest occupational exposure to hazardous substances.[28-30]

Proportional mortality ratios were calculated separately for white wage and salary workers. A significant excess (PMR = 1.52, $\chi^2 = 5.36$, $p < 0.025$) of digestive cancer was observed among wage employees. Thirty of the total 36 digestive cancers observed among all white workers occurred in the wage group. The distribution of these 30 cases by specific cause and age group resulted in excesses of deaths for several younger age categories. A significantly elevated proportion (PMR = 1.74, $\chi^2 = 4.45$, $p < 0.05$) of genito-urinary cancers

was seen among wage workers. The heart disease excess previously observed in the 20-34 age group in all white workers became more apparent when wage workers were examined separately.

Salary workers, on the other hand, demonstrated an elevated proportion of non-malignant digestive diseases in the 75 and older age group.

In order to control for the effects of birth place, PMRs were calculated separately for U.S. and foreign born. The foreign born were found to have a greater risk (PMR = 1.93, χ^2 = 5.37, p <0.024) for digestive cancers, while the U.S. born demonstrated increased risk (P = 1.81, χ^2 = 4.30, p <0.05) for genito-urinary cancer. Cancer of the large intestine and rectum was also proportionately elevated (P = 2.26, χ^2 = 4.19, p <0.05) among the foreign born. U.S. born, likewise, demonstrated proportional elevations in cancers of specific digestive sites, but none demonstrated statistical significance. In many instances, because of small numbers, tests for significance are not reliable.

Further examination of the 109 foreign born decedents was made by separating them into Eastern European and other foreign born, since previous studies have shown that Eastern Europeans tend to have increased risks for digestive cancers.[22-25] Approximately the same proportion of digestive cancers were found to occur in each group. Six cases occurred among 56 decedents of Eastern European birth. The same number of cases occurred among 53 decedents born in other foreign countries.

Although country of birth may explain part of the excess of digestive cancer in the study population, there still appears to be some other factor affecting the observed excess of digestive cancer deaths.

Racial Effects

Twenty-four of the 465 identified deaths were among black wage employees. Although no unusual mortality excesses were noted among blacks, it is interesting that several proportions of deaths from selected causes were essentially the same as in whites. While 5 (20.8%) of the black deaths were attributed to cancer, 92 (20.9%) deaths were attributed to cancer in whites. Comparable proportions were observed also for deaths due to cerebrovascular lesions. One of 24 (4.2%) black deaths were due to cerebrovascular lesions, and 18 (4.1%) occurred among white deaths. Heart disease, however, varied slightly between the two races. Thirty-three percent of black deaths were due to heart disease, whereas forty-seven percent of white deaths were attributed to heart disease. The PMRs calculated for black deaths for selected causes did not reveal any unusual mortality excesses.

DISCUSSION

The apparent excess of each of the digestive cancer sites seems to indicate some common relationship in etiologic agents. The excess of digestive cancers may suggest the ingestion of hazardous substances. In an least one previous study of a chemical factory, workers in dusty jobs experienced an excess of stomach and colo-rectal cancer.[31] Particles which are too large to be deposited

in the alveoli of the lungs are cleared by ciliary action and deposited in the mouth and nose. The particles may then be transported into the pharynx and passed through the rest of the digestive tract. Particles which are small enough to be deposited in the alveoli are partially cleared by another mechanism, the alveolar transport system.[32-33] The lack of cancer excesses of the respiratory organs may indicate an etiologic agent consisting of particles too large to be deposited in the alveoli.

The increased risk for genito-urinary cancers does not seem related to the excess of digestive cancers. Rather, it may suggest that another independent etiologic agent is at work.

The methods used here have several limitations which should be recognized in interpreting the results. The data collected in this study represents only a portion of the total deaths which actually occurred during the interval 1950-1976. Therefore, study design may have influenced mortality patterns observed in the plant population. As previously discussed, the distribution of deaths by age and year of death might have been affected. No differential effect, however, on cause-specific mortality should have resulted, since there seems to be no explainable bias toward particular causes of death.

