The Vinyl Institute

NEWS

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For Immediate Release

PIONEERING WORK ON VINYL CHLORIDE SAFETY WINS INDUSTRY'S AWARD

WAYNE, New Jersey, June 21, 1988 -- The individuals whose work helped eliminate the health hazards once associated with the manufacture of vinyl chloride monomer have received this year's safety award from The Vinyl Institute, the trade association representing the leading manufacturers of PVC (polyvinyl chloride, or vinyl), VCM (vinyl chloride monomer) and PVC modifiers and additives. Dr. Maurice Johnson, retired from the BFGoodrich Company, and Dr. John Creech, of the University of Louisville, shared the award, which is given annually in recognition of outstanding contributions toward improved safety and health within the PVC industry.

Johnson and Creech established a link between the manufacture of vinyl chloride monomer and a hazard to human health in 1974, when they noted an unusually high rate of rare liver cancer -- angiosarcoma -- among workers in vinyl chloride monomer manufacturing plants. Until that time, exposure to vinyl chloride monomer had been studied in rodents, but the potential hazard to humans was unknown.

(more)

In presenting the award, Dr. Roy T. Gottesman, executive director of The Vinyl Institute, noted that many lives no doubt would have been lost without the work of both men, whose findings mobilized an entire industry into corrective action. Following the discovery of the link, new manufacturing practices were adopted throughout the PVC industry that virtually eliminated the health hazard related to the production of vinyl chloride monomer, the principal raw ingredient used to manufacture PVC, or vinyl, plastic.

The industry's reaction to the findings of Drs. Johnson and Creech often has been cited as a model of industry responsiveness and accountability in a time of crisis. Today, PVC is used in countless consumer and industrial products and is the world's second largest selling plastic material.

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HEPATIC DISEASE AMONG WORKERS AT A VINYL CHLORIDE POLYMERIZATION PLANT

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SUMMARY

Eleven cases of hepatic disease, including 7 cases of hepatic angiosarcoma, have been identified to date among men employed at one vinyl chloride polymerization plant. The earliest diagnosis was made in April, 1964. The two most recent cases, both angiosarcoma, were diagnosed in February, 1974 as a result of systematic medical screening for liver abnormalities among workers at the plant. Ages at diagnosis have ranged from 28 to 58 years and durations of employment prior to diagnosis from 5 to 29 years (average duration, 22.6 years). All 11 persons had worked in close and continuous contact with various phases of the vinyl chloride polymerization process. Review of pathologic material suggests the presence in both tumor and non-tumor cases of portal fibrosis and atypical sinusoidal lining cells. A direct etiologic relationship with exposure to vinyl chloride monomer is postulated.

Creech and Johnson (1) recently reported the occurrence of 3 cases of angiosarcoma of the liver among workers at a polyvinyl chloride (PVC) production plant in Louisville, Kentucky. Because this tumor is extraordinarily rare (only 15-25 cases are estimated to occur each year in the United States), the existence of such cases in this particular setting strongly suggests an etiologic relationship to some phase of the PVC production process. Recent animal studies being conducted in Italy (2) support(s) the idea that exposure to vinyl chloride monomer (VCM) may be the agent involved.

Beginning in late January 1974, intensive efforts have been devoted to clinical and epidemiologic studies of past and present workers at the Louisville plant, as well as at other PVC production plants elsewhere, in order to define more precisely the extent of health risks amony vinyl chloride workers. This report summarizes findings to date with respect to both malignant and non-malignant hepatic disease among workers at the Louisville plant, particular emphasis being given to epidemiologic features.

Background

The production of PVC by polymerization of VCM began in Germany about 40 years ago, with production in the United States starting about 5 years later. The industry grew rapidly after World War II, and growth has continued in recent years at a rate of about 14% per year. Currently in the United States 14 plants employing about 1,500 workers produce VCM, while 37 plants employing about 5,000 workers, polymerize PVC from VCM. Current annual production of PVC in the United States is estimated at approximately 4.3 billion pounds or 25% of world production.

