Brief Report

Update to Vinyl Chloride Mortality Study

The mortality experience of 593 Dow Michigan Division employees potentially exposed to vinyl chloride was recently updated through 1982. An earlier study evaluated this group for the time period 1942 through 1973 and found an excess of total malignancies among the subgroup considered to have the highest exposure.[1] However, during the additional period of follow-up (1974 to 1982), a deficit of malignant neoplasms was observed for this subgroup.

During the 9 years of additional follow-up, the total deaths in the study group increased by 63 (to 155) and the person-years by 4,234 (to 17,114). The study's primary focus was directed at cancers of the liver (including angiosarcoma), biliary tract, lung, and brain, and to emphysema because of previous reports linking these conditions to vinyl chloride exposure in some work forces.[2,3] No deaths from angiosarcoma were found and there were no statistically significant excesses for any of the other conditions of interest.

Only those employees identified for the original study were included in the recent investigation and their work histories and exposures were updated through 1977 to ensure a minimum 5-year induction latency period. Consistent with past practice, separate analyses were conducted first including and then excluding the subset of employees with prior, competing exposures to arsenic insecticides.[4]

Cause-specific mortality among the study group was contrasted both with expected levels based on age- and era-specific mortality rates for US white men, and also with the mortality experience of 24,410 other male employees from this company location[5] who were not exposed to vinyl chloride or arsenic.

The comparison with US men is shown in the Table. Total malignant neoplasms were less than expected in the updated follow-up period (1974 to 1982), 12 *v* 16.3 and for the total follow-up period (1942 to 1982), 27 *v* 31.7. There were no statistically significant excesses for any of the diseases of interest. Similar results were seen for the comparison with other employees from this company location.

One death from liver cancer occurred in the study group (0.6 expected). The pathology and autopsy reports indicated that it was an intrahepatic bile duct carcinoma. There were two deaths from brain cancer (1.2 expected), one of which occurred less than 5 years after first exposure to vinyl chloride. Both individuals had only short-term exposure in jobs categorized as less than 200 ppm time-weighted average (TWA).

There were six deaths from emphysema or chronic obstructive pulmonary disease (approximately 5 expected). None of these individuals worked in areas where there was a potential for exposure to polyvinyl chloride dust.

An increased risk of lung cancer was not found (standardized mortality ratio = 89) and the study was capable of ruling out more than a 60% increase with 95% certainty. The lack of an association with lung cancer is consistent with the findings from the majority of other studies, especially those with the greatest statistical power to detect an excess.[6]

To examine for a dose-response relationship, subjects were allocated to one of four intensity-of-exposure categories (undetermined, less than 25 ppm TWA, 25 to 200 ppm TWA, and 200+ ppm TWA) based on their highest rated job assignment held for at least 1 month. During the updated follow-up period (1974 to 1982), a deficit of total cancer occurred in the highest exposure category (3 observed *v* 5.4 expected); however, for the entire follow-up period this subgroup experienced an excess which was confined principally to the men with at least 5 years of exposure above 200 ppm (7 observed *v* 2.8 expected). As was noted in the original study,[1] there was no predominant tumor site nor was there a unique histologic type among this subgroup, weakening any interpretation of a causal link with vinyl chloride.

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TABLE
Observed and Expected Deaths for Selected Causes Excluding Employees with Prior Exposure to Arsenic Insecticides

Cause of Death (ICD-8)	Period of Follow-up							
	1942–1973		1974–1982		1942–1982			
	Obs	Exp*	Obs	Exp	Obs	Exp	SMR†	95% CI‡
All causes	80	84.9	50	67.0	130	151.9	86	72–102
All cancers (140–209)	15	15.4	12	16.3	27	31.7	85	56–124
Digestive system cancer (150–159)	6	4.4	3	4.1	9	8.5	106	48–201
Liver cancer (155, 156)	0	0.3	1	0.3	1	0.6	—§	4–899
Respiratory system cancer (160–163)	4	4.9	6	6.4	10	11.3	89	42–163
Brain cancer (191, 192)	1	0.6	1	0.5	2	1.1	—§	23–682
All circulatory diseases (390–458)	38	41.1	27	34.3	65	75.4	86	67–110
All nonmalignant respiratory diseases (460–519)	6	4.2	3	4.4	9	8.6	104	48–198
All accidents (E800–E949)	7	8.4	1	2.5	8	10.9	74	32–144

* Expected numbers are based on calendar time and age-specific mortality rates of US white men.
† Standardized mortality ratio; observed divided by expected number of deaths × 100.
‡ CI, confidence interval of Obs/Exp.
§ Not calculated when expected number of deaths fewer than 5.

