RISK OF DUST-INDUCED LUNG DISEASE IN OIL SHALE WORKERS

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ABSTRACT

Risks of non-neoplastic lung diseases for future U.S. oil shale worker dust exposures were estimated. In the absence of an active industry, health effects rates from surrogate industries were utilized. The risk of chronic bronchitis, chronic airway obstruction, and pneumoconiosis was quantified from British coal worker data. The risk of occupational silicosis in oil shale miners was estimated using Peruvian metal mines and Vermont granite worker data. Ten percent SiO_2 composition of the dust in the respirable range from oil shale mining and crushing was used. Risk estimates were calculated at the nuisance dust threshold limit value (TLV) of 5 mg/m^3 and at the current SiO_2 TLV of 100 $\mu g/m^3$. Silicosis was the dominant pulmonary health effect, but at the 5 mg/m^3 dust level, pneumoconiosis, chronic bronchitis, and chronic airway obstruction are also important risks. Designing oil shale facilities to meet the nuisance dust TLV may not provide adequate protection to the future generations of workers involved in oil shale extraction and processing.

INTRODUCTION

The analysis of health risks in the emerging oil shale industry can aid in the prevention of occupational accidents and diseases. The analysis can enable health professionals, industry decision makers, and engineers to reduce the long-term costs of oil shale technology and guard against long-term health concerns. The objective of an oil shale risk analysis reported in 1982 (1) was to estimate the potential human health and environmental concerns to establish important research needed to reduce the associated uncertainties. The analysis is for an occupational workforce of a one million barrels-per-day steady state production scenario described elsewhere (2). The results indicate that occupational exposure to dust will be a primary health concern for a future oil shale industry. The purpose of this paper is to describe the potential non-neoplastic dust-induced pulmonary diseases in the oil shale workforce and examine health risk implications of the 5 mg/m^3 nuisance dust level as a design criteria for ventilation of oil shale mines.

BACKGROUND

All humans are normally exposed to dust-laden air. The respiratory system has the ability to cope with these particles, but the ability for self-protection and repair of injury can be exceeded. Excessive dust deposition can cause airway reactivity, hyperproduction of mucus, hypertrophy of mucus-secreting glands, macrophage recruitment and ingestion of particles, chronic proliferative or inflammatory reaction, fibrosis, and cell metaplasia or malignant transformation. The occurrence of any of these conditions is dependent on the nature and number of specific particles deposited and retained as well as coexisting inhaled agents and the response of the individual. Inhalation of dust as a result of oil shale mining, crushing, and retorting operations, and adverse physiological reaction to that dust is likely in the oil shale workforce. The non-neoplastic lung response to respirable dust can be categorized under the following: pneumoconiosis, silicosis, chronic bronchitis, and chronic airway obstruction.

In order to present a comprehensive picture of the risk of non-neoplastic pulmonary disease in the oil shale industry, the relationship of pneumoconiosis, silicosis, chronic bronchitis, and chronic airway obstruction needs to be understood. The VENN diagram shown in Figure 1 demonstrates this relationship. The overlapping areas indicate that two or more of these health effects can be present in the same individual simultaneously. There are two forms of pneumoconiosis, simple and complicated. While simple and complicated pneumoconiosis can exist in

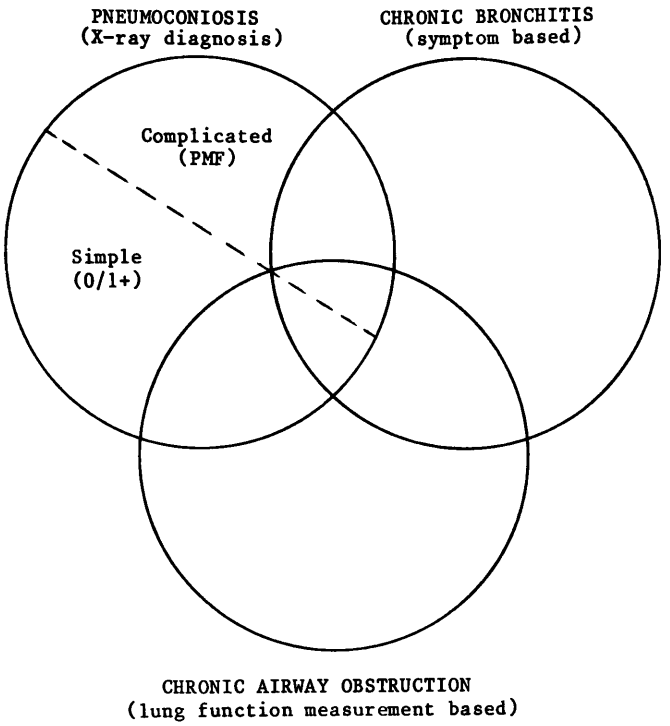

Figure 1. Relationship of Respiratory Disease Risk Measures (VENN Diagram for Dust in Lungs)

the same individual, in the analysis a person with both is considered to be in the complicated pneumoconiosis group.

