CHAPTER 31

NIOSH STUDIES OF OIL SHALE WORKERS

Joseph Costello, MSc
National Institute for Occupational Safety and Health
Division of Respiratory Disease Studies
Morgantown, West Virginia

INTRODUCTION

At the time the NIOSH shale oil studies were planned, oil prices were going up and there was renewed interest in such synfuels as shale oil and coal conversion products. Until recently, most of this work had been done on a demonstration project basis with a small workforce [1]. The Union Oil Company of California and the Colony project [2] of TOSCO and EXXON have begun commercial-scale projects in shale oil production on the Colorado plateau. Thus, it is important to assess the health effects of exposures encountered in the production of shale oil or kerogen. Three NIOSH studies involving this synfuel will be discussed: (1) a mortality study, (2) a case control study, and (3) a morbidity study.

METHODS

The first study initiated was the mortality study [3]. The universe involved was found to include over 1200 people. From this master group, a nonrandom sample of 713 white males was chosen from mining, retorting, maintenance or supervisory jobs involving actual production of shale oil. Clerical workers, short-term (less than one month) personnel and employees for whom we had names, but no other data, were excluded from the final cohort. Of this cohort, 321 men and 4 women were examined in the morbidity study. Table I presents a breakdown of the cohort by vital status.

Table I. Final Cohort

	In Morbidity Study	In Mortality Study
Known Living	325	485
Known Deceased		205
Death certificates		(181)
No death certificates		(24)
Vital Status Unknown		23
GRAND TOTALS	325	713

Standard populations for determining expected deaths were: (1) all white males in the states of Colorado and Utah; and (2) all white males in the United States. Mortality statistics were averaged for the years 1968, 1969 and 1970 to calculate expected death rates. The U.S. population was used only for all causes to check for the "healthy worker effect." Standardized Mortality Ratios (SMR) were calculated using a modified life table procedure. Death certificates were coded by a nosologist utilizing the *Eighth Revision, International Classification of Diseases Adapted for Use in the United States* [4].

During the mortality study, it was determined that 16 men had died of cancer of the trachea, bronchus and lungs, and 15 men of cancer of the digestive organs and peritoneum. In an attempt to clarify the effects of certain risk factors, namely smoking, radioactive exposure and metal mining exposure, a case control study was done of 12 oil shale workers dying from respiratory cancer and 15 workers dying from digestive cancer. Two separate control groups were used: the first group comprised 27 oil shale workers who had died from diseases of the circulatory system; the second comprised 27 living oil shale workers. Workers were matched as closely as possible on the basis of age, job classification and length of service in that job. Odds ratios were calculated by the method of Guy [5]. The third study, the morbidity study, was related to the mortality study in that 485 known living persons of the mortality study formed the cohort from which 321 men and 4 women had agreed to participate. The remaining 160 workers could not be located, were unwilling to participate or were living at locations too costly or too impractical to visit. A control population was chosen on the basis of age from coal miners working primarily in three mines in Utah. Other possible control populations were considered, but because of health hazards such as radioactivity exposure in uranium and vanadium miners and millers, arsenic and other exposures in smelter workers, etc., it was felt that coal miners were the best of the poor choices. Each worker was questioned about his work history, smoking history, medical problems and respiratory symptoms. A complete dermatological examination was given by competent dermatologists, and sputum and urine cytologies were done. Only the results of the skin examination, sputum and urine cytologies will be reported here.

RESULTS

Mortality Study

Table II lists the SMRs for all causes and several specific causes of death utilizing Colorado-Utah white males as controls. We also show an SMR for all causes using all U.S. males as a control. Statistically significant increases in SMRs are seen for all malignant neoplasms and cancer of the colon. Nonstatistically significant increases are seen for cancer of the digestive organs and peritoneum, of the respiratory organs and of the trachea, bronchus and lungs. Statistically significant decreases are seen for all causes (U.S. control), diseases of the circulatory system and ischemic heart disease. The significant decrease in all causes may be a consequence of the "healthy worker effect" and is a common characteristic of working populations [6].