Only the 1967 U.S. male population was used for comparison with the plant population, and only the Seventh revision of the International Classification of Disease was used for coding causes of death. These methods were selected,

although the study interval covers a 26 year period because of the small number of identified deaths. Moreover, use of one revision of the ICDA maximized consistency in death coding, since variations in coding occurred between the Seventh and Eighth revisions of the ICDA.

The paucity of deaths occurring in an analysis of one plant resulted in small numbers when sub-dividing the deaths into categories by age, race, class of worker, place of birth, and year of death. Frequently, excesses or deficits were observed but lacked statistical significance due to the small number of observed deaths in a given category.

Consideration of geographic variations in cancer rates has also been made. While cancer rates for most sites are higher in Massachusetts than for the United States, county rates should not be overlooked.[34] The county where the plant is located demonstrated higher rates of cancer than the state for many of the digestive organs, although they are not significantely different. The largest difference in rates was observed for cancer of the stomach as the state rate is 17.7 and the county rate is 18.8 per 100,000 white males. Although the mortality patterns of the plant may be reflecting those of the local area, the local area might in turn be reflecting trends influenced by the industrial population.

The proportional mortality method compares the proportion of deaths from specific causes to the total deaths observed. It cannot provide information such as the rate of mortality in a population nor measure the incidence or

prevalence of a disease. The PMR, however, does provide information describing the general pattern of cause-specific mortality in a population. Results of the PMR analysis should be used in planning follow-up studies for which more definitive methods are employed.

Information on work history and occupational exposures should be acquired for all the decedents, particularly for those whose deaths were attributed to digestive cancers. A case control method of study, utilizing deaths from all sources, would allow the comparison of disease frequencies between those exposed and not exposed to suspected occupational hazards.

Table 1. Total Observed Deaths
Classified by Race and Type of Job

	WAGE	SALARY	COLUMN TOTAL
WHITE	358	83	441
BLACK	24	0	24
ROW TOTAL	382	83	465

Table 2. Frequency of Deaths by Age, Race, And Type of Job

AGE	WAGE WHITE	WAGE BLACK	SALARY WHITE	TOTAL
20-34	9 (2.5)	3 (12.5)	2 (2.4)	14 (17.4)
35-44	27 (7.5)	3 (12.5)	9 (10.8)	39 (30.8)
45-54	62 (17.3)	10 (41.7)	22 (26.5)	94 (85.5)
55-64	127 (35.5)	5 (20.8)	24 (28.9)	156 (85.2)
65-74	92 (25.7)	2 (8.3)	21 (25.3)	115 (59.3)
75-99	41 (11.5)	1 (4.2)	5 (6.0)	47 (21.7)
Column Total	358	24	83	465

Table 3. PROPORTIONAL MORTALITY RATIOS AMONG WHITE MALES FOR SELECTED CAUSES OF DEATH

Cause Death	ICDA Code Seventh Revision	PMR	N=Number of Deaths
All Causes	001-999	1.00	441
Malignant Neoplasms	140-205	1.09	92
Buccal Cavity & Pharynx	140-148	0.36	1
Digestive Organs & Peritoneum	150-156A, 157-159	1.49 *	36
Respiratory System	160-164	0.71	20
Genito-urinary Tract	171-179, 180, 181	1.64 *	16
Prostate	177	1.94 *	9
Bladder & Other Urinary Organs	181	1.60	4
Neoplasms of Lymphatic & Hematopoietic Tissues	200-203, 204, 205	0.95	8
Diabetes	260	0.31	2
Cerebrovascular Lesions	330-334	1.02	30
Heart Disease	400-402, 410-443	1.07	206
Diseases of Respiratory System	VIII	1.20	28
Diseases of Digestive System	IX	0.91	21
Cirrhosis of Liver	581	0.56	7
Other+		0.82	62

+"Other" ICDA Codes are all those not mentioned above
* $p = 3.84$, $\alpha = 5\%$