Pneumoconiosis is the deposition of dust in the lungs with inflamation commonly leading to fibrosis of the lungs. Some forms of pneumoconiosis have serious health implications while others do not. Specific names for pneumoconiosis include anthracosis, byssinosis, silicosis, and coal worker's pneumoconiosis (CWP). These are all different forms of pneumoconiosis. Silicosis is a form of pneumoconiosis resulting from inhalation of free silica (quartz) dust, characterized by formation of small, discrete nodules. In advanced cases, a dense fibrosis and emphysema with impairment of respiratory function may develop. Silica dust is considerably more fibrogenic than other dust and the risk from this exposure was determined separately. Silicosis has the same relationship to chronic bronchitis and chronic airway obstruction as that depicted in Figure 1 for pneumoconiosis.

The diagnosis of pneumoconiosis is made on the basis of history of exposure and chest X-ray. Radiological classification of simple pneumoconiosis has been developed primarily for epidemiological purposes and the current international system is known as the International Labour Office 1980 (3) system and consists of twelve steps. Category 0/1 is the third step and represents the first suspicion that pneumoconiosis is present. While simple pneumoconiosis is generally thought not to be related to increased mortality, a recent 22-year follow-up study of simple pneumoconiosis has shown consistent and statistically significant reduction in life expectancy (4). Complicated pneumoconiosis is diagnosed radiologically when one or more large opacities at least one centimeter in diameter are present. Complicated pneumoconiosis is termed Progressive Massive Fibrosis (PMF) because, unlike simple pneumoconiosis, PMF can progress even after exposure to dust has ceased and is associated with significant morbidity and mortality. In general, pneumoconiosis and silicosis are radiographically indistinguishable.

Chronic bronchitis is characterized by a chronic or recurrent excess of mucus secretion in the bronchial tree. The Medical Research Council modified definition (5, 6) "presence of cough and phlegm on most days for at least three months in the year for at least three successive years" was used. The predominate pathological change is hypertrophy and hyperplasia of the mucus secreting glands of the trachea, bronchi, and the bronchioles. Chronic bronchitis has many causes and occurs in significant proportions of the general population. It can be caused by inhalation of dust and fumes, but cigarette smoking is the most important etiologic factor. Since the major lesion for chronic bronchitis is in the larger airways, it has been argued that total dust would be a better predictor of risk than respirable dust. However, a recent study in the coal mining industry showed that respirable dust was just as good a predictor as total dust (7).

Chronic airway obstruction is an increased resistance to the passage of air in and out of the lung due to narrowing of the small airways of the bronchial tree. It is usually diagnosed by determining that the amount of air forcibly expired from the lung is less than normal. The diagnosis of chronic airway obstruction is based on pulmonary function rather than symptomatology (as used for chronic bronchitis) or radiologic analysis (as used for pneumoconiosis). The most commonly used pulmonary

function measure for airway obstruction is a reduction in the Forced Expiratory Volume in one second (FEV_1) to some percentage of the expected value: the lower the percentage, the more stringent the criteria for inclusion. An FEV_1 <80% predicted is considered outside the normal range and an FEV_1 <65% is frequently associated with clinical symptoms. Chronic airway obstruction may be caused by a number of irritants or diseases which damage the small airways. In addition to dust, this includes pulmonary tuberculosis, cigarette smoking, and silicosis. Among indices of chronic respiratory disease, chronic airway obstruction is the strongest predictor of diminished life expectancy.

OIL SHALE DUST DISEASE

Pneumoconiosis in Shale Miners

Recently, Seaton described four patients having diagnosed pneumoconiosis with no other occupational experience other than a lifetime's work in oil shale mines (8). The clinical and histological features resembled the simple and complicated pneumoconiosis of coal miners (CWP), though the macrophages contained brown particles rather than black ones. The dust in the lungs of three of the subjects were found by infra-red analysis to be similar to that of the Scottish oil shale: 70% ash content composed largely of silicates, kaolin, and mica and approximately 10 to 15% free silica. The mineralogy of oil shale and coal is similar except for the increased mineral to organic content ratio of oil shale (indicating a greater amount of quartz in oil shale). Perhaps most convincingly, X-ray diffraction analysis of dust from one patient was shown to be similar to that of the oil shale seam he had worked, including identical peaks for quartz, kaolinite, illite/montmorillonite, and feldspar. Two of the patients had PMF with the lesion in one patient consisting of a 4.5 centimeter cavitating lesion in the right upper lobe.

