PUBLIC HEALTH RISKS FROM AN OIL SHALE INDUSTRY

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ABSTRACT

Public health inhalation risks associated with a hypothetical one million barrels-per-day shale oil industry have been estimated for both individual carcinogens and all air pollutants as modeled by a surrogate. The populations at risk which were considered are the local oil shale region and the entire continental United States. The lifetime cancer risks from As, Cd, Cr, Ni, PAH and radiation are less that 10^{-7} occurrences per person per year. The air pollution surrogate, sulfate as a measure of all air pollution exposure, yields a result of 10^{-5} deaths per person per year with large uncertainties. Based on the sulfate surrogate model, the public mortality estimate associated with the refining of imported high sulfur oil which could be replaced by shale oil is significantly larger than the public mortality estimate associated with oil shale production. The dispersion models, health dose-response models, and key uncertainties are discussed. Public health risks associated with oil shale solid waste leachates are considered. Results of a peer review of the analysis and research needs are also presented.

INTRODUCTION

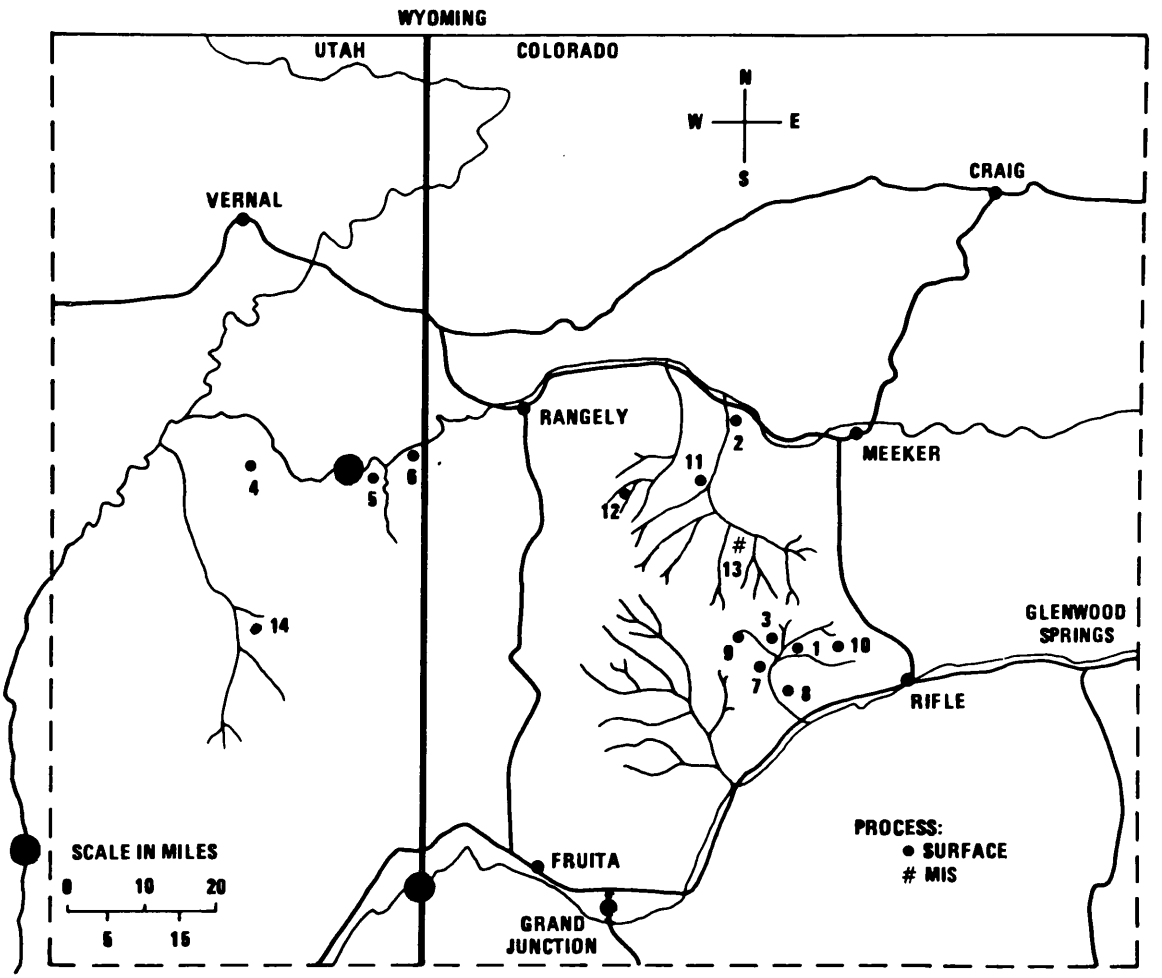
A goal of an oil shale risk analysis is to estimate the potential public health risks associated with a hypothetical one million barrels-per-day (BPD) oil shale industry. The purpose of this analysis is to establish research needs to aid in the formulation and management of a program of environmental research. The results were reported in the Health and Environmental Effects Document (HEED) for oil shale (1) and are not intended for regulatory purposes.

