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Bernadino v. JC
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October 10, 1990

John Downey, Esq.
Union Carbide Corporation
Law Department E3-285
39 Old Ridgebury Road
Danbury, CT 06817-0001

Re: Bernadino v. Union Carbide Corporation

Dear John:

Enclosed please find a copy of the preliminary expert report dated September 25, 1990 by Dr. Samuel Epstein on behalf of the plaintiff in this matter.

Sincerely,



ROBERT L. HOLLINGSHEAD

RLH/da

Enclosure

cc: Mr. Nick Wheeler, Jr.
Mr. R.C. Wise
Raymond A. Deckert,
Kemper National Insurance
Companies
(with enclosure)

PRIVILEGED AND
"CONFIDENTIAL MATERIAL
SUBJECT TO PROTECTIVE
ORDER"

UCC 075137

OCT 12 1990

J. R. DOWNEY

BERNARDINO v. UNION CARBIDE

Samuel S. Epstein, M. D.
professor of Occupational and Environmental Medicine
University of Illinois Medical Center Chicago

UCC 075138

Based on a review of the available documentation and records of Mr. Bernardino and the relevant scientific literature, I conclude that his occupational exposures at Amboy Terminals Co. (ATC) were causally related to the renal cancer from which he died in 1988 age 62. This conclusion is based on the following considerations:

1. Mr. Bernardino worked at ATC from 1960-1986: as Office Manager from 1960-1971; as Comptroller from 1971-1981; and as Terminal Manager from 1981-1986. During this 26 year period, he was exposed to a wide range of toxic and carcinogenic chemicals, gases, dusts and fumes, from the packaging and warehousing of polyvinyl chloride (PVC), polystyrene, and bisphenol and polyethylene resins.

2. Mr. Bernardino was continually exposed to these toxic and carcinogenic contaminants in his office, located directly within the ATC plant. He was also exposed to higher levels of these contaminants for some 2-3 hours daily during which he inspected operations throughout the plant. Finally, Mr. Bernardino was exposed, more directly, to still higher contaminant levels when he physically handled resin products during strikes at the plant, notably during the October/November 1976 strike when he lived and worked at the plant for some six weeks.

3. Based on available monitoring data, it has been concluded that from 1960-1976, Mr. Bernardino was exposed to VC levels in excess of 1 ppm, and to average levels in the 5 ppm range in Warehouse A.

4. Additional evidence on Mr. Bernardino's exposure is based on: descriptions of the very dusty conditions inside the plant; the contamination of his work clothing with dust; and the findings of dust on cars parked some two blocks away from the plant.

5. Mr. Bernardino was also exposed to respirable PVC dust containing high levels of residual and adsorbed VC, and also to PVC dust on his clothing with ensuing further secondary exposure at his home. In the absence of in-plant monitoring data, PVC dust levels were probably within the 1-19 mg/m³ range (Wagoner, 1983).

6. VC is a highly potent carcinogen inducing cancer in rodents at 1 ppm, the lowest concentration tested (Maltoni, 1977), and after only one hour exposures (Hehir et al, 1981).

7. VC is also a highly potent human carcinogen as reflected by case reports on angiosarcomas of the liver, beside other malignancies, in residents of communities up to two miles away from VC/PVC plants (Wagoner, 1983).

8. VC induces malignancies in a wide range of organ sites in rodents and humans, including the kidney (Appendix 1); VC is also nephrotoxic in rodents and humans.

9. Other occupational carcinogens which could have potentiated the renal carcinogenicity of VC in Mr. Bernardino include asbestos (Selikoff et al, 1979) and welding fumes (NIOSH, 1988).

10. While a statistical association between tobacco smoking and kidney cancer is recognized, this is relatively weak compared with the tobacco lung cancer

relationship (Appendix 2); the association is still weaker for parenchymal rather than pelvic renal cancers. Furthermore, these associations could well have been confounded by unrecognized occupational variables (Davis et al, 1981). Nevertheless, a contributory role of tobacco in the causation of Mr. Bernardino's renal cancer cannot be excluded.