The Scottish oil shale industry existed from 1865 to 1962 in a small area of Scotland just west of Edinburgh. Since two of Seaton's four cases of pneumoconiosis from oil shale exposure came from autopsies at Bangour General Hospital located in the middle of the oil shale region, a review of autopsies from 1965 to 1982 was undertaken in collaboration with Dr. G. Sclare, Chief Pathologist. Review of the 3092 autopsies in males at Bangour General Hospital from 1965 to 82 yielded 266 (8.6%) with a signout diagnosis of pneumoconiosis (Table 1). An additional 52 cases, many of whom died at home or in a local infirmary, were sent for autopsy to Bangour General Hospital during this period by the Pneumoconiosis Review Panel (PRP). The records for 1963-64 contained an additional 36 cases of pneumoconiosis, most of whom appeared to have been referred by the PRP and are not included in the analysis because the records were not as well kept as those beginning in 1965. The pneumoconiosis benefits program is administered differently in Scotland than in England where autopsy is mandatory for anyone receiving or seeking benefits. The program is voluntary in Scotland, leaving it up to the next-of-kin whether or not to have an autopsy. However, the autopsy is required for obtaining previously refused benefits.

The occupation of 195 (73%) of the 266 pneumoconiosis cases found in routine autopsy was mining. In only 65 was there further designation of occupation with 8 being oil shale miners and 57 being coal miners, an oil shale miner to coal miner ratio of 0.14. These data may provide an estimate of the relative risk of pneumoconiosis in oil shale miners relative to coal miners living in the same time period. West Lothian census figures show the following oil shale to coal miners ratios for the years indicated: 0.63 for 1891, 0.50 for 1911, 0.49 for 1921, 0.33 for 1931, and 0.19 for 1951. The ratio of 0.14 for autopsies from 1965 to 1982 was judged to be a reasonable reflection of the population ratio. Therefore, the unadjusted data indicate the risk of pneumoconiosis in the two groups is equal. However, the ratio of oil shale miners to coal miners in the pneumooniosis group may not be representative of the ratio in the entire autopsy group. Furthermore, a consistent difference in autopsy-seeking behavior among oil shale miners as compared to coal miners would introduce a bias difficult to overcome. Excluding the PRP cases (all of those the PRP specifically sent to Bangour General Hospital for autopsy) was one method chosen to minimize this bias. One may suspect that coal miners would have tended to seek autopsy more frequently than oil shale miners because of the PRP benefits program, but this conjecture cannot be tested. Three studies are currently funded by the U.S. Department of Energy to further document the health effects from the Scottish oil shale industry and those results may have further implications for the oil shale risk analysis effort (9).

Table 1. Bangour General Hospital Autopsy Review for Diagnosis of Pneumoconiosis 1965-1982

Year	Total Male Autopsies	Pneumoconiosis Cases from Routine Autopsy						Pneumoconiosis Cases from Pneumoconiosis Review Panel		Total Cases Pneumoconiosis
		Total (%)	Occupation Coal Miner	Shale Miner	Miner Unspec.	Miner Total	Other	Total	Occupation Coal Miner	
1965	185	44 (24)	5	3	27	35	9	5	5	49
1966	205	39 (19)	11	0	16	27	12	5	5	44
1967	221	38 (17)	8	0	21	29	9	4	4[a]	42
1968	202	15 (7)	3	0	9	12	3	4	4	19
1969	197	24 (12)	2	0	16	18	6	8	8	32
1970	204	12 (6)	4	0	4	8	4	4	4[b]	16
1971	229	14 (6)	2	1	9	12	2	5	5	19
1972	175	14 (8)	4	1	5	10	4	5	5	19
1973	191	16 (8)	3	2	5	10	6	4	4	20
1974	174	10 (6)	4	0	4	8	2	5	5	15
1975	154	9 (6)	5	0	3	8	1	0	0	9
1976	169	11 (6)	1	0	4	5	6	0	0	11
1977	148	5 (3)	0	0	3	3	2	2	2	7
1978	100	3 (3)	1	0	2	3	0	0	0	3
1979	95	1 (1)	1	0	0	1	0	0	0	1
1980	146	2 (1)	1	0	0	1	1	0	0	2
1981	158	4 (2)	1	0	1	2	2	1	1	5
1982	139	5 (4)	1	1	1	3	2	0	0	5
Total	3092	266 (8.6)	57	8	130	195	71[c]	52	52	318

[a] 1 shale miner 9 years
[b] 1 shale miner 23 years
[c] 21 of these were foundry workers, and one each quarry worker, brickworker, and oil worker.