Oil shale airborne emissions and waterborne leachates may increase the risk of health effects in the public populations living in and around the oil shale region. A million barrels-per-day industry will emit a wide variety of air pollutants including the criteria pollutants, trace elements and hydrocarbons. Under the goals of "zero discharge," there should be no direct discharge of oil shale process waters into surface waters. However, oil shale solid waste leachates are a potential environmental problem which may extend for several centuries after final abandonment of facilities. Percolating water from rainfall and snowmelt, or groundwater intrusion into abandoned disposal sites may migrate through spent shale and dissolve a portion of the spent shale matrix. This organic- and mineral-laden water may migrate to underground aquifers and eventually contaminate surface drinking water supplies. Many of these pollutants have been associated with adverse health effects through toxicologic investigation or epidemiologic research.

This paper elaborates and updates the previously presented public health risks associated with a steady-state oil shale production scenario (2). Several risk analysis components are needed to estimate public health risks: a site-specific production scenario, the controlled pollutant source terms, an environmental transport model, the population at risk, health effect dose-response functions. The resulting risk estimates are then put in perspective with appropriate sensitivity analysis and uncertainty analysis. This process is described below for air emissions and spent shale solid waste leachates.

SCENARIO

The risk analysis scenario shown in Figure 1 has fourteen production sites distributed along the major creek systems feeding the Colorado River. This hypothetical scenario occurs in the year 2010. The production level for sites 1, 5, 7, 8, 12, and 13 is 100,000 BPD. All other sites are projected to produce 50,000 BPD. The production is based on underground room-and-pillar mining with above-ground retorting (AGR) for all sites except site 13 which is



Numbered sites refer to million BPD sites.
●represents location of leachate impact calculation points.
Lees Ferry, Arizona not shown.

Figure 1. Location of Leachate Calculation Points and the Oil Shale Risk Analysis Development Scenario Sites

an MIS (modified-in-situ) operation with 50% of the production coming from the MIS.

HEALTH RISKS OF AIR EMISSIONS

Source Terms

Generic emission terms for the basic retorting process types were estimated from prevention of significant deterioration (PSD) permit air emission data. For the three basic above-ground retorting processes, the estimates were based on averaging data from Union Oil Co. (3, 4), TOSCO Development Corp. (5), and Paraho Development Corp., (6). For the MIS process, Cathedral Bluffs Shale Oil Co. (7) was used. Trace element source terms were found by scaling the particulate source terms by the concentration of each element in raw and spent shale from the Colony site (8). The generic source terms are summarized in Table 1.

The scenario included two power plants to support the oil shale industry and the regional population. The power plants were sited at Moon Lake, Utah and Grand Junction, Colorado. The air emissions from each four unit power plant were 10,000 kg/day of sulfur dioxide, 2,640 kg/day particles, and 48,600 kg/day of nitrogen based on the Bonanza Power Plant (9).

Based on the one million BPD scenario and the generic air source terms, the emissions for each site were computed. Modeling the dispersion of these pollutants is the next step in the risk analysis process.

Transport and Transformation

The results of Huang and Sandusky (10) were adapted to estimate public exposure to air pollution within the oil shale region. These results were based on a Gaussian plume distribution with no correction of plume centerline near elevated terrain. A joint frequency distribution of wind speed, wind direction, and stability was obtained from data taken at Tract C-a and was used to construct a long-term average two-dimensional wind field. All of the reported results are for a non-buoyant release, which overpredicts concentrations closer to the source. Air exposure contours were calculated by a computer program which integrated the plumes of the fourteen oil shale sites and the two power plants.

The model of Fay and Rosenzweig (11) was used to estimate public exposure to air pollution across the United States. Fay and Rosenzweig solved a steady state two-dimensional diffusion equation which is a suitable model for predicting ambient air pollutant concentrations averaged over a long time period at distances greater than about 100 km from the source. The model uses a constant horizontal diffusion coefficient to determine horizontal puff dispersion. Pollutants are assumed to be mixed uniformly in the vertical direction up to a constant mixing height. Precipitation rate, dry deposition rate, transformation rate, and wind speed/direction were considered as fixed parameters over the geographic region of interest.