References

1. Ott MG, Langner RR, Holder BB: Vinyl chloride exposure in a controlled industrial environment. *Arch Environ Health* 1975;30:333–339.

2. Wong O, Whorton MD, Ragland D, et al: An update of an epidemiologic study of vinyl chloride workers. 1942–1982. Prepared for the Chemical Manufacturer's Association. October 17, 1986.

3. Purchase IFH, Stafford J, Paddle GM: Vinyl chloride: An assessment of the risk of occupational exposure. *Food Chem Toxicol* 1987;25:187–202.

4. Ott MG, Holder BB, Gordon HL: Respiratory cancer and occupational exposure to arsenicals. *Arch Environ Health* 1974;29:250–255.

5. Bond GG, McLaren EA, Cartmill JB, et al: Cause-specific mortality among male chemical workers. *Am J Ind Med* 1987;12:353–383.

6. Beaumont JJ, Breslow NE: Power considerations in epidemiologic studies of vinyl chloride workers. *Am J Epidemiol* 1981;114:725–734.

Nurses' Little Helpers: Care on the Cheap

The [British] government last week gave broad approval to the proposed changes to nurses education put forward 15 months ago by the United Kingdom Central Council for Nursing and Midwifery (UKCC). Nurses at the Royal College of Nursing's annual conference in Brighton gave John Moore, the Secretary of State for Health, a standing ovation. Only the fresh air or the nurses' recently fattened pay packets can explain the euphoria.

An analysis of Mr Moore's response gives grounds for concern for two reasons: first, the changes will mean most of the hands-on-care will be done by non-nurses or "nurse helpers," putting standards of patient care at risk; second, these changes smack of a plan to make "caring" cheaper.

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Who will take on nurse helper jobs? Work that has been labeled as menial and is poorly paid is unlikely to lead to a satisfied work force. Yet these are the people who will look after hospital patients in the future.

—From "UPFRONT" by Pamela Holmes in *New Statesman*, 1988;115(2984):5.

PROPOSAL FOR SUMMER 1980

INVESTIGATION AT B. F. GOODRICH

BACKGROUND

The vinyl chloride Medical Surveillance Program has provided data indicating that most of the standard biochemical (blood) tests for liver dysfunction are not effective in the detecting of early liver injury.

The tests with Indocyanine Green (ICG) dye clearance performed at the B. F. Goodrich Louisville Plant by the University of Louisville have shown it to be the most sensitive and specific test yet evaluated for detecting early damage. However, this test requires injection of dye into the vein, and a few (12/6000), minor reactions have occurred.

Bile acids (natural substances made by the liver for the gall bladder) were found to work similarly to ICG and are very effective as a screening test for liver disease. Again, however, the bile acids have to be injected intravenously and can cause phlebitis (inflammation of the vein).

A recent report (Gilmore and Thompson, GUT, Feb. 1980) indicates that bile acids could be administered orally, with the tests results being as or more sensitive in detecting chronic liver disease than when intravenously administered.

OBJECTIVE

We propose to compare the ability of ICG and orally administered bile acids in detecting early liver damage.

The advantages of the oral procedure would be several fold. It does not require any substance to be injected. It does not have to be analyzed immediately as does ICG (i.e. bloods can be mailed to central lab). If it is as good as ICG, it can be recommended to OSHA as a replacement for all the presently required federal tests, significantly reducing the expense of the large number of blood tests currently being done.

NUMBER OF EMPLOYEES: Biostatistically, 50 employees would be required. Most of these could be seen at the time of their regular blood work. Additional workers could be tested during the physical examination period, thus reducing additional time away from their job.

LENGTH OF TEST: The oral bile acid/ICG test will require 1 hour to be completed from the time of employee's arrival at testing place. If the oral test is found to be effective, it could be done in 15-20 minutes in the future (i.e. will not need to do ICG confirmation tests at that time).

NUMBER OF TESTS: 6 times x 2 (duplicate) x 50

= 600

COST

Test done locally:

Bile acid RIA testing kit cost $350./100 tests.

It would cost $2,100.00 to do the test locally and complete results by the end of the summer.

Test done via Abbott Laboratories:

none - results take 4-5 months