British Coal Miner Data Dust Surrogate

There is an extensive literature base concerning pulmonary health effects in coal miners in Britain largely resulting from the Pneumoconiosis Field Research sponsored by the National Coal Board through the Institute for Occupational Medicine (Edinburgh). This includes cumulative respirable dose-response information for pneumoconiosis, both simple and PMF (10, 11), chronic bronchitis (12), and chronic airway obstruction as defined by loss in FEV (13). Differences in population groups, methods of analysis and, updating of dust exposures make it difficult to obtain a comprehensive picture of these health effects of interest from published studies. Recent reanalysis of the miners followed for ten years in twenty mines throughout Britain (which served as the basis of the current coal dust Threshold Limit Value (TLV) of 2 mg/m^3 in the U.S.) permits an integrated presentation of disease prevalence (14, 15). Table 2 presents the prevalence of the health effects of interest in the 2907 smokers and 567 non-smokers age 25 to 64 by four cumulative respirable dust exposure groups. The mean levels in units of gh/m^3 (gram-hours/cubic meter) were 42 gh/m^3, 129 gh/m^3, 201 gh/m^3, and 347 gh/m^3. The prevalence of pneumoconiosis is shown for the combined group since smoking has been shown to have no effect on pneumoconiosis risk (16). Smoking, however, is a major risk factor for both chronic bronchitis and chronic airway obstruction. For chronic airway obstruction, two levels of FEV_1 were considered. Persons with FEV_1 <80% are considered abnormal, while those with FEV_1 <65% have shortened life expectancy. The common occurrence of bronchitis and chronic airway obstruction are also indicated in Table 2. It can be seen that dust exposure has a major impact on these conditions in both smokers and non-smokers.

Silicosis Surrogate Data

The relationship between exposure to free silica in dust and the risk of silicosis is presumably not specific to a particular workforce or industry. Risk estimates in this area must be regarded as only approximate since a recent NIOSH review (17) made the following statement about information gaps: "Additional laboratory and epidemiological studies are needed to determine the relationship between silica dose, as measured by the respirable mass method, and the risk of developing silicosis." Quantitative estimates from Peruvian metal miners (18) and Vermont granite workers (19) were used to construct a dose-response function for this disease. Unfortunately, no distinction was made between simple and complicated silicosis so that the risk estimates are for their combined prevalence. Silicosis appears to be a cumulative effect over decades of exposure, with the free silica reaching the alveoli. The criterion for presence of the disease entity silicosis was defined as radiographic evidence of silicosis, consistent with the textbook definitions (20) which rely on X-ray changes and an occupational exposure history.

Paretto (18) conducted an investigation of silicosis in Peruvian metal miners expressly to evaluate the adequacy of the recommended TLV. Thirteen mines with adequate numbers of workers, exposure data, and health outcome information were included. Exposures were calculated as an average person-weighted dust level multiplied by time. It was shown that the TLV formula then in effect would not be adequate to protect all workers from silicosis, perhaps due to the altitude (over 3000 meter) or the 48-hour work week. The linear regression equation (with an r-squared of 0.628) from these data is as follows:

$$Y(\%\text{silicosis}) = -4.68154 + .00428\ X,$$

where X is cumulative exposure in $\mu g\ SiO_2\text{-yr/m}^3$. A series of studies on silicosis in Vermont granite shed workers (21, 22, 23) reported on approximately 800 workers employed in 1971. Although these data served as the basis of the current NIOSH recommended silica standard of 50 $\mu g/m^3$, this standard has been sharply criticized (24). One weakness identified was with the X-ray interpretation which showed a 30% prevalence of silicosis for all miners exposed to 1650 $\mu g\text{-yr/m}^3$ or less. An earlier presentation of the Vermont granite shed worker study results enabled the construction of a linear dose-response curve which avoided this anomalous result (19). The prevalence data used was that of 485 granite shed workers X-rayed at the time dust control was introduced in 1937-40 and had been estimated to have been exposed to an average of 20 mppcf (million particles per cubic feet) or 160 ug/m using the conversion factor suggested by Theriault (21) of 10 mppcf = 0.08 mg/m^3. Although the 502 granite cutter workers X-rayed at the same time had a higher prevalence, the estimated exposure level was over too wide a range (40-60 mppcf) to permit use of these data. The linear regression equation (with an 4-squared of 0.969) from