The B. F. Goodrich PVC polymerization plant in Louisville first began operations in 1942. The number of persons employed at the plant directly in PVC polymerization has steadily increased, reaching a fairly stable level of 250-300 workers by the late 1950s. At present, in addition to 271 persons engaged directly in PVC polymerization, about 850, persons are employed at the plant in other capacities such as synthetic rubber production, compounding and milling operations, managerial and clerical positions, and maintenance work outside PVC polymerization areas.

Until 1966, VCM as well as PVC was produced at the Louisville plant. Since that time, however, all VCM utilized at the plant has been shipped by tank car from other facilities. The VCM is unloaded, stored, and then piped into large polymerization reactor vats through an essentially closed system. Each reactor receives a measured amount of VCM along with appropriate catalysts, stabilizers, emulsifiers, and additives (and other monomeric compounds if co- or ter-polymers are being produced), and the reaction is carried to the desired end point. Polymerized material is dropped into secondary tanks from which unreacted VCM is recovered and recycled through a closed system; it then enters tertiary tanks from which it is concentrated, dried, and packaged. The end product consists of 3 different materials: a) PVC resin (a powder with the texture of refined sugar), b) PVC paste (a very fine powder with the texture of processed flour), and c) PVC latex (a stable suspension of PVC in liquid).

For workers in PVC polymerization the point of greatest probable exposure to VCM occurs after polymerization when reactors are opened and cleaned. Although the air within reactors is replaced several times

before opening, a short burst of VCM may be released from reactors immediately upon opening. In addition, some PVC remains encrusted within reactors which, because of its porous structure, may retain significant amounts of entrapped VCM. In the process of chipping and cleaning this material from within reactors, retained VCM is released, Until the late 1960s, this cleaning process was done manually by a man lowered into the reactor for that purpose. Since that time, high pressure water hoses have been introduced for cleaning reactors, manual cleaning being done much less frequently. This change in work practice was designed primarily to prevent acroosteolysis, a disease peculiar to PVC workers and characterized by Reynaud's phenomenon, scleroderma-like changes of the hands, and lytic lesions in the distal phalanges (3, 4).

Clinical and Pathologic Findings

Table 1 summarizes salient features for each case diagnosed to date of hepatic angiosarcoma and of non-malignant liver disease. Review of death records for plant employees revealed a total of 5 cases of angiosarcoma of the liver diagnosed in PVC workers over the 10-year period 1964-1973 (cases 1 - 5). No cases were found diagnosed prior to 1964, and no cases were found of hepatic carcinoma or of angiosarcoma primary at other sites. A further review of medical histories of PVC workers currently employed at the plant identified 4 additional men with a known history of non-malignant liver disease documented by tissue biopsy (cases 8 - 11). Because of concern aroused by the discovery of hepatic angiosarcoma in workers, a medical screening program aimed at detecting hepatic abnormaliti was instituted at the Louisville plant. All current employees, whether

engaged in PVC polymerization work or not, were evaluated. As a result of this program 2 additional cases of angiosarcoma of the liver were diagnosed, both in PVC workers (cases 6 and 7). At the present time, no cases of angiosarcoma of the liver or of biopsy-proven non-alcoholic liver disease have yet been identified among non-PVC employees at the plant. All patients have been white males.

Cases of Hepatic Angiosarcoma (cases 1 - 7): Ages at diagnosis for the 7 tumor cases have ranged from 36 to 58 years (average age 46.7 years). Initial clinical features have varied widely, there being no signs or symptoms in case 7. Four patients (cases 1, 2, 5, and 6) presented with weakness and tiredness, 2 of whom also had intermittent pleuritic pain. These symptoms by themselves were not sufficiently severe to warrant medical evaluation until the appearance of acute abdominal pain, pronounced weight loss, or positive findings on serologic screening. Two patients (cases 3 and 4) were entirely asymptomatic until the abrupt onset of gastrointestinal bleeding. Two patients presented with clinically obvious hepatosplenomegaly (cases 2 and 5), but 4 patients (cases 3, 4, 6, and 7) had normal physical findings. While all 7 patients had evidence of liver function abnormality at time of initial workup, there was no consistent pattern, and in several instances abnormalities were only slight (cases 2, 3, 4, 6, and 7). In several cases, relatively mild hepatic dysfunction coexisted with either far-advanced portal hypertension (cases 3 and 4), unresectable angiosarcoma (case 6) or angiosarcoma with extensive portal fibrosis (case 7). In cases 1 and 2, large hepatic masses were present