Population at Risk

The oil shale regional population was estimated by adding a baseline population to an "oil shale worker" population. The baseline population was estimated using a growth rate of 3.2% per year (12, 13) on the estimated 1980 population of 152,000 persons. The 33,200 oil shale workers were multiplied by 6.8 to account for other developments, indirect employment, and worker families result in 226,000 persons moving in to the region. Adding the "oil shale worker" population and baseline results in an estimated 616,000 persons at risk for the public regional population. The distribution of the projected population throughout the region was based on historical patterns and studies on the commuting patterns of the oil shale workers. Most of the population will continue to be concentrated along the Colorado River corridor.

The U.S. population was estimated to be 313 million persons in the year 2010. The 1980 population of 226 million was extrapolated 30 years at a growth rate of 11.4% per decade (14).

Health Effects

Estimation of public health risk at low doses requires consideration of the existence of health effects thresholds. The potentially important public health effects can be considered as threshold or non-threshold. A threshold is an exposure level below which there are no health effects in a population. An apparent threshold is an exposure level below which there are no observed health effects. An apparent threshold may be due to the difficulty in detecting very small effects in the population. In this analysis, carcinogenic and air-pollutant surrogate risks were assumed to have no threshold.

Table 1. GENERIC VALUES FOR OIL SHALE AIR EMISSIONS

Process Type	Controlled Emissions (lb per barrel)							Emissions (10^{-6} lb per barrel)								
	SO_2	NO_x	CO	HC	Particulates	H_2S	CO_2	As	Be	Cd	Cr	Pb	Hg	Ni	Se	V
AGRs:																
Union	0.22	0.42	0.16	0.16	0.091	NA	NA	3.9	.077	.099	3.2	2.2	.016	1.9	1.1	4.4
TOSCO	0.13	0.87	0.022	0.18	0.13	0.0012	274	5.0	.069	.12	4.1	3.2	.11	2.2	.69	5.5
Paraho	0.23	0.60	0.090	0.017	0.12	.0013	NA	4.2	.10	.017	3.2	2.5	.022	2.5	.27	8.3
Range–All Sites	0.067 to 0.23	0.42 to 0.92	0.010 to 0.20	0.017 to 0.22	0.087 to 0.15	.0000086 to 0.0013	NA	2.9 to 7.1	.052 to .10	.017 to .099	2.3 to 5.9	1.5 to 26	.0061 to .28	1.2 to 3.1	.27 to 1.1	3.1 to 8.3
Generic AGR:																
Nominal	0.19	0.63	0.091	0.12	0.11	0.0012	274	4.3	.082	.079	3.5	2.6	.049	2.2	.69	6.1
Range	0.063 to 0.23	0.42 to 0.92	0.009 to 0.20	0.012 to 0.24	0.09 to 0.15	.0000086 to 0.0013	NA	2.9 to 7.1	.041 to .10	.016 to .099	2.3 to 5.9	1.4 to 26	.0061 to .22	1.2 to 3.1	.27 to 1.1	3.1 to 8.3
Generic:																
MIS Site	0.38	1.55	0.54	0.002	0.15	0	NA	9.7	NA	NA	5.5	7.0	.14	7.4	.39	.25

NA=Not Available

Note: Trace element source terms are based on particulate source terms and elemental concentrations in raw and spent shale.

All other effects were assumed to have thresholds. A particular pollutant may be associated with multiple effects. In this way, a pollutant could have both a threshold effect and a non-threshold effect.

The pollutants analyzed as having threshold effects were sulfur oxides, particulates, nitrogen oxides, ozone, carbon monoxide, hydrogen sulfide, arsenic, beryllium, cadmium, chromium, fluoride, lead, mercury, nickel, selenium and vanadium.

Sulfur oxides and particulates have been associated with acute and chronic respiratory disease including respiratory tract infection, increased bronchitis and decreased lung function (15). Nitrogen dioxide, a strong oxidant, has been associated with changes in pulmonary function with chronic exposure (16). Ozone can affect pre-existing respiratory disease although the effect of ozone is difficult to separate from other pollutant effects (17). Carbon monoxide chronic exposure has not been determined to cause detrimental effects in humans although there is evidence of behavioral effects of elevated carboxyhemoglobin levels (18). Hydrogen sulfide was considered in this analysis for its nuisance odor potential although it can cause eye and respiratory effects at much higher exposures (19).