Table 2. Prevalence of Non-neoplastic Pulmonary Health Effects in British Coal Miners [a]

Group	Condition	Percentage Prevalence			
		Cumulative Dust Exposure [b] (gh/m^3)			
		0-99	100-159	160-249	250+
Pneumoconiosis for Smokers and Non-smokers	Simple Pneumoconiosis (0/1+)	8.4	11.1	29.0	51.5
	Complicated Pneumoconiosis	0.1	0.5	2.0	5.9
	(Number of Miners)	(906)	(811)	(856)	(901)
CB and CAO for Non-smokers	Bronchitis	10.6	12.1	16.8	30.2
	FEV_1 <80%	8.0	15.2	23.6	27.8
	Both Bronchitis and FEV_1 <80%	0.6	2.8	6.7	11.8
	FEV_1 <65%	1.8	6.3	7.4	6.7[c]
	Both Bronchitis and FEV_1 <65%	0	1.7	3.6	4.1
	(Number of Miners)	(125)	(146)	(140)	(156)
CB and CAO for Smokers	Bronchitis	31.5	39.3	46.0	51.0
	FEV_1 <80%	21.7	24.8	33.5	41.0
	Both Bronchitis and FEV_1 <80%	10.3	14.7	20.5	27.0
	FEV_1 <65%	7.5	9.9	11.2	15.4
	Both Bronchitis and FEV_1 <65%	4.3	7.2	7.6	11.0
	(Number of Miners)	(781)	(665)	(716)	(745)

[a] Age distribution used is that of entire 4119 coal miners in the study who worked 10 years or longer in 20 coal mines in U.K. as follows: ages 25 to 34 as 8.6%, ages 35 to 44 as 33.1%, ages 45 to 54 as 35.9%, and ages 55 to 64 as 22.4%.

[b] Mean levels for groups are 42, 129, 201, and 347 gram-hours/cubic meter (gh/m^3).

[c] May be anomolous point due to small sample size.

these data is as follows:

Y(% silicosis) = -17.825 + 0.015 X

where X is cumulative exposure in μg SiO_2-yr/m^3. The silicosis prevalence was estimated using the average of the two regression equations.

Risk Estimates of Pulmonary Health Effects

An estimate of the oil shale dust concentration and duration of exposure to that concentration is needed to derive a risk estimate. The only available oil shale mine dust measurements come from studies of pilot operations and may not adequately represent exposures in a commercial operation. The current dust standard in oil shale mines is the nuisance dust TLV of 5 mg/m^3 respirable dust and was selected as the estimate for dust exposures at a commercial level. A current estimate of the average respirable SiO_2 content in oil shale dust in western Colorado is 10% (25), indicating the 5 mg/m^3 dust level may contain 500 μg/m^3 SiO_2. Consequently, risk estimates were also calculated at the current SiO_2 TLV of 100 ug/m^3 which corresponds to a dust level of 1 mg/m^3.

Duration of exposure is provided for several different durations of employment and for the average in a steady state industry. Multiplication of the exposure terms (dust concentration) by the number of hours worked per year and by the number of years worked indicates the cumulative respirable dust exposure in gh/m^3 for the given prevalence rate of pneumoconiosis, chronic bronchitis, and chronic airway obstruction. The British surrogate data are based on 1740 hours as the yearly average. Consequently, for 40 years of work at the TLV of 5 mg/m^3, lifetime exposure is estimated as 348 gh/m^3 and at 1 mg/m^3 it is 70 gh/m^3. In order to derive estimates from the British surrogate data, an assumption was made about age composition of the workforce. The silicosis surrogate data are reported in μg-yrs/m^3 units and does not require assumption of number of hours worked per year nor age composition of the workforce.

The anticipated distribution of the workforce in terms of years worked in the oil shale industry was needed for all estimates. Estimated risks are presented specifically for workers employed 10, 20, 30, and 40 years. To derive an aggregate risk estimate, it was assumed that a 10-year worker is replaced with another worker who stays 10 years, and so on for 20-, 30-, and 40-year employees. If one simulates a 120-year industry, there would be 12 sets of 10-year workers, 6 sets of 20-year workers, 4 sets of 30-year workers, and 3 sets of 40-year workers. The ratio 12:6:4:3 corresponds to percentages of 48, 24, 16, and 12. These were the weights applied to derive an overall risk estimate. Table 3 contains a summary of prevalence of all four health effects of interest under the workforce employment conditions.