11. Although a possible contributory role of chronic lithiasis in the causation of Mr. Bernardino's renal cancer cannot be excluded, with the exception of a case-control study based on only 24 cancers (McLaughlin et al, 1984), this is not generally recognized as a risk factor.

12. Mr. Bernardino's malignancy is consistent with those in other accountants employed in PVC plants. A case of angiosarcoma of the liver was reported in a "47 year-old man — employed for the ten preceding years as an accountant in a factory which produces vinyl sheets and processes PVC resins; it is reported that he frequently visited the production area of the plant" (Christine, 1974). In a second case, angiosarcoma of the penis and angiomas of the liver were reported in a 61 year-old man "who had worked in a PVC factory as an accountant for ten years" (Ghandur-Mnaymneh & Gonzalez, 1981).

13. Mr. Bernardino's sustained exposure to VC/PVC was exacerbated by the failure of Union Carbide to reduce residual VC levels in PVC resins until 1975, by failure to supervise and remediate the primitive work practices at ATC, including the absence of effective area and local exhaust ventilation and personal protective equipment, failure to act on complaints regarding work practices, and failure to train, instruct, warn and label. Such a pattern of failure was further exacerbated by Union Carbide's unique expertise and extensive prior state of the art information on the toxic and carcinogenic hazards of VC/PVC.

References

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Davis, D. et al, Estimating Cancer Causes: Problems in Methodology Production and Trends. In, Quantification of Occupational Cancer, eds. Peto & Schneiderman, pp. 304-306, 1981.

Ghandur-Mnaymneh, L. & Gonzalez, M.S., Cancer 47:1318-1324, 1981.

Hehir, R.M. et al, Env. Hlth. Perspec. 41:63-72, 1981.

Maltoni, C., Vinyl Chloride Carcinogenicity: An Experimental Model For Carcinogenesis Studies, In, Origins of Human Cancer, Cold Spring Harbor Symposium, eds. Hiatt et al, pp. 119-146, 1977.

McLaughlin, J.K. et al, J. Nat. Cancer Inst. 72:275-284, 1984.

NIOSH, Criteria for a Recommended Standard: Welding, Brazing and Thermal Cutting, 1988.

Selikoff, I.J. et al, Ann. N. Y. Acad. Sci. 330:91-116, 1979.

Wagoner, J.K., Env. Hlth Perspec. 52:61-66, 1983.

NEPHROTOXIC AND CARCINOGENIC EFFECTS OF VC/PVC

Torkelson et al, 1961 (Am. Indust. Hyg. Assoc. J. 22:354-361)

Inhalation exposure of rats to 500 ppm VC over 4.5 months induced "-- interstitial and tubular changes in the kidneys."

Viola, 1970 (Med. Lav. 61:174-180)

Intermittent inhalation exposure of rats to 30,000 ppm VC over 12 months induced tubular nephrosis and chronic interstitial nephritis.

Kramer & Mutchler (Dow Chemical), 1972 (Am. Ind. Hyg. Assoc. J., 19-30, January, 1972)

A statistically significant excess of "kidney stone and bloody urine" was reported in a group of 66 "healthy male workers exposed routinely to VC for periods up to 25 years", compared to 605 non-exposed controls (9.2% vs. 3.0%, $p < 0.05$).

Industrial Biotech Laboratories, 1974 (Report to MCA, 8/16/74; see, Congressional Research Service: Vinyl Chloride; An Occupational Health Hazard, 1/17/55)

Inhalation exposure of mice and rats to VC induced a dose-related incidence of kidney tumors in mice over a concentration range of 50-2500 ppm.

Maltoni & Lefemine, 1975 (Ann. N.Y. Acad. Sci. 246:195-218)

Inhalation exposure of rats to VC over a concentration range from 50-2,500 ppm for 12 months (BT1) induced a dose-related incidence of 2-10% nephroblastomas versus 0% incidence in controls. In another test (BT2), inhalation exposure of rats to 100 ppm VC induced an approximate incidence of 10% renal tumors, in contrast to less than 1% of hepatic angiosarcomas. The authors emphasized the rarity of these kidney tumors which -- "have never been described as spontaneously occurring in rats. -- As far as we know, this is the first time that nephroblastomas have been experimentally reproduced."