The non-carcinogenic effects of arsenic exposure include gastrointestinal involvement, skin changes, peripheral neuropathy and liver damage (20). Chronic beryllium disease, berylliosis, is a systemic intoxication characterized by effects of the respiratory system (21). Chronic exposure to cadmium is associated with two major health effects: decreased lung function and, at lower exposures, renal damage (22). Chromium dusts have caused skin ulcers, dermatitis, allergic responses and the classic chromium-related symptom, perforation of the nasal septum (23). At elevated exposures, fluoride causes acute fluoride poisoning, dental fluorosis, and crippling skeletal fluorosis (24). While the most sensitive adverse effect of lead toxicity is subclinical peripheral toxicity, a range of effects occur at higher levels including anemia, renal damage and encephalopathy (25). Classic mercuralism, caused by inhalation of mercury vapors, is characterized by a variety of neurologic disorders including tremor, lung irritation, and possibly proteinuria as a result of renal damage (26). Nickel carbonyl causes systemic poisoning with acute exposures. Pulmonary effects predominate the clinical course but parenchymal degeneration is observed in the liver, kidneys and other organs (27). Selenium chronic effects are damage to the liver and kidneys (28). Finally, vanadium effects include chronic bronchitis, chronic rhinitis and pharyngitis (29).

The analysis for threshold effects involved addition of the calculated air concentration due to oil shale to a background concentration and comparison of the total to the threshold. The expected number of persons having an exposure above the threshold was calculated. The threshold analysis was only done for the oil shale region. Table 2 presents the thresholds and estimates of pre-development background concentrations used in the analysis.

Table 2. Health Effects Thresholds and Background Concentrations for the Public Health Air Exposure Risk Analysis

Pollutant	Threshold $\mu g/m^3$	Background $\mu g/m^3$
Sulfur Oxides	120	25.0
Particulates	180	50.0
Nitrogen Oxides	100	-
Ozone	100	-
Carbon Monoxide	3300	100.0
Hydrogen Sulfide	50	-
Arsenic	60	0.01
Beryllium	0.01	0.0001
Cadmium	0.4	0.002
Chromium	160	0.01
Fluoride	10	0.05
Lead	5	0.8
Mercury	1	0.02
Nickel	30	0.01
Selenium	17	0.01
Vanadium	10	0.02

Four trace elements have been associated with increased cancer risks: arsenic, cadmium, chromium, and nickel. Cadmium is associated with prostate cancer while the others are related to respiratory cancers. Polycyclic aromatic hydrocarbons (PAHs) are a class of organics which includes several carcinogenic species. Benzo(a)pyrene (BaP) is often used as an indicator of the carcinogenicity of the class. Use of BaP assumes that BaP concentrations are proportional to the carcinogenicity of the entire class. The dose-response relationships are shown in Table 3. The arsenic, cadmium and PAHs (BaP) functions are from the EPA Carcinogen Assessment Group, (30, 31, 32). The chromium and nickel dose-response functions are based on occupational epidemiologic studies

Table 3. Dose-response Relationships for Carcinogenic Health Effects for the Public Health Air Exposure Risk Analysis

Pollutant	Site	Cancers/year/100,000/$\mu g/m^3$
Arsenic	Respiratory	4.14
Cadmium	Prostate	2.9
Chromium	Lung	0.13
Nickel	Respiratory	0.105
PAHs (BaP)	Respiratory	0.124×10^3

(33, 34, 35, 36). Public exposure to radon gas and particulate-borne radioactivity can cause an increased cancer risk. The BEIR III dose-response model (37) of 403 cancer deaths per million person-rads per year was used to estimate this risk. These dose response functions were applied to the estimated oil shale region exposures.

To assess the effect of air pollution below the apparent thresholds, a sulfate health damage function of 3.5 premature deaths per year per 100,000 persons per $\mu g/m^3$ of sulfate was employed. This function is the result of combining several expert opinions (38) including Lave and Seskin (39) who examined the differential mortality rates of 117 Standard Metropolitan Statistical Areas in the United States. This controversial model uses sulfates as a surrogate for all air pollution and has large uncertainties, especially when applied to a new mix of air pollutants. The sulfur surrogate model was applied to both the oil shale region population exposure and to the U.S. population exposure calculated with the Fay and Rosenzweig transport model.