Table 3 results show the 5 mg/m^3 dust, 500 μg/m^3 SiO_2 level, a level of SiO_2 five times the current TLV, results in a considerable prevalence of silicosis. For this extreme exposure level, the overall workforce estimates range from 36% to 80%, with an average of 58%. This average estimate ranges between 33% at 5% SiO_2 and 83% at 20% SiO_2. Even the lowest risk group, 10-year workers, have a prevalence range from 17% to 57%, with an average of 37%. Risk of silicosis calculated at the current 100 μg/m^3 TLV is considerably lower. No cases are expected for 10-year workers, but among 40-year workers the risk would vary between 12% and 42%, with an average of 27%. The overall workforce risk would be lower, but still significant at the level of between 4% and 12%, with an average of 8%. Table 3 presents the individual surrogate results to illustrate the potential range for this estimate.

At the 5 mg/m^3 respirable dust level, the prevalence of simple pneumoconiosis in 10-year workers begins at a low of 8%, increasing to 52% in 30- and 40-year workers for a workforce prevalence of 25%. PMF prevalence is much lower among 10- and 20-year workers, but at 30 and 40 years, a 6% prevalence is expected with a workforce estimate of 2.2% prevalence. At the 1 mg/m^3 respirable dust level, the workforce simple pneumoconiosis prevalence is 8.4% and PMF is 0.1%.

Unlike silicosis and pneumoconiosis, chronic bronchitis (CB) and chronic airway obstruction (CAO) occur with an expected frequency in general populations not undergoing occupational exposures. These cases need to be excluded from risk estimates associated with the oil shale industry. The background prevalence rate is estimated as the rate in the workers exposed to less than 100 gh/m^3 respirable dust. The excess prevalence of each disease at each age is obtained by subtracting this estimate background rate from the total prevalence for each exposure and age. In effect, this workforce with the low exposure becomes an internal comparison group and avoids the criticism of external comparison groups.

Table 3. Post-exposure Prevalence of Silicosis, Pneumoconiosis, Excess Chronic Bronchitis, and Excess Chronic Airway Obstruction as a Function of Exposure Level and Years Worked

Duration of Employment (years)	Respirable Dust Exposure Level (mg/m^3)	Respirable SiO_2 Exposure Level ($\mu g/m^3$)	Workers at each Duration (%)	Cumulative Silica Exposure (μg-yr/m^3)	Silicosis Prevalence(%) Vermont Granite Worker Surrogate	Peruvian Metal Miner Surrogate	Average	Cumulative Dust Exposure (gh/m^3)	Prevalence (%) Pneumoconiosis Simple (0/1+)	PMF	Chronic Bronchitis S	NS	Chronic Airway Obstruction FEV_1 <65% S	NS
10	5	500	48	5000	57.2	16.7	37.0	87	8.4	0.1	0	0	0	0
20	5	500	24	10000	100	38.1	69.1	174	29.0	2.0	14.5	6.2	3.7	5.6
30	5	500	16	15000	100	59.5	79.8	261	51.5	5.9	19.5	19.6	7.9	4.9
40	5	500	12	20000	100	80.9	90.5	348	51.5	5.9	19.5	19.6	7.9	4.9
Composite			100		79.4	36.4	57.9		25.4	2.2	8.9	7.0	3.1	2.7
10	1	100	48	1000	0.0	0.0	0.0	17.4	8.4	0.1	0	0	0	0
20	1	100	24	2000	12.2	3.9	8.0	34.8	8.4	0.1	0	0	0	0
30	1	100	16	3000	27.2	8.2	17.7	52.2	8.4	0.1	0	0	0	0
40	1	100	12	4000	42.2	12.4	27.3	69.6	8.4	0.1	0	0	0	0
Composite			100		12.3	3.7	8.0		8.4	0.1	0	0	0	0

Prevalence numbers based on Table 2.
S - smokers, NS - non-smokers.

The workforce excess CB prevalence at 5 mg/m^3 is 9% for smokers and 7% for non-smokers. Likewise, the workforce excess CAO prevalence is 3.1% for smokers and 2.7% for non-smokers. The excess prevalence of cases with FEV_1 <80% is not shown because change in mortality risk has not been demonstrated at this level of reduced pulmonary function. The prevalence of excess cases of CB and CAO becomes insignificant at the 1 mg/m^3 respirable dust exposure level. A result of this analysis is that the excess cases of CB and CAO are of equal magnitude among smokers and non-smokers.