at initial evaluation. Three patients (cases 1, 4, and 5) were not diagnosed until autopsy despite multiple liver biopsies.

Preliminary pathologic review suggests that in all 5 cases where non-malignant hepatic tissue is available (cases 2, 3, 4, 5, and 7) similar non-malignant hepatic lesions exist consisting of portal fibrosis, sinusoidal dilatation, and atypical sinusoidal lining cells. Conceivably such lesions may represent a precursor stage in the development of hepatic angiosarcoma.

Non-malignant Hepatic Disease (cases 8 - 11): Ages at diagnosis for these 4 cases range from 28 to 56 years (average age 46.5). Initial clinical presentations varied widely: 1 patient (case 8) presented with gastrointestinal bleeding; 1 (case 9) with chest pain and weight loss; and 2 (cases 10 and 11) with unrelated problems. On physical examination hepatosplenomegaly was present in 1 instance (case 9), splenomegaly alone in 2 (cases 8 and 10), with normal findings in case 11. Three patients underwent splenectomy either for marked splenomegaly (case 9) or as part of splenorenal shunt procedures (cases 8 and 10). Results of liver function tests varied widely and showed no consistent patterns in relation to clinical picture.

All 4 patients were found on liver biopsy to have some degree of hepatic fibrosis. Preliminary pathologic review of specimens available from all 4 patients suggests a close histologic similarity to the picture of portal fibrosis and sinusoidal changes described in the cases of angiosarcoma. Three patients (cases 8, 9, and 11), as well as case 7 in the

angiosarcoma group, were found at surgery to have a peculiar white, speckled appearance to the surface of the liver, which on pathologic section was seen to reflect diffuse subcapsular fibrosis.

Epidemiologic Findings

The seven men with angiosarcoma had been employed at the plant for 12 to 28 years (average 17.9 years), and the 4 with non-malignant disease between 5 and 28 years (average 20.6 years) (Table 2). Ten of the 11 patients (cases 2 - 11) worked exclusively or predominantly in one or more of the 4 PVC polymerization buildings at the plant (Table 2, Buildings A, B, C, and D). Patient 1 worked about half of his total employment time in a PVC polymerization building, (Building A) and for an almost equal time in a separate PVC drying and packaging building, (Building E). Three patients never worked elsewhere than in the PVC polymerization buildings (cases 4, 6, and 11). As can be seen from Table 2, non-polymerization buildings, (Buildings E through L and all others) were only sparcely represented in relation to total employment. For the 4 polymerization buildings which in 1973 employed a total of 160 persons, 184 man-years of employment are represented among the 11 cases. For the remainder of the plant with a total employment of about 950 persons in 1973, only 21 man-years were recorded.

Buildings A and B, opened in 1942 and 1947 respectively, are the older of the 4 polymerization buildings, with Building C (1947) and Building D (1948) being somewhat newer. Buildings C and D have more reactors (48 each) thaneither Building A (35 reactors) or Building B (25 reactors); the newer reactors have approximately a one-third greater capacity. Employment among

all 11 patients involved all 4 polymerization buildings. Three patients had worked in all 4 buildings; 5 had never worked in Building D, 3 had never worked in Building A or Building C, and although all 11 had worked at some time or another in Building B, 3 had worked there less than 1 month. It appears unlikely that some chemical or procedure unique to any one building can be implicated as a causative factor; more than twice as many man-years of employment, however, were represented among cases for Buildings B and D (121 years, 3 months) as for Buildings A and C (63 years, 3 months). While this difference may or may not be meaningful, some variations do exist between the various buildings which could conceivably be important as risk factors. Buildings A and C produce homopolymer resin exclusiveiy while various co- and ter-polymers are produced in Building B and all PVC paste and almost all PVC latex is produced in Building D.