Prodan et al, 1975 (Ann. N. Y. Acad. Sci. 246:159-163)

Intermittent inhalation exposure of guinea pigs to VC over 3 months induced "histopathological and histochemical lesions" in renal glomeruli and tubules.

Manufacturers Chemists Association, 1976 (Supplementary Epidemiological Study of Vinyl Chloride Workers II: Final Report, September 1976)

Reported excess SMRs of 179 for urinary organ cancers among 298 PVC workers and 183 ($p < 0.01$) for "major cardiovascular and renal diseases" among 26 VC/PVC/Copolymer workers with over 25 years exposure. "An unexpected finding was the relatively high ratio of deaths attributed to cardiovascular and renal diseases", especially in the mixed exposure groups.

Organization Resources Counselors Inc., 1976 (Report on a Mortality Study Covering Employees of PVC Fabricators, February 1976)

A statistically significant excess PMR of 245 (O/E = 11/4.5) for urinary organ cancers was reported among 594 deaths from all causes for women employed in 17 PVC fabricating plants from 1964-1973. "-- there seems to be a great excess in urinary cancer."

Maltoni, 1977 (In, Hiatt et al. eds., Origins of Human Cancer, Cold Spring Harbor, New York, pp. 119-146, 1977)

Gavage of rats four times weekly for one year with 16.6 and 50 mg/kg VC solutions induced an approximate 10% incidence of renal tumors.

NIOSH, 1978 (Current Intelligence Bulletin 28, September 21, 1978)

Vinyl chloride induces kidney tumors in both mice and rats.

Chiazze et al, 1981 (Env. Hlth Perspec. 41:137-143)

Based on a cross sectional mortality study of 3847 deaths among employees of 17 PVC fabricating plants from 1964-1973, PMRs of 1.25 and 3.42 ($p < 0.05$) were noted for kidney cancer in males and females, respectively.

ILLUSTRATIVE STATEMENTS IN THE LITERATURE ON THE ASSOCIATION
BETWEEN SMOKING AND KIDNEY CANCER

Hammond (ACS), 1966 (Nat. Cancer Inst. Monog. 19:127-204)

In a cohort mortality study, based on 104 male kidney cancers, mortality ratios of 1.42 and 1.57 were reported for cigarette smokers age 45-64 and 65-79, respectively; the corresponding ratios for lung cancer were 7.8 and 11.6. Among 12 cancers and diseases with higher mortality rates in cigarette smokers than in non-smokers which were listed in the report's Summary, there was no reference to kidney cancer.

Kahn, 1966 (Nat. Cancer Inst. Monog. 19:1-125)

In a cohort mortality study based on an 8.5 year follow-up of 290,000 veterans and 81 male kidney cancers, an overall kidney cancer mortality ratio of 1.54 was reported for current cigarette smokers; the corresponding ratio for lung cancer was 10.9.

Bennington & Lauscher, 1968 (Cancer 21:1069-1071)

In a case control study based on 100 renal adenocarcinomas, excess risks of 5.1 and 5.4 were reported for smokers of over 10 cigarettes daily and for "all tobacco use", respectively.

Schmauz & Cole, 1974 (J. Nat. Cancer Inst. 52:1431-1434)

Based on 18 cancers of the renal pelvis and ureter, relative risks of 1.1-1.3 and 10.0 were reported for males smoking < 0.5 - 2.5 and > 2.5 packs/day, respectively.

Wynder et al, 1974 (J. Nat. Cancer Inst. 53:1619-1634)

Based on a case control study of 94 renal adenocarcinomas in male cigarette smokers, dose-dependent relative risks of 1.5 - 2.2 were reported. The authors concluded: "-- the relationship between cigarette smoking and renal adenocarcinoma is moderate, with the overall relative risks for cigarette smokers = approximately 2. The relationship is of lesser magnitude than for bladder cancer and renal pelvic cancer."