Smoking is always an item of interest when increased SMRs for respiratory and lung cancer are seen. Table III summarizes the smoking history data that was available for this cohort. Unfortunately, these data were only available for 325 living workers and 53 deceased workers. Smokers and ex-smokers account for 307 of the 378 men, or 81.2%, of this group. However, the value of 37.8% for smokers is a low prevalence for smokers in an industrial population [7].

Table II. Standardized Mortality Ratios

Cause of Death	Observed	Expected	SMR
All Causes (U.S. control)	205	246	83[a]
All Causes (CO & UT controls)	205	221	93
All Malignant Neoplasms	49	36	136[b]
Digestive organs and peritoneum	15	10	150
Colon	7	3.1	226[b]
Respiratory organs	16	11	145
Trachea, bronchus and lung	16	10	160
Diseases of Circulatory System	76	102	75[a]
Ischemic Heart Diseases	55	72	76[b]
Diseases of Respiratory System	18	18	100
Accidents	18	15	120

[a]Statistically significant, $p < 0.01$.
[b]Statistically significant, $p < 0.05$.

Table III. Smoking Data (morbidity cohort + case control cohort)

Smoking Status[a]	Number	Percentage
Ex-smokers	164	43.4
Smokers	143	37.8
Nonsmokers	71	18.8
TOTAL	378	100.0

[a]Smokers + ex-smokers = 307, or 81.2%.

The number of persons employed in the oil shale industry has been erratic, and lengths of employment have varied from weeks to months. Length of employment is skewed toward short-term employment, with a median of 9 months and an average of approximately 30 months. More than 50% of the workers have had only 2 years or less of employment in oil shale work. This limits the implications of oil shale exposure as a causative agent in the development of chronic disease.

Operating jobs, defined as those that required exposure to mining and retorting of the shale, were divided into the following four categories: mining, retorting, maintenance and miscellaneous. Table IV shows the SMRs (Colorado and Utah controls) for each of the four job categories by five broad causes of death. In the mining category of jobs, only accidents showed an elevated SMR and that was not statistically significant. Retorting had elevated SMRs for All Causes and cardiovascular diseases. Maintenance workers had elevated SMRs for all classifications of diseases shown except cardiovascular disease, for which these workers had a significantly reduced SMR. Workers in the miscellaneous category had decreased SMRs for three of the five causes, with an increased SMR for respiratory diseases and malignant neoplasms.

Table IV. Job Activity SMRs (operating staff)

Job Activity	All Causes	Malignant Neoplasms	Cardiovascular Diseases	Respiratory Diseases	Accidents
Mining	72	50[a]	56	109	152
Retorting	134	90	183	97	84
Maintenance	101	150	74[b]	120	157
Miscellaneous	89	139	68	165	67

#Men = mining-105; retorting-60; maintenance-311; miscellaneous-68.

[a]Underlined values mean fewer than five observed.
[b]Significant at 5% level.

Case Control Study

Table Va gives a breakdown of the various categories of smoking by disease. It is quite apparent that smoking is an important factor in lung cancer. Smokers account for all but one in both the case group and the deceased control group. For digestive cancer, smokers account for the majority in both the case and deceased control groups.

The results of the case control study are numerically illustrated in Table Vb. Looking at the results for lung cancer, we see elevated odds ratios* for radioactive exposure in the study group versus both control groups. The values are 7.2 using the deceased control and 7.9 using the living control. We also see an elevated odds ratio for smoking using the living control. Not surprisingly, the odds ratio is 1.1 for smoking using the deceased control group. As an association between smoking and various diseases of the circulatory system is suspected [8], there would be competition by both lung cancer and circulatory system disease, with the end result being a depressed odds ratio for smoking.

Table Va. Smoking–Case Control Study

Disease	Category	Case	Living Control	Deceased Control
Lung Cancer	Smokers	11	6	10
	Ex- and Nonsmokers	1	6	1
Digestive Cancer	Smokers	9	2	11
	Ex- and Nonsmokers	6	13	3

Table Vb. Odds Ratio–Case Control Study

		Odds Ratio	
Cause of Death	Exposure	Living Controls	Deceased Controls
Lung Cancer	Smoking	11.0	1.1
	Radioactivity	7.9	7.1
	Metal mining	1.3	0.3
Digestive Cancer	Smoking	9.8	0.4
	Radioactivity	2.2	7.2
	Metal mining	---	1.1

*The *odds ratio* is an important measure of the degree of association between an antecedent factor and a disease. The odds ratio is the odds (or probability) of the disease when the factor is present relative to the odds when the factor is absent.