Table 3.1. PROPORTIONAL MORTALITY RATIOS AMONG WHITE MALES BY AGE FOR MALIGNANT NEOPLASMS OF THE DIGESTIVE ORGANS AND PERITONEUM

CAUSE OF DEATH	ICDA CODE SEVENTH REVISION		AGE GROUPS 20-34	35-44	45-54	55-64	65-74	75+	All Ages
Malignant Neoplasms of Digestive Organs and Peritoneum	150-156A, 157-159	PMR	0.0	3.48	1.72	1.30	1.38	1.30	1.49*
		N	(0)	(4)	(7)	(12)	(10)	(3)	(36)
Esophagus	150	PMR	0.0	0.0	2.84	2.16	0.0	0.0	1.49
		N	(0)	(0)	(1)	(2)	(0)	(0)	(3)
Stomach	151	PMR	0.0	0.0	4.22	1.89	0.76	0.0	1.60
		N	(0)	(0)	(3)	(3)	(1)	(0)	(7)
Large Intestine & Rectum	153,154	PMR	0.0	0.0	0.62	1.62	1.61	2.81	1.50
		N	(0)	(0)	(1)	(6)	(5)	(3)	(15)
Liver	155,156	PMR	0.0	18.74	0.0	1.71	4.40	0.0	2.77
		N	(0)	(1)	(0)	(1)	(2)	(0)	(4)
Pancreas	157	PMR	0.0	7.52	2.15	0.0	1.39	0.0	1.20
		N	(0)	(2)	(2)	(0)	(2)	(0)	(6)

* $p = 3.84$, $\alpha = 5\%$

Table 4. Number of White Deaths
Per Five-Year Intervals by
Cause of Death
1950 - 1976

Cause of Death	Number of White Deaths						
	1950-54	1955-59	1960-64	1965-69	1970-74	1975-77	1950-1976
All Causes	39	55	79	98	124	46	441
All Cancers	7	15	12	21	25	12	92
Cancers of Digestive Organs and Peritoneum	3	5	4	8	11	5	36

Table 5. PROPORTIONAL FREQUENCIES OF WHITE DEATHS FOR THE INTERVALS: 1950 - 1964 AND 1965 - 1976

CAUSE OF DEATH	ICDA CODE SEVENTH REVISION	1950 - 1964 PMR	1950 - 1964 NO.	1950 - 1964 % of DEATHS IN INTERVAL	1965 - 1976 PMR	1965 - 1976 NO.	1965 - 1976 % of DEATHS IN INTERVAL
All Causes	001-999	1.00	173	100	1.00	268	100
Malignant Neoplasms	140-205	1.01	34	19.6	1.14	58	21.6
Buccal Cavity & Pharynx	140-148	0.0	0	0.0	0.60	1	0.3
Digestive Organs & Peritoneum	150-156A, 157-159	1.29	12	6.9	1.62*	24	8.9
Esophagus	150	0.0	0	0.0	2.48	3	1.1
Stomach	151	2.42	4	2.3	1.10	3	1.1
Large Intestine & Rectum	153, 154	1.05	4	2.3	1.77	11	4.1
Liver	155, 156	1.76	1	0.5	3.43	3	1.1
Pancreas	157	1.51	3	1.7	0.99	3	1.1
Respiratory System	160-164	0.94	11	6.3	0.54	9	3.3
Genito-urinary Tract	171-179, 180, 181	1.79	6	3.4	1.57	10	3.7
Prostate	177	2.20	3	1.7	1.83	6	2.2
Bladder & Other Urinary Organs	181	2.26	2	1.1	1.24	2	0.7

* p =3.84, α= 5%

Table 5. (Continued)