Risks and Uncertainty

The following pollutants were found to have no general population exposures greater than the chosen thresholds for potential health effects previously described: sulfur oxides, particulates, carbon monoxide, hydrogen sulfide, beryllium, fluoride, lead, mercury, selenium, and vanadium.

Table 4 shows the results and uncertainty of the public health risk analysis. The largest individual pollutant cancer risk was due to arsenic at 1.01×10^{-3} cancers per year in a population of 616,000 persons. The uncertainty of the source term, the exposure transport models, and the health effect functions were determined independently. They were then combined by a root-mean-square approach in the log domain to yield the uncertainty factors.

The risk estimates due to air pollution using sulfates as a surrogate were 15 premature deaths per year (with a range of 0-76 deaths) in the oil shale region population of 616,000 persons and 12 premature deaths per year (with a range of 0-220 deaths) for the U.S. population of 313 million persons in the year 2010. The total risk estimate was 27 premature deaths per year with a range of 0-296. Results of a sensitivity analysis were used in the determination of the uncertainty factors.

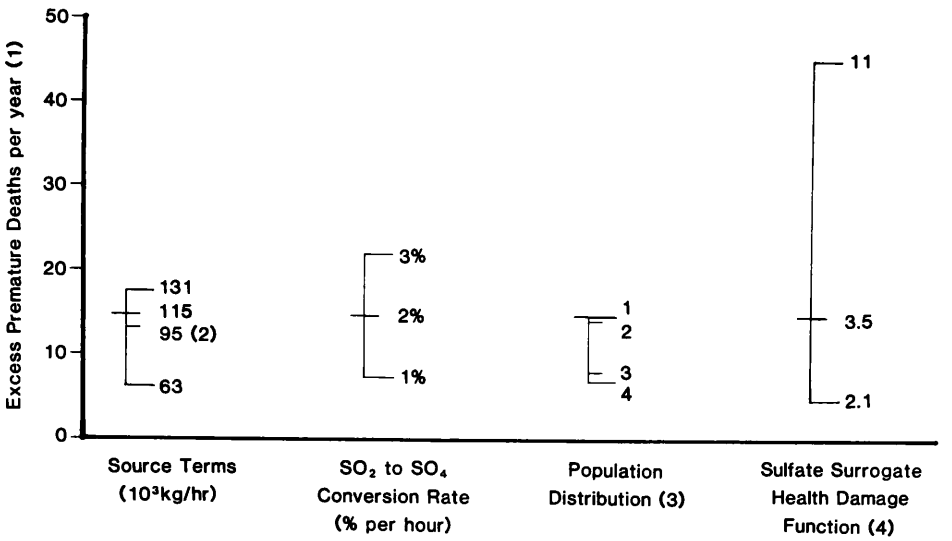
A sensitivity analysis was performed on the sulfate surrogate model by independently varying different parameters of the model across their reported ranges. The resulting sensitivities are shown in Figures 2 and 3. In the oil shale region, the dominating parameter is the sulfate health damage function. For long distance transport, the wet deposition rate constant is the most sensitive parameter followed by the sulfate health function and the dry deposition velocity. The results are less sensitive to changes in conversion rate and source strength.

Tradeoff Analysis

As shale oil is produced, an equivalent amount of imported petroleum could be replaced in U.S. refineries. Despite the uncertainty and controversy regarding the sulfate surrogate model, it is useful to compare the health damage due to oil shale development with the health benefit caused by reduction of petroleum refinery emissions in the densely populated midwest and northeast regions of the United States.