Metal mining exposure was not related to lung cancer in this case control study.

A similar situation exists for digestive cancer. Again, we see elevated odds ratios due to radioactive exposure, except that while both ratios are elevated, that using the deceased control group is much higher: 7.2 versus 2.2. As we are concerned with relatively small numbers, an increase of two people makes a major change in the results. Smoking shows an elevated odds ratio for digestive cancer in the living control group of 9.8 and a depressed odds ratio of 0.4 in the deceased control group. As smoking is known to be associated with circulatory disease deaths (see above reference) and as the cause of death for the deceased population was circulatory system problems, the low odds ratio in this control group is expected. Metal mining exposure seems to have little influence as far as digestive cancer is concerned.

In summary, the case control study suggests that smoking and radioactive exposure have a stronger association with lung and digestive cancers than does oil shale exposure in this study.

Morbidity Study

Table VI presents a brief summary of the most important risk factors in the morbidity study. Although the study and control groups are well matched for age, the oil shale workers had worked longer in other oil-related occupations, uranium mining and farming. In addition, the oil shale workers had spent more of their working time outdoors than had the controls. Fewer of the oil

Table VI. Age, Smoking, Occupation and Other Variables for the Oil Shale and Control Groups

	Oil Shale	Control
Number	325	323
Mean Age, yr	56	56
Current Smokers, %	28	37
Mean Pack-Years	35	25
Mean Years in Oil Shale Work	6.0	0
Mean Years in Oil Shale Production Work	2.9	0
Mean Years in Other Oil Work	1.3	0.1
Mean Years in Uranium Mining	1.9	0.2
Mean Years in Other Mining	2.0	29.8
Mean Years in Farming	3.4	2.5
Mean Percentage of Time Spent at Work Out of Doors Over Last 20 Years	47	23
Mean Percentage of Time Spent Off Duty Out of Doors Over Last 20 Years	58	51

shale workers were smokers; despite this, however, their pack-years were considerably greater. These imbalances of risk factors, coupled with the short duration of exposure to oil shale, make interpretation difficult.

The mean of six years conceals a rather skewed distribution of work in oil shale; 50% of the oil shale group had worked fewer than four years. For work in oil shale production, the figures were 2.9 years for the mean and 1.3 for the median, with 90% having worked fewer than 8.4 years.

Sixteen men in both the study and control groups were suspected as having one or more tumors. Most of the suspected tumors were biopsied; eight basal cell carcinomas were identified in the coal miners group, while two basal cell and two squamous cell carcinomas were found in the oil shale group. The latter four tumors were on the nose, face, neck and finger, respectively. The corresponding exposures for these four men were as follows:

1. two years as office manager in oil shale for the first.
2. two years in oil shale as miner and foreman, but 19 years in uranium mining for the second.
3. three years in oil shale as powderman and carpenter, but nine years processing uranium and vanadium plus 38 years in ranching for the third.
4. four years in oil shale in mechanical design for the fourth.

The results of the dermatological examinations are shown in Table VII. Three statistics are given because the skewness of the distributions makes any single index either misleading or inadequate. Oil shale workers have a higher mean number per person of pigment changes and keratoses, a higher median number per person of nevi and a higher percentage of persons having nevi and keratoses. The control population has a higher mean per person of nevi, telangiectasiae and papillomata; a higher median number per person of telangiectasiae; and a higher percentage of persons having pigment changes, telangiectasiae and papillomata.

The results presented so far indicate that oil shale workers, despite their greater exposure to sun, oil shale, uranium and other oil work, and even with

Table VII. Summary Statistics of Dermatological Examination Findings

	Mean Number per Person		Median Number per Person		Percentage with Entity	
Entity	Oil Shale	Control	Oil Shale	Control	Oil Shale	Control
Nevi	4.3	5.4	2	1	77	64
Pigment Change	0.8	0.7	0	0	31	34
Telangiectasiae	18.5	22.0	12	14	80	83
Keratoses	3.0	1.9	0	0	43	37
Papillomata	1.7	1.9	0	0	45	47

fewer darker skinned people, have fewer skin abnormalities than do the controls. However, it was thought prudent to examine the effect of years of exposure using the oil shale group alone. This aspect was analyzed by fitting a logistic model to the data, using age, exposure to sunlight, other oil work, uranium mining and oil shale work as predictors. Standardization was not attempted as there were too many variables to correct for simultaneously in relation to the number of observations.