CAUSE OF DEATH	ICDA CODE SEVENTH REVISION	1950 - 1964 PMR	1950 - 1964 NO.	1950 - 1964 % of DEATHS IN INTERVAL	1965 - 1976 PMR	1965 - 1976 NO.	1965 - 1976 % of DEATHS IN INTERVAL
Neoplasms of Lymphatic & Hematopoietic Tissues	200-203, 204, 205	0.57	2	1.1	1.22	6	2.2
Diabetes	260	0.0	0	0.0	0.51	2	0.7
Cerebrovascular Lesions	330-334	1.26	12	6.9	0.91	18	6.7
Heart Disease	400-402, 410-443	1.13	83	47.9	1.04	123	45.8
Diseases of Respiratory System	VIII	0.82	7	4.0	1.42	21	7.8
Diseases of Digestive System	IX	0.92	9	5.2	0.89	12	4.4
Cirrhosis of Liver	581	0.36	2	1.1	0.72	5	1.8
Other+		0.92	28	16.1	0.86	34	12.6

+"Other" ICDA codes are all those not mentioned above

* p = 3.84, α = 5%

Table 6. PROPORTIONAL MORTALITY RATIOS FOR CANCERS OF THE DIGESTIVE ORGANS AND PERITONEUM BY COUNTRY OF BIRTH AMONG 441 WHITE DECEDENTS

Birthplace	Number	Digestive Cancers	
		No. Cases Observed	PMR
Total White Decedents	441	36	1.49*
U.S. Born	332	24	1.34
Foreign Born	109	12	1.93*

* $p = 3.84, \alpha = 5\%$

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ANNOUNCEMENT

TO: MONSANTO-SPRINGFIELD EMPLOYEES:

About a year ago, we informed you of the results of a mortality study on Springfield plant employees conducted by the University of Pittsburgh, Graduate School of Public Health. Those results showed no new evidence of worker health problems at the Springfield plant.

As a second phase in the study, the University of Pittsburgh was to examine the work histories of former employees who died of digestive system and genito-urinary tract cancers. While we had expected this second phase of the study to be completed in 1980, it is only now nearing completion.

However, very preliminary results have been reported to us by Dr. Marsh of the University of Pittsburgh who indicated that no correlation of work place and incidences of cancer has been determined. Final study results should be available early in the second quarter.

In addition, you should know that we recently became aware of the death of a former Monsanto-Springfield employee who had an angiosarcoma of the liver. As you know this form of cancer has been associated with exposure to vinyl chloride. Dr. Marsh has been advised of this case and will make a special effort to determine whether there is any additional evidence in the study data relating to this type of cancer. We are currently unaware of any such cases in the study.

Final results of the second phase of the mortality study will be communicated to you as soon as received.

J. L. Shriver

/SBN
3-20-81

ANNOUNCEMENT

On Wednesday, January 20, the "Valley Advocate" published what it stated was the second in a three-part series on the "Monsanto Company's Springfield Operation."

Predictably its authors Bush and Michak have cleverly woven together a rehash of past news, questions out of context, misrepresentations, innuendos and informational errors purporting to demonstrate Monsanto is conducting a conspiracy of silence to keep important information from its employees concerning worker health and cancer. It specifically attempted to link vinyl chloride exposure to alleged employee health problems ignoring a comprehensive study by the University of Pittsburgh which showed no such connection.

As you know, Monsanto has consistently and in a timely manner, advised its employees, local union officials, and appropriate government agencies of the results of any health studies it conducted or had conducted by outside impartial professional organizations. None of these studies has conclusively shown a causal relationship between cancer and a particular workplace exposure. For your reference, the attached prior bulletin board notices openly summarize previous studies, checkpoints and results which indicate the facts of the matter and refutes the alleged cover-up claim.

Our actions in handling employee health studies have been as follows:

1. Monsanto has always cooperated with governmental agencies requesting data on worker health evidenced by a joint Monsanto-NIOSH health study completed in 1976.

2. Monsanto has communicated frequently and truthfully on the status of its health study results to all employees and to other concerned parties (see attached notices).

3. The health studies either conducted by Monsanto or by the University of Pittsburgh School of Public Health have been designed with the best statistical approaches and historical information.