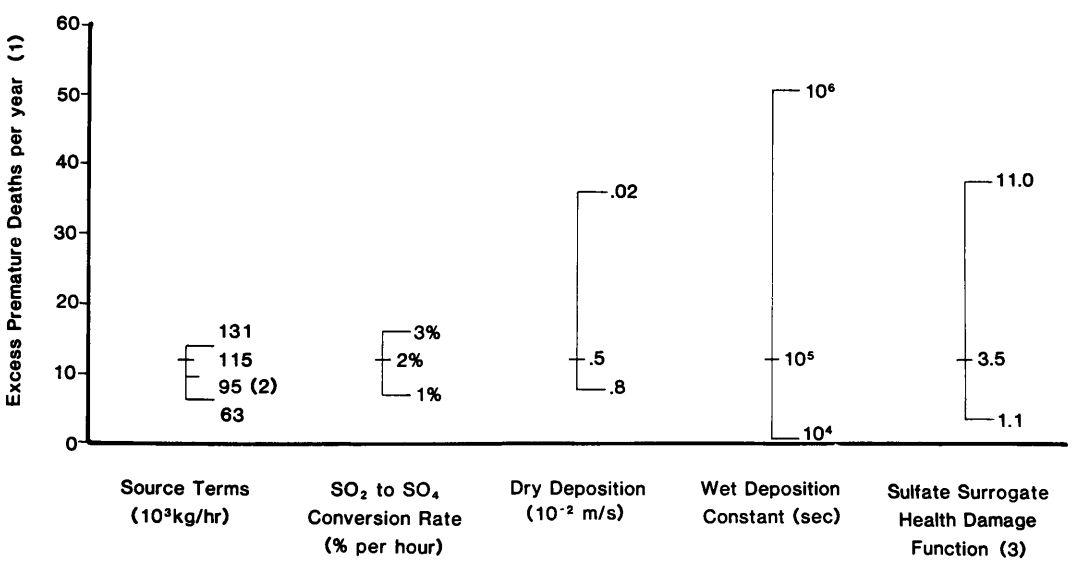
The emission from the refining of one million barrels of oil shale syncrude is estimated to be less than 0.54 metric tons (M.T.) of sulfur dioxide per day assuming 20 ppm sulfur by weight in syncrude (40) and 10% emission of syncrude sulfur (41). The emission for the refining of one million barrels of petroleum is 168 M.T. of sulfur dioxide per day (42, 43). Therefore, the replacement can decrease refinery sulfur dioxide emissions about 167 M.T./day.

The Fay and Rosenzweig (11) long-range transport model was used to calculate air exposure contours for this emission assuming it is to be refined in Illinois (30%), Pennsylvania (20%), New Jersey (18%), Ohio (16%), and Indiana (16%). The result was 600 premature deaths per year for the U.S. population. The oil shale production, from extraction to trans-



(1) Population at risk is the region population in the year 2010, 616,000 persons
(2) Without power plants
(3) 1-best estimate, 2-low density omitted, 3-concentration in corridor, 4-northern concentration
(4) Units are premature deaths per year per ug/m³ per 100,000 persons

Figure 2. Sensitivity Analysis for Oil Shale Region Transport of Oil Shale Industry Air Emissions



(1) Population at risk is the U.S. population in the year 2010, 313 million persons
(2) Without power plants
(3) Units are premature deaths per year per ug/m³ per 100,000 persons

Figure 3. Summary of Risk Results and Sensitivity Analysis for Long-range Transport of Oil Shale Industry Air Emissions Based on the Fay and Rosenzweig Model

Table 4. Public Health Risks Due to Airborne Oil Shale Industry Emissions and Associated Uncertainty

Pollutant	Site	Uncertainty Factors [1] U_S	U_E	U_H	U_R[2]	Cases per Year [3]	Uncertainty Range
Arsenic	Respiratory	1.4	2.1	2.2	3.1	1.01×10^{-3}	$0 - 3.13 \times 10^{-3}$
Cadmium	Prostate	1.3	2.2	2.6	3.5	5.55×10^{-6}	$0 - 1.95 \times 10^{-5}$
Chromium	Lung	1.3	2.2	3.16	4.1	2.33×10^{-5}	$0 - 9.55 \times 10^{-5}$
Nickel	Respiratory	1.3	2.2	3.16	4.1	1.26×10^{-5}	$0 - 5.17 \times 10^{-5}$
PAH (BaP)	Respiratory	1.9	2.4	10.00	12.7	2.49×10^{-5}	$0 - 3.16 \times 10^{-4}$
Radiation	Respiratory	1.4	2.1	2.2	3.1	2.50×10^{-3}	$0 - 7.75 \times 10^{-3}$

Transport Distance	Uncertainty Factors [4] U_S	U_C	U_{DD}	U_{WD}	U_P	U_H	U_R[5]	Premature Deaths Per Year	Uncertainty Range
Regional	2.2	2.0	---	----	1.8	3.1	5.3	14.5	0- 76
Long Distance	1.8	1.7	3.0	10.0	---	3.1	18.3	12.0	0-220
Total								26.5	0-296

[1] S-Source Terms; E-Exposure Model; H-Health Dose-response; R-Risk Estimate

[2] Computations based on $\log U_R = \sqrt{(\log U_S)^2 + (\log U_E)^2 + (\log U_H)^2}$

[3] Population at risk is 616,000 persons for all cancers except prostate for which it is half this number.