To show the effect of correcting for all variables except oil shale work on the prevalences, the full model, less the oil shale factor, was fitted. The observed prevalences and those predicted from that model are shown in Table VIII. The observed and predicted values for telangiectasiae do not show any systematic departures, while those for pigment changes, and particularly for keratoses, show systematic divergencies following the exposure gradient. Papillomata, on the other hand, show a contrary gradient in relation to exposure.

Table VIII. Prevalence of Various Skin Abnormalities in Relation to Exposure (oil shale workers)

	Exposure Years		
	0	2	5
Signs			
Pigment Changes			
Observed	26	34	38
Predicted[a]	28	32	33
Telangiectasiae			
Observed	76	86	83
Predicted[a]	79	85	86
Keratoses			
Observed	34	47	61
Predicted[a]	35	44	53
Papillomata			
Observed	70	48	38
Predicted	44	44	48
Age and Exposure			
Age, yr	55	55	61
Sun Exposure, yr	23	33	35
Other Oil, yr	1.4	1.6	0.9
Uranium, yr	1.8	2.7	1.2
Oil Shale, yr	0.4	3.1	9.1
n	177	79	69

[a]From model logit (prevalence/100) = a + b (age) + c (sun exposure) + d (other oil) + e (uranium).
Logit (p) = log (p/1-p) where p = prevalence/100.

The statistical significance of allowing for oil shale exposure was assessed by adding the extra (oil shale exposure) term to the model. Actinic keratoses showed a significantly increased prevalence wtih exposure ($p < 0.01$), while pigment changes showed a similar tendency but without displaying statistical significance at the conventional levels. Papillomata showed a decreased prevalence but, again, not at the usual significance levels.

Sputum samples were taken for most of the participants. After unsatisfactory samples were rejected, 289 valid control samples and 289 valid oil shale samples remained. These were evaluated for metaplasia, dysplasia and neoplasia. One case of neoplasia was detected in the control group. The remainder were classified as metaplasia without dysplasia, and dysplasia (mild, moderate and severe) without regard to metaplasia. Nearly all the dysplasia was in the mild grade. Table IX presents the cytology findings for controls and oil shale workers by smoking group. Among smokers, the oil shale group had a higher proportion with metaplasia than the controls; however, this did not constitute a statistically significant difference ($\chi^2 = 2.48$). Correction for age and pack-years did not reduce the observed disproportion to any great extent. The ex-smokers showed a similar tendency to a higher rate of metaplasia, while the nonsmokers revealed the opposite, though again nonsignificant, effect ($\chi^2 = 0.58$). For dysplasia, both smokers and ex-smokers showed slight excesses among the oil shale workers, but nonsmoking controls had about twice the prevalence as the oil shale group. Statistical significance was not achieved ($\chi^2 = 1.98$).

A logistic model relating years of work in oil shale production to presence or absence of signs of metaplasia and dysplasia was fitted to the oil shale group divided into current smokers and nonsmokers. For metaplasia, there was a positive relation with production work in both smoking groups ($p < 0.05$ for both), and the two coefficients were similar in magnitude. For

Table IX. Pulmonary Cytology by Smoking Habits and Study Group

	Normal Cytology		Metaplasia, No Dysplasia		Dysplasia		n	
	Oil Shale	Control	Oil Shale	Control	Oil Shale	Control	Oil Shale	Control
Smokers	60	51	34	24	26	25	88	105[a]
Ex-smokers	45	51	31	26	24	23	143	117
Nonsmokers	78	53	22	28	10	19	58	67
						TOTALS	289[b]	289[b]

[a]One additional control had an invasive malignancy.
[b]Fewer than overall totals of missing and unsatisfactory samples.

dysplasia, both smoking groups showed a negative trend with years of exposure, although these were both statistically significant. One rather strange feature of these analyses is that age did not appear to be related to either metaplasia or dysplasia. In addition, pack-years among the smokers was not significantly associated with these two factors.