4. Monsanto has always been and will continue to be concerned with worker health and safety. Our continuing environmental and industrial health programs are evidence of this concern.

It's obviously impossible for me, in this communication, to answer every issue raised in the "Valley Advocate" article. However, if anyone has any additional concerns or questions, please feel free to contact me or Paul Bureau.

J. L. Shriver per RJB

J. L. Shriver, Plant Manager

1-22-82
Att.

1. INTERPRETATION, STATUS, PROCEDURES

Monsanto

FROM A. P. Torrenzano - Springfield cc R. L. Bourget

March 21, 1974

SUBJECT Interpretation of Health Standard Recommended By NOISH, 3-11-74(Relative to Springfield Plant)

REFERENCE

TO File

I. BASIS Angiosarcoma of liver

Detected in deceased	5 at BFG, Louisville
	1 at UC, South Charleston
	1 at Goodyear, Niagara
Detected in living	2 at BFG, Louisville

II. GENERAL APPROACH

A. Define "Regulated Area"

1. Where there is a potential for measurable concentration of VCM... VCM handling, polymerization, drying, packaging, whse.

2. Implicit -- any area where there is a measurable amount of VCM, such as locker rooms, smoking rooms, offices, etc.

B. In "Regulated Area"

1. Monitor VCM concentration frequently

2. If VCM concentration is detectable:

   a. wear full face air respirators

   b. decontaminate all exhaust ventillation air before emitting to environment (action on dryer air exhaust is vague)

   c. develop control plan to reduce VCM concentration and report results to OSHA

3. If VCM concentration is not detectable:

   a. Full face air respirator not required, however other safeguards still necessary -- as listed below.

RSV 0020163

   b. Coveralls and gloves

   c. No eating, drinking, smoking (or possession of tobacco) or chewing

3. d. Specific procedures and clothing for specific jobs such as kettle entry, VCM transfer, maintenance of equipment

e. Records of personnel

f. Posting of specific warning labels

g. Decontamination procedures for clothes, equipment, waste resin and people.

III. TIMING

A. Within 60 days report to OSHA

1. Brief description of in-plant location of "Regulated Areas", including number of employees normally in the area.

2. Control Plan

a. Initial VCM conc. survey

b. Program and timing to reduce VCM concentration

B. Every 6 months report to OSHA performance versus goals established in the Control Plan.

IV. EFFECT ON FUTURE PLANTS

A. Outside plant
B. Remote operation, closed kettle plant
C. Complete degassing of slurry before drying and packaging, and possibly in some cases, extraction of hydrocarbons before drying and packaging.
D. Complete accounting for all VCM with closed loops; approach 100% VCM yield.

V. EFFECT ON EXISTING PLANT

A. If dryer exhaust gases must be decontaminated--then shutdown of existing plant would be required until process is defined and equipment installed.

B. If building ventillation gases must be decontaminated, as specified--then shutdown of existing plant would be required until process and equipment installed.

V. EFFECT ON EXISTING PLANT (cont.)

C. All other practices specified could be implemented, pending purchase of monitoring equipment and protective equipment.