Premature Mortality from Pulmonary Health Effects

The approach to this estimate involved determining the relative risk (R) of mortality from all causes for each of the health effects. The case fatality rate (attributable portion expected to die as a result of the specified health effect) was calculated using the formula (R-1)/R. This proportion is then applied to the average number of new cases occurring each year in the steady state oil shale industry to determine the average number of premature deaths due to the disease.

Life table methods were used to determine premature mortality among over 1000 persons receiving silicosis benefits in Canada from 1940 to 1975 (26). The follow-up period was divided into four parts according to the time period when benefits were begun. Since 31% of the observed deaths in the first ten years were due to tuberculosis, R was taken as the average of the standardized mortality ratio for the remaining three time periods which were 1.54, 1.25, and 1.68 for a mean of 1.49. Support for this figure is provided by additional analysis of the deaths by cause for the period from 1950 to 1959. The excess nontuberculosis respiratory disease deaths accounted for 70% of the difference between observed and expected deaths from all causes. The average loss of life expectancy from respiratory disease deaths (excluding tuberculosis) was 5.5 years for persons dying during the 1940 to 1949 period and 6.4 years for persons dying during the 1950 to 1959 period.

A 22-year follow-up of mortality among coal miners according to radiological evidence of pneumoconiosis (simple or PMF) provided R data for these health effects (4). These data allowed computation of the relative mortality risk using the age distribution of the workforce noted in Table 2. Workers with no evidence of pneumoconiosis served as the population against which survival in the two diseased groups was compared. The R for simple pneumoconiosis (0/1+) was 1.19 while that for PMF was 1.30.

Data from the Tecumseh Study were used for the mortality rates for CB and CAO (27). The mortality ratio after four years of observation was 1.8 for CB and 1.6 in males with low FEV_1 scores (index below 8.5). Recent age-standardized mortality ratios, based on a 15-year follow-up, are 1.6 for CB and 1.8 for persons with FEV_1 <65% (28). Since these were based on a longer follow-up, they were used in the analysis.

The case fatality rates derived from these R are 0.33 for silicosis, 0.16 for simple pneumoconiosis, 0.23 for PMF, 0.37 for CB, and 0.44 for FEV_1 <65%. An estimate of the annual number of premature deaths can be derived by applying these rates to the annual incidence of each pulmonary health effect. The post-exposure prevalence rates in Table 3 may be taken as an approximation of lifetime incidence. Dividing these rates by an assumed 40 year life expectancy spreads the incidence equally over this interval. Table 4 contains a summary of the premature deaths from these health effects using these assumptions according to the two levels of dust exposure. In the case of CB and CAO, it is assumed that the workforce is 50% smokers. However, since the excess cases of CB and CAO are almost identical for smokers and non-smokers, changes in this proportion will not appreciably alter the number of cases from dust exposure. Treating each health effect independently, 24 new cases per 1000 workers are expected at the 5 mg/m^3 dust level and 500 $\mu g/m^3$ SiO_2 level, while only four new cases per 1000 workers should be expected at the 1 mg/m^3 dust level and 100 $\mu g/m^3$ SiO_2 level. Over seven premature deaths per 1000 would be expected with the former and one premature death per 1000 with the latter. Under both scenarios, approximately one half of the new cases will be lung silicosis and these cases will account for two thirds of the premature deaths.

The lung disease and fatalities for the exposures levels shown for a steady state oil shale industry are summarized in Table 4. Again, treating each health effect independently, 140 fatalities result at the extreme exposure level. This analysis will be continued and expanded to include uncertainty estimates under the current oil shale risk analysis.