[4] S-Source Term; C-Sulfur Oxide to Sulfate Conversion Rate; DD-Dry Deposition; WD-Wet Deposition; P-Population Distribution; H-Health Dose-response Function; R-Risk Measure.

[5] Computations based on $\log U_R =$

$$\sqrt{(\log U_S)^2 + (\log U_C)^2 + (\log U_{DD})^2 + (\log U_{WD})^2 + (\log U_P)^2 + (\log U_H)^2}$$

portation of the syncrude, was found to have a sulfur oxide emission best estimate of 115 M.T./day which would cause 27 premature deaths per year. Therefore, this substitution analysis, while based on a controversial model, shows a significant reduction in premature deaths as a result of oil shale production. Another serious difficulty is that the sulfate surrogate model predicts premature deaths due to all air pollution, not just sulfates. Therefore, the result is only as valid as the surrogate model and the assumption that other pollutants follow the same substitution.

SOLID WASTE LEACHATES

Surface water quality changes

Surface water quality changes from solid waste leachates are difficult to estimate because of the uncertainties in operator water treatment, water and waste disposal practices, and abandonment requirements. Limited data from current research were used to perform a crude calculation of ground and surface water quality changes due to leachates percolating through spent shale piles generated from a one million barrels-per-day oil shale industry. A series of simplifying assumptions, listed in the HEED (1), were necessary to proceed with the analysis which should be considered an extreme case.

The spent shale piles were assumed to be generated over a 30-year operating life. Surface processes were estimated to generate 1.4 tons spent shale/bbl-oil and MIS processes were estimated to generate 2.3 tons spent shale/bbl-oil. MIS processes were assumed to result in 0.4 ton spent shale/bbl-oil disposed above ground and 1.9 ton spent shale/bbl-oil disposed in the abandoned retorts. Leachate migration from abandoned MIS retorts was not included in the analysis.

The estimated production in the drainage systems of the Parachute Creek, Piceance Creek, White River, and Green River was 450,000 BPD, 300,000 BPD, 150,000

BPD, and 100,000 BPD, respectively. Spent shale volume was distributed over the oil shale region based on these production rates Leachate concentrations for Paraho and TOSCO retorted shale were averaged to obtain the values used in the analysis. A water flux of 0 to 8 cm/year (44) and the pile surface area were combined to obtain the volume of leachate entering each drainage which was assumed to be flowing at the minimum flow rate on record (45). The diluted leachate concentration was found for each downstream river segment.

Estimated increases in concentrations at the White River at Watson, Utah, Green River at Green River, Utah, Colorado River at the Colorado-Utah state line, and the Colorado River at Lees Ferry, Arizona (locations shown in Figure 1) are presented in Table 5.

Table 5. Upper Bound Increases in Concentration of Selected Leachate Trace Elements at Selected Locations in the Colorado River System (mg/l)

Trace Element	White River (Watson, UT)	Green River (Green River, UT)	Colorado River (Co-Ut line)	Colorado River (Lees Ferry, AZ)
F	0.11	0.031	0.018	0.032
Na	41.	11.	6.8	12.
As	2.5×10^{-4}	4.6×10^{-5}	2.7×10^{-5}	4.7×10^{-5}
Se	2.9×10^{-4}	8.0×10^{-5}	4.8×10^{-5}	8.3×10^{-5}

Health Effects

Estimating public exposure to leachates is complicated by the transport mechanisms, water treatment, ingestion patterns, contributions from other pathways, and health response variability. The potential health risk from increases in concentration of pollutants in drinking water is based on two key assumptions: two liters of water were assumed to be ingested per day and the increased pollutant concentrations were added to average ingestion levels for pollutants from all sources.

The health effects associated with ingestion of arsenic, fluoride, selenium, and sodium were considered. Ingestion of arsenic compounds has been associated with increased risk of skin cancer (46). The dose-response relationship of 0.59 skin cancers per year per 100,000 per milligram of arsenic was used. This relationship is considered very conservative due to the problem of extrapolation from a very high dose to a low dose. Fluoride in drinking water is protective against dental caries at low concentrations but can cause dental mottling with intake as low as 4 mg/day. At very high concentrations, fluoride can cause crippling skeletal fluorosis. A conservative threshold of 20 mg/day is used in the analysis for this effect. A background estimate of 2 mg/day was assumed (47). Selenium may be an essential element although chronic health effects are seen at high doses. Sakurai and Tsuchiya (48) recommended a maximum daily intake of 500 μg/day. It was assumed that as much as 100 μg/day was from inhalation, resulting in a conservative threshold for daily ingestion of 400 μg/day. Background concentrations were estimated at 100 μg/day for food (48) and 6 μg/day from water (28). Drinking water sodium has been shown to be a contributor to hypertension in healthy populations (49). Persons on sodium-restricted diets (3% of population) have been shown to have adverse health effects with high sodium drinking water (50). The NAS (47b) recommended sodium maximum intake of 200 mg/day was used in the analysis with a background intake of 150 mg/day.