VI. CLARIFICATION REQUIRED

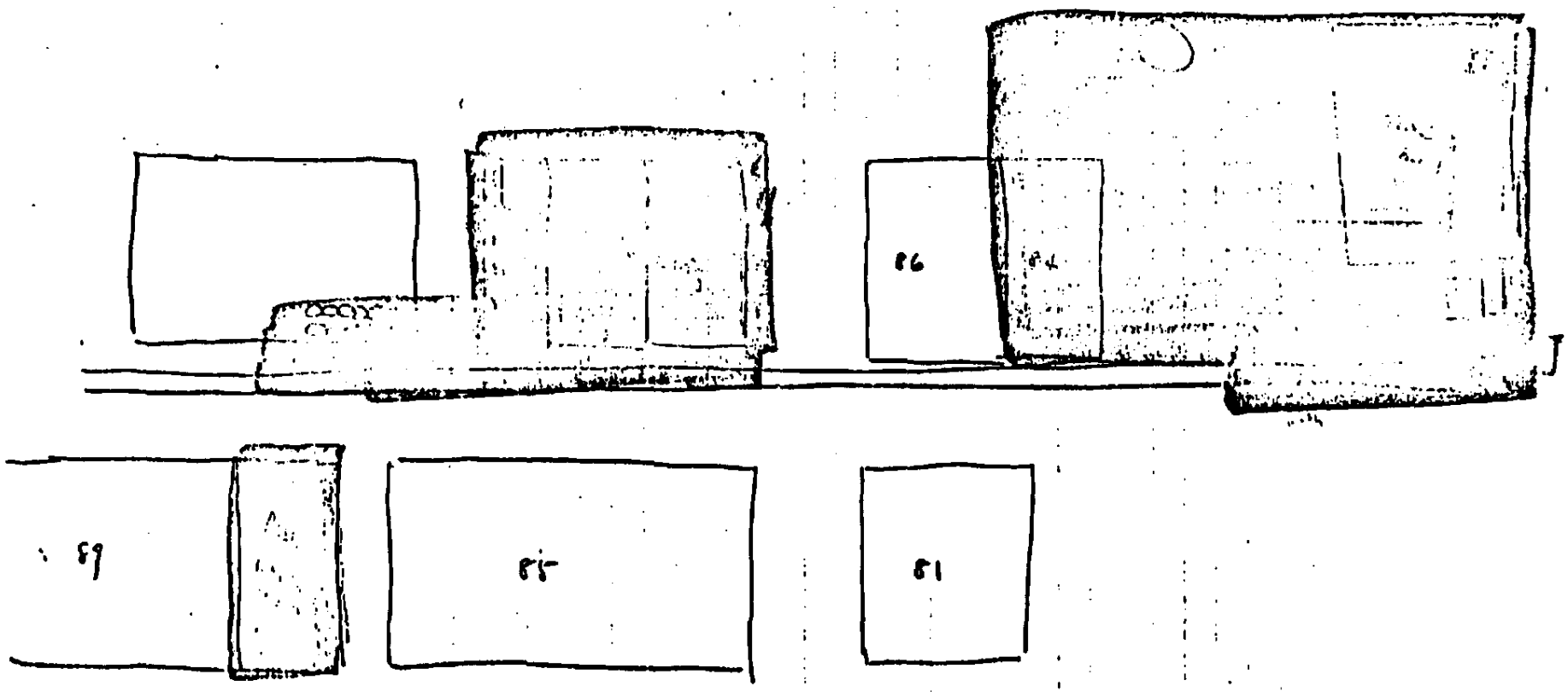
A. Dryer exhaust gases to be decontaminated.

B. Available process to meet decontamination of building ventillation air.

A. P. Torrenzano

/dh

REGULATED ARL



BASED ON Initial VCM IN AIR SURVEY plus DEFINITION OF Regulated Area

RSV 0020166

Approx Amount of Air To Be Decontaminated

				Est VOC #/hr
Bldg 88	Ventilation Dryer	25,000 - 125,000 CFM (100 - 500 hp)		1-50 500-600
Bldg 84	Ventilation Dryer	25,000 - 75,000 CFM (100 - 300 hp)		1-50 500-600
	TOTAL	120,000 - 300,000 CFM	@ 200 ppm	
		9,000 - 23,000 #/min	× 4 #/min	

× 4 #/min = 180,000 # VOC/hr valued @ $11,000

OR $\frac{180{,}000\ \#/hr}{70 \times 10^6\ \#\text{VOC/yr input}} \times 1\text{Yr} \times 100 = 3\%$ Loss via exhaust

Problem: (As it now exists) -
Decontaminate 120-300,000 CFM of air from ≈ 200 ppm of VOC
to < 1 ppm.

How: 1. Scrub with water?

2. Absorb & Recovery