Table 4. Summary of Pulmonary Disease Risk for 20,400 Workers Exposed to Dust

Health Effect	Case Fatality Rate (%)	Exposure Level	Excess Cases per 1000 per Year	Cases per Year	Premature Fatalities per Year
Silicosis	33	A	14.5	295	97
		B	2.0	41	13
Simple Pneumoconiosis	16	A	6.4	130	21
		B	2.1	43	7
Complicated Pneumoconiosis	23	A	0.55	11	2.6
		B	0.03	0.5	0.1
Chronic Bronchitis	38	A	2.0	41	15
		B	0.0	0	0
Chronic Airway Obstruction (FEV_1 <65%)	44	A	0.73	15	6.6
		B	0.0	0	0

A is exposure at the nuisance dust level:
5 mg (dust)/m^3 with 10% free silica (500 $\mu g/m^3$)

B is exposure at the silica TLV level:
1 mg (dust)/m^3 with 10% free silica (100 $\mu g/m^3$)

DISCUSSION

The results present the best estimates currently available from the oil shale risk analysis. A more precise dose-response estimate for SiO_2 is one of the greatest needs since this health effect plays such a dominant role when dealing with western Colorado oil shale which yields 10% SiO_2 in the respirable fraction. Control technology will undoubtedly be hard pressed to keep the respirable dust level at 1 mg/m^3 in order to stay at or below the current SiO_2 TLV. A recent summary of the OSHA inspection program involving free silica in mines indicated 20% of the samples were twofold or greater than the current TLV (17). Inclusion of a violation rate of this magnitude for the oil shale industry would seriously elevate the health risks, especially since a significant risk exists for silicosis at the current TLV. Design of the ventilation system of oil shale mines will directly affect worker respiratory health.

Further refinement of the estimates of the British coal miner data is expected because the data presented here in terms of four dust exposure groups are currently being analyzed to produce multiple logistic regression equations (15). This refinement will permit calculation of expected health effects at zero dust level for comparison with specific levels. This may result in somewhat higher rates of CB and CAO at the lower dust levels since the current baseline rate for these health effects is from miners with cumulative dust levels up to 100 gh/m^3.

A major issue to be addressed in the current analysis is the extent to which overlapping prevalence of the four pulmonary health effects occurs in the same population. That is, the overlap refers to how much of the prevalence for each disease is due to individuals with more than one of the diseases. By adding all four health effects in Table 3 for the 5 mg/m^3 dust and 500 $\mu g/m^3$ SiO_2 exposure level, a 96.4 combined prevalence of these conditions is indicated. While correcting for overlap is important for giving a more realistic estimate of total cases, the estimate of premature mortality will probably not change appreciably since an independent prevalence rate is used for each condition. Assuming the diseases are independent (no synergism or antagonism), an individual with multiple diseases would have a combined prevalence rate equal to the sum of the individual prevalence rates. The British coal miner population currently being analyzed will provide specific information on overlap of pneumoconiosis, CB, and CAO (15).

Another research need is the understanding of the cause of pneumoconiosis. It is important to know what component or components in coal dust cause pneu-

moconiosis and what the exposure is to these components in the oil shale industry. Thus, a better understanding of the cause of pneumoconiosis would facilitate extrapolation to oil shale mining. There are considerable mine-to-mine variations in the risk of pneumoconiosis even within the coal industry (11). The health effects ratios reported in this analysis are the average for all coal mines together. Mineralogical comparisons of coal mine exposures with different reported risks of pneumoconiosis have succeeded in ruling out quartz as a major contributor to this disease at quartz levels below 5% (29). The much greater mineral content in oil shale compared to coal may indicate an increased pneumoconiosis risk in oil shale. However, recent inhalation toxicological studies in Sprague-Dawley rats suggests the fibrogenicity of SiO_2 when contained in oil shale may be one half of that seen with SiO_2 alone (30).

All mechanization in the British coal mining industry has been electric, while the U.S. oil shale mining industry will be dieselized. The modification of any non-neoplastic respiratory effects in the oil shale industry, relative to the coal workers' experience, must be determined. Diesel exhausts have little effect on acute health risks, but long-term effects have not been adequately studied (31).

Finally, it is usually the unanticipated health risks that produce the major concern to a newly developing industry. There has been considerable debate about the potential role of silicosis in causing lung cancer (32). Data may soon accumulate which permit a risk estimate for this health effect. Perhaps the most intriguing source of new data for health effect risk for oil shale will come from the three current studies of the now defunct Scottish industry which include worker morbidity, worker mortality, and community mortality approaches (9).

SUMMARY

The non-neoplastic lung diseases for a future oil shale industry represent a risk of significant concern. At the nuisance dust level, the TLV for free silica may be exceeded by a factor of five, resulting in 97 annual premature fatalities. At this level, the combined annual fatalities due to CB, CAO, and pneumoconiosis is about half this number. However, at the 1 mg/m^3 dust level, corresponding to the 100 $\mu g/m^3$ free silica TLV, there are 13 silicosis deaths, with the dust diseases again at about half that number. Based on these results, the dust exposure within oil shale mines should be a key occupational health concern in future development and design.

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