Those workers who are probably most exposed to VCM are chemical helpers whose principal job is to clean reactors. As can be seen in Table 3, all 11 patients worked at some time as helpers. While virtually every employee at the plant has worked as a helper before being promoted to more advanced work, the average work duration (i.e., time from starting work at the plant to date of diagnosis) was 23.2 years for the 5 patients who spent 14 months or less as helpers and 15.0 years for the 6 patients who spent 20 months or more. This suggests indirectly a possible relationship between intensity of exposure and latent period for liver disease.

All 11 patients, or members of their immediate families, were individuall interviewed regarding past hepatic disease and possible exposure to hepatotoxic agents. None of the patients had a prior history of hepatitis or of

exposure to hepatitis, and none had taken hepatotoxic drugs. Three patients (Cases 1, 7, & 9) may have had significant alcohol intake. No patient, except for Case 7, recalled exposure to possible hepatotoxic chemicals outside the work environment, in particular to either arsenic or thorium dioxide, 2 chemicals previously implicated as causes of hepatic disease and angiosarcoma in humans (5-8). Patient 7 gave a history of exposure to arsenical insecticides on the family farm between the ages of 6 and 15; he both mixed and sprayed the insecticides 2-3 times a year for about 3 hours on each occasion.

Discussion

Prior to the report by Creech and Johnson (1), the only evidence that VCM might be oncogenic came from animal experiments. In 1971, Viola, et al., published data suggesting VCM oncogenicity in rats at very high doses, tumors of many tissue sites, lung, bone, and skin being recorded (9). Preliminary results of a more recent animal study in Italy by Maltoni and coworkers (2) appears to indicate that angiosarcoma of liver as well as of other tissues can be induced in rats by atmospheric levels of VCM not uncommon in the human workplace environment. In light of these observations, it appears likely that exposure to VCM is responsible for the Louisville cases. Further support for this hypothesis is needed, of course, from additional epidemiologic data concerning workers at other PVC and VCM plants. Further toxicologic analyses are also needed to address the possibility that the active oncogenic material may be some metabolite of VCM rather than VCM itself.

Whatever the precise mechanism for oncogenicity and hepatotoxicity of VCM, the Louisville data suggest that relatively high levels of VCM exposure and relatively long latencies (20 years or so) are involved. This does not rule out the possibility of less marked health effects at lower doses or shorter latent periods, but it does for the present focus attention on the immediate problem of assessing the health status of persons exposed in the remote past to high doses. None of the Louisville cases involved men working at the plant less than 6 years prior to diagnosis and it can be

safely assumed that levels of VCM exposure during earlier years of PVC production were considerably higher than at present due to less stringent work practice procedures and less attention to minimizing possibilities of VCM exposure in places of work.

In humans, both thorium dioxide and arsenic have previously been reported as causes both of hepatic disease and of angiosarcoma of the liver. In only one case at the Louisville plant (Case 7) was there any history of exposure to either of these 2 materials (arsenic in insecticide spray). Likewise there is little evidence among the Louisville cases that excessive alcohol intake plays any accelerating or potentially cocarcinogenic role.

Various data now suggest that VCM (or some derived metabolite) may produce in addition to frank angiosarcoma of the liver a nonmalignant hepatic disorder characterized by portal fibrosis and portal hypertension. This is suggested by the fact that such fibrosis was present in at least 5 of the Louisville tumor cases and was in addition observed in 4 other Louisville vinyl chloride workers without tumor. Recent observations in Germany (10), together with earlier reports from Eastern Europe (11) suggest that hepatic fibrosis and portal hypertension represent a not uncommon occupational disease of vinyl chloride workers. Conceivably such fibrotic liver disease represents a premalignant state. If this proves to be so, the early detection of such liver disease may be of greater industrial and public health importance than detection of tumor itself, both because hepatic fibrosis may be a more frequent condition in vinyl chloride workers and because it is possible that the fibrotic condition, unlike the tumor, may be reversible (or non-progressive following removal from the high-risk areas).