In summary, actinic keratoses showed a significant and positive association with oil shale exposure after allowing for age, sunlight and other exposures. In addition, the prevalence of metaplasia was positively associated with years of oil shale production work.

CONCLUSIONS

SMRs were calculated for a cohort of 713 oil shale workers who were employed primarily at the U.S. Bureau of Mines Anvil Points oil shale facility at Rifle, Colorado from 1947 through 1969. In addition, 325 living oil shale workers from the above cohort were examined in a morbidity study primarily aimed at dermatological changes. The following conclusions were generated from the data collected:

1. Oil shale workers appear to exhibit the "healthy worker effect." With one major exception (see No. 4), the SMRs for major disease classification have either been normal or less than normal.
2. Oil shale workers show decreased deaths for all causes and for diseases of the circulatory system, including ischemic heart disease. They show no difference in SMR for diseases of the respiratory system and a slight, but not significant, increase for accidents.
3. Maintenance workers tend to have the highest SMRs and mining workers the lowest.
4. Oil shale workers show a significant increase of malignant neoplasms, particularly for the colon, and fewer for cancer of the trachea, bronchus and lung. In light of the other carcinogenic risk factors previously mentioned and investigated in the case control study, it is difficult to implicate oil shale exposure, per se, as the responsible agent for the increase in SMR for respiratory and digestive cancers. The case control study indicates a stronger association between these cancers and exposure to radioactivity and smoking than with exposure to oil shale.
5. The prevalence of actinic keratoses was associated significantly and positively with oil shale work exposure after allowing for age, sunlight exposure and other exposures.
6. The prevalence of metaplasia was positively associated with years of production work in oil shale.

ACKNOWLEDGMENTS

The author gratefully acknowledges the following persons who assisted in the morbidity study: Dr. John Burkart and Mr. Gene Turner of UBTL for the collection of data; Drs. G. Kruger, J. Zone and W. Rom of the University of Utah for dermatologic services and medical expertise; and Mr. Michael Attfield of NIOSH for statistical analysis.

REFERENCES

1. Costello J: Morbidity and mortality study of shale oil workers in the United States. Environ Health Perspect 30:205–208, 1979.
2. Firms begin major synfuels studies. Oil and Gas J March 30:41–42, 1981.
3. Costello J: Retrospective mortality study of oil shale workers, 1948–1977. 13th Oil Shale Symposium Proceedings, CSM, Golden, Colorado, 1980, pp. 369–375.
4. U.S. Department of Health, Education, and Welfare. Eighth Revision International Classification of Diseases, adopted for use in the United States. Washington, DC: National Center for Health Statistics, 1967. Public Health Service Publication Number 1693.
5. Guy WA: Contributions to a knowledge of the influence of employments on health. J Roy Stat Soc 6:197–211, 1843.
6. McMichael AJ: Standardized mortality ratios and the "Healthy Worker Effect," scratching the surface. JOM 18(3):165–168, 1976.
7. Ortmeyer CE, Costello J, Morgan WKC, Swecker S, Petersen M: The mortality of Appalachian coal miners, 1963–1971. Arch Environ Health 29:67–72, 1974.
8. U.S. Department of Health, Education and Welfare. Smoking and health, a report of the Surgeon General. Washington, DC: U.S. Public Health Service, 1979 (DHEW Publication No. (PHS)79-50066, pp. 4-1–4-76).

HEALTH ISSUES RELATED TO METAL AND NONMETALLIC MINING

Edited by
WILLIAM L. WAGNER
WILLIAM N. ROM
JAMES A. MERCHANT

BUTTERWORTH PUBLISHERS
Boston • London
Sydney • Wellington • Durban • Toronto

An Ann Arbor Science Book

Ann Arbor Science is an imprint of Butterworth Publishers

Library of Congress Catalog Card Number 82-48649
ISBN 0-250-40610-1

10 9 8 7 6 5 4 3 2 1

Butterworth Publishers
10 Tower Office Park
Woburn, MA 01801

Printed in the United States of America