Armstrong et al, 1976 (Brit. J. Cancer 33:127-136)

Based on 106 cases of parenchymal and 33 cases of pelvic cancers, the relative risks for current cigarette smokers were 1.1 and 1.8, respectively. The authors concluded: "The data presented -- confirm the findings of Schmauz & Cole (1974) of a positive association between cigarette smoking and cancers of the renal pelvis in men -- In contrast, the data on cancers of the renal parenchyma suggest that this disease is not associated positively with tobacco use."

Rogot & Murray, 1980 (Public Hlth Reports 95:213-222)

In a cohort mortality study based on a 16 year follow-up of 290,000 veterans and 175 kidney cancer deaths, overall kidney cancer mortality ratios of 1.4 and

1.2 were reported for current and ex-cigarette smokers, respectively; the corresponding lung cancer ratios were 11.3 and 4.0.

Davis, Bridbord & Schneiderman, 1981 (In, Quantification of Occupational Cancer, eds, Peto & Schneiderman, pp. 304-306, Cold Spring Harbor, 1981)

In a review of attributable risks in various smoking-related cancers including kidney, it was emphasized that: "The proportion of smokers is highest in those occupations that are most likely to involve occupational exposures to other environmental hazards. -- smoking is confounded by occupation."

Morrison & Cole, 1982 (In, Cancer Epidemiology and Prevention, eds. Schottenfeld & Fraumeni, W. B. Saunders, pp. 925-935, 1982)

"-- cigarette smoking and kidney cancer are associated. Whether the association is causal or reflects some other cause with which smoking is associated remains to be learned."

Surgeon General, 1982 (The Health Consequences of Smoking: Cancer. Report of the Surgeon General, pp. 113-122, USDHEW, 1982)

Mortality ratios of kidney cancer in six prospective and four retrospective studies reviewed ranged from 1.4 - 2.7 and 1.1 - 10.0, respectively; the 10-fold risk finding was based on a study of 18 renal pelvis cancers and smokers in excess of 2.5 packs/day. Among the four retrospective studies reviewed, the only study differentiating pelvic from parenchymal cancers (Armstrong et al 1976), reported mortality ratios of 1.8 and 1.1, respectively. The report emphasized: "Other studies also reported an increasing relative risk of renal adenocarcinoma and cancer of the renal pelvis in cigarette smokers --." The report concluded: "Thus, the strength of the association related to kidney cancer risk is less marked than that for cancer" of other sites such as lung, larynx, esophagus and mouth.

McLaughlin et al, 1983 (J. Nat. Cancer Inst. 71:287-291)

Based on a case control study of 74 cancers of the renal pelvis, odds ratios of 7.6 and 5.8 were reported in male and female cigarette smokers, respectively; the odds ratios exceeded 10.0 for heavy smokers of both sexes. The authors concluded that "-- cigarette smoking -- is the major cause of this cancer."

McLaughlin et al, 1984 (J. Nat. Cancer Inst. 72:275-284)

Based on a case control study of 495 renal cell cancers, odds ratios of 1.6 and 1.9 were reported for male and female cigarette smokers, respectively, with significant dose-response relationships in both sexes. The authors, however, emphasize that "-- smoking is a more potent risk factor for cancer of the renal pelvis."

McLaughlin et al (NCI) 1987 (Brit. J. Ind. Med. 44:119-123)

"-- The effect of cigarette smoking appears greater for renal pelvis cancer rather than renal cell cancer."

Schulte et al, 1987 (Occup. Med. State of the Art Reviews 2:85-107)

In an extensive review of a wide range of occupations associated with excess kidney cancer risk, it was emphasized that: "The etiology of tumors of the renal pelvis appears, in many respects, to be similar to that of the bladder. -- For both renal cell cancer and cancer of the renal pelvis, there is a clear association (with smoking), although the relationship is much stronger for cancer of the renal pelvis." The report also emphasized that kidney cancer -- "is a relatively rare cancer, and many of the studies that have been performed could not detect risks smaller than four-fold."