Because of these findings, there is an urgent need to develop effective means for detecting these particular kinds of liver abnormality in vinyl chloride workers. Unfortunately, the present series of cases suggest that currently available liver function tests are not sensitive predictors; in most if not all cases of angiosarcoma hepatic disease appears to have run a quite asymptomatic course, with liver abnormalities developing only as a late manifestation. Ideally what is needed is some testing procedure which can identify early hepatic fibrosis in the absence of hepatocellular injury and which at the same time can be efficiently applied to testing large numbers of workers without great expense or delay.

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TABLE 1
Clinical and pathologic findings of cases of liver disease

Case number	Age at diagnosis, race, sex	Month and year of diagnosis	Month and year of death	Initial symptoms	Physical findings
Cases with hepatic tumors					
1	52 WM	4/64	4/64	8/63-fatigue 1/64-RUQ pain	1/64-RUQ tenderness
2	43 WM	8/67	1/68	9/66-fatigue, "pleurisy" 8/67-epigastric "knot" and pain	8/67-epigastric mass and splenomegaly
3	36 WM	5/70	9/71	1/70 and 5/70-melena	1/70-negative 5/70-hepato-splenomegaly
4	49 WM	3/73	3/73	12/63 and 5/65-melena and hematemesis	12/63-negative 5/65-splenomegaly
5	58 WM	12/73	12/73	7/73-weakness and weight loss	7/73-hepato-splenomegaly
6	45 WM	2/74	-	8/73-fatigue, "pleurisy"	2/74-negative
7	43 WM	2/74	-	Asymptomatic	2/74-negative
Cases with non-malignant hepatic disease					
8	46 WM	12/68	-	10/68 and 11/68-melena	11/68-splenomegaly
9	28 WM	1/72	-	9/71-chest pain, weight loss	9/71-hepato-splenomegaly
10	56 WM	9/73	-	2/73-hospitalized for hernia repair, icterus noted	8/73-splenomegaly
11	56 WM	9/73	-	9/73-hospitalized for cholelithiasis	9/73-negative

TABLE 1 (continued)

	Hepatic workup*	Pathologic findings**
(Case 1)	1/64-moderate elev. TB, SGOT	1/64-OLB-slight focal hepatitis 3/64-NLB-unchanged 4/64-PM-hepatic angiosarcoma with metastases
(Case 2)	8/67-mild elev. AP, SGOT, LDH platelets 33,000 liver scan: large defect, splenomegaly	8/67-OLB-angiosarcoma 1/68-PM-hepatic angiosarcoma with spread to diaphragm and abdominal wall
(Case 3)	5/70-mild elev. TB, AP, SGOT, LDH liver scan: large defect esophogram: varices	5/70-OLB-angiosarcoma No PM
(Case 4)	5/65-elev. SGOT esophagoscopy: varices	5/70-OLB-toxic hepatitis 10/70-NLB-heaptitis, cirrhosis 10/70-OLB-slight hepatitis 3/73-PM-hepatic angiosarcoma
(Case 5)	7/73-marked elev. AP mild elev TB, SGOT liver scan: diffuse disease, hepato-splenomegaly	7/73-NLB-fibrosis OLB-periportal inflammation and fibrosis 12/73-PM-hepatic angiosarcoma with spread to duodenum
(Case 6)	2/74-mild elev. LDH liver scan: possible defect	2/74-OLB-angiosarcoma
(Case 7)	11/73-mild elev. TB, AP liver scan and angiogram: 4 cm. defect	2/74-OLB-angiosarcoma and extensive portal fibrosis, subcapsular fibrosis
(Case 8)	10/68-marked elev. AP,BSP; mild elev. SGOT; esophagram: varices	11/68-OLB-portal fibrosis, subcapsular fibrosis
(Case 9)	9/71-mild elev. TB, SGOT 10/71-liver scan: splenomegaly	12/71-OLB-portal fibrosis, subcapsular fibrosis
(Case 10)	3/73-moderate elevated TB & AP	9/73-OLB-slight portal fibrosis
(Case 11)	9/73-moderate elev. SGOT; mild elev. TB,LDH	9/73-OLB-chronic hepatitis with focal fibrosis