Risks

Based on the leachate analysis, only the population receiving water from the Watson, Utah area (the maximum of the extreme analysis) may be exposed to sodium concentration 16% above the NAS recommendation. Selenium and fluoride thresholds are not exceeded for any segment of the population. The analysis assumed no water treatment. Considering the extreme nature of the assumptions, such as minimum recorded flow rate, the risks due to water exposure are felt to be minimal for the pollutants analyzed.

Individuals in Watson, Utah drinking two liters of untreated water per day (with an elevated arsenic concentration of 2.5×10^{-4} mg/liter) may have an increased individual risk of excess skin cancer of 1.5×10^{-9} per year.

DISCUSSION

The estimate of premature deaths due to air pollution (as found with the controversial sulfate surrogate model) is the predominating result of the public health risk analysis. The total public cancer risk is over three orders of magnitude less than the sulfate surrogate premature death risk. No part of

the population was exposed to pollution concentrations over the threshold for the other effects analyzed.

Since these results were first presented in the HEED, the National Research Council has reviewed the analysis (51). While the review was generally favorable, several important criticisms were made. The criticisms, along with possible alternatives and corrective action, provide a useful perspective on the public health risk analysis.

The NRC Committee was quick to remark that "there is no agreement in the scientific community on whether sulfates have adverse health impacts at current ambient concentrations. Whereas some analysis has suggested that sulfate concentration is correlated with increased mortality, no direct evidence implicates sulfate or other sulfur oxides as the causal agent." Further research is needed to resolve this issue. However, alternative methods can be applied to provide another risk estimate as a surrogate for all air pollution. For example, the 1983 Particulate Matter HEED (52) recommends the use of fine particles (d< 2.5 um) as a surrogate. Alternative approaches are being researched for the current analysis.

The use of a Gaussian plume diffusion model for the exposure assessment was considered inappropriate by the committee primarily because it fails to account for the complex terrain and strong channeling effects within the canyons. The residents of the towns at the mouth of Parachute Creek could receive exposure much greater than predicted by the Gaussian plume model. Conclusions of a DOE-sponsored experts' workshop on dispersion modeling stated, "Modeling of dispersion in complex terrain typically involves simple adjustments or modifications to steady-state, flat-terrain modeling approaches." In the current effort, the impact of channeling effects will be considered in this manner.

Concerning the water analysis, the committee took exception with the concept of zero discharge "since true zero-discharge is rarely achieved." Accidental release of holding pond water could be addressed by studying the probability and magnitude of leaks.

Another omission noted in the analysis was the health effects of waterborne organics. A large variety of organic compounds are known to be present in retorted shales and waters. However, the uncertainty of environmental transformation and removal of these organics was too great to allow an analysis comparable to the trace element analysis. Furthermore, the health effect dose-response is unknown for complex organic mixtures in water. Research is necessary on both aspects of organic water pollution.

Significant research efforts are underway to study the health of the public around the now defunct Scottish oil shale industry. The results of this research may provide substantial insight and data for estimation of public health risks which have not yet been considered in past risk analysis efforts. As production begins at the first commercial U.S. oil shale plant, source term data and operation procedures should be reviewed for appropriate revision of the public health risk analysis.

These issues and others will be addressed with the continuation of the oil shale risk analysis process.

SUMMARY

The analysis of public health risks from a hypothetical one million barrels-per-day steady state oil shale industry indicates that air emissions present the predominant risk. A tradeoff analysis suggests that replacement of high sulfur imported oil can provide an overall health benefit for the U.S. population. Analysis of surface water quality changes from solid waste leachates show minimal public health risk for those pollutants considered, although further analysis is needed on waterborne organics. The results to date do not indicate the oil shale industry will pose any significant risks to the public beyond those of other fossil fuel energy cycles. The large uncertainties in various components of the analysis serve as a guide to research needs for the reduction of risk uncertainties. Data from the study of past, present, and future oil shale plants will be used in future iterations of this analysis process.

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