Footnotes for Table 1:

*TB = total bilirubin
SGOT = serum glutamic oxalacetic transaminase
AP = alkaline phosphatase
LDH = lactic dehydrogenase
BSP = bromosulfophthalein

**OLB = open liver biopsy
NLB = needle liver biopsy
PM = post mortem examination

Table 2
Duration of employment for patients with angiosarcoma of the liver and non-malignant hepatic disease among workers at the B.F. Goodrich PVC-polymerization plant in Louisville, Kentucky, by building of employment

Case number	Total duration of employment	PVC-polymerization buildings				Other buildings*								all others
		A	B	C	D	E	F	G	H	I	J	K	L	
1	19 yr/8 mo	8/11	0/3	0	0	8/5	2/1	0	0	0	0	0	0	0
2	17 yr/11 mo	0/1	12/5	2/0	0	0/6	0	2/9	0/2	0	0	0	0	0
3	13 yr/1 mo	1/2	1/7	5/5	4/4	0	0/3	0	0	0/2	0/2	0	0	0
4	16 yr/5 mo	1/2	14/9	0/6	0	0	0	0	0	0	0	0	0	0
5	28 yr/0 mo	0/6	2/8	0	24/5	0/5	0	0	0	0	0	0	0	0
6	11 yr/10 mo	0	0/1	2/3	9/6	0	0	0	0	0	0	0	0	0
7	18 yr/0 mo	0	0	11/7	5/5	0	0/10	0	0	0	0/2	0	0	0
8	24 yr/5 mo	4/1	4/1	0	14/0	1/4	0	0	0	0	0	0/11	0	0
9	5 yr/6 mo	1 wk	1 wk	3/4	2/0	0	0	0	0	0/1	0	0	0	0
10	23 yr/8 mo	0	19/7	1/4	0	1/6	0	0	0	1/2	0	0	0/1	0
11	28 yr/11 mo	21/8	7/1	0/2	0	0	0	0	0	0	0	0	0	0
Total	207 yr/5 mo	36/8	61/7	26/7	59/8	12/2	3/2	2/9	0/2	1/5	0/4	0/11	0/1	0
No. yrs in operation	32	32	30	27	26	27	27	29	19	32	26	32	26	(32)
Total no. of employees in 1973		36	36	48	40	14	1	12	0	52	72	18	8	about 600

*E - PVC drying and packaging
F - formerly monomer synthesis - now alcohol synthesis
G - PVC chlorination
H - formerly monomer synthesis - no longer in operation
I - compounding and milling
J - synthetic rubber
K - formerly warehouse and receiving - now compounding
L - PVC drying

Table 3

Work histories for patients with angiosarcoma of the liver and non-malignant hepatic disease among workers at the B.F. Goodrich PVC-polymerization plant in Louisville, Kentucky

Case Number	Date of first liver disease diagnosis	Total duration of work prior to diagnosis (years/months)	Total duration of work as chemical helper in PVC-polymerization buildings (months)
1	Apr 1964	19/8	6
2	Aug 1967	17/11	2
3	May 1970	13/1	51
4	Mar 1973	16/5	20
5	Dec 1973	28/0	8
6	Feb 1974	11/10	43
7	Feb 1974	18/0	32
8	Dec 1968	24/5	47
9	Jan 1972	5/6	58
10	Sept 1973	23/8	14
11	Sept 1973	28/11	9