An Alternate Characterization of Hazard in Occupational Epidemiology: Years of Life Lost Per Years Worked

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Background *Standardized mortality ratios (SMRs) and other measures of relative risk by themselves may not suffice as descriptors of occupational hazards for many audiences including decision-makers and those at direct risk from hazardous work. To explore other approaches, we calculated excess years of potential life lost and excess lifetime risk for both lung diseases and fatal injuries in a cohort of uranium miners with historical records of exposure to radon gas.*

Methods *We used relatively simple life table (SMR) methods and also analyzed lung cancer mortality with Poisson regression methods permitting control for smoking.*

Results *Among uranium miners hired after 1950, whose all-cause SMR was 1.5, 28 percent would experience premature death from lung diseases or injury in a lifetime of uranium mining. On average, each miner lost 1.5 yr of potential life due to mining-related lung cancer, or almost 3 months of life for each year employed in uranium mining. As a consequence of all excess lung disease and injury risks combined, a year of mining was associated with 5.9 months loss of potential life. For each year actually working underground, miners lost more than 8 months of potential life. When controlled for smoking (and healthy worker effect) with Poisson regression, the estimates for radon-related lung cancer effects were slightly larger. Although chronic disease deaths dominated in excess years of life lost (due to radon, silica and possibly other exposures), more years were lost on average per individual injury death (38 yr), than per excess lung cancer (20 yr) or other lung disease death (18 yr). Fatal-injury dominated the potential years of life lost up to about age 40.*

Conclusions *Years of life lost per years employed provides another, more intuitive summary of occupational mortality risk.* Am. J. Ind. Med. 42:1–10, 2002.
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KEY WORDS: attributable risk; excess lifetime risk; fatal mining injury; radon; silicosis; uranium mining; worker notification; YPLL

INTRODUCTION

Epidemiologists generally present findings using measures of relative risk or rates such as the standardized mortality ratio (SMR), rate ratio, odds ratio (OR) or occasionally the attributable risk fraction. These measures have proven to be powerful tools for identifying etiologic relationships [MacMahon and Pugh, 1970; Rothman, 1986], but may not be sufficient for conveying the public health or individual significance of study results. This is because

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relative risk depends on both the strength of the association and the background rate or risk [Peacock, 1971]. Thus, relative risk measures by themselves may be unintuitive and not very useful in communicating risk. Excess lifetime risk [BEIR, 1988] or years of potential life lost [Haenszel, 1950; Gardner and Sanborn, 1990] are alternative characterizations of risk that may be more interpretable by the lay public and decision makers. In this investigation, we present several characterizations of risk derived from both elementary life table and regression modeling approaches as applied to the mortality experience of a cohort of uranium miners. We then discuss their relative utility in summarizing risk.

MATERIALS AND METHODS

Uranium Miners Cohort

The Colorado Plateau Uranium Miners Cohort was assembled by the U.S. Public Health Service (U.S. PHS) based on medical examinations administered between 1950 and 1960 in response to health concerns in the growing nuclear industry [Lundin et al., 1971]. Subsequent annual surveys by the U.S. PHS into the early 1970s collected occupational, medical, and smoking histories. Most of these uranium mines were closed by 1970. The National Institute for Occupational Safety and Health (NIOSH) within the Centers for Disease Control and Prevention, has maintained this database [Roscoe, 1997], updating smoking status in 1984. The detailed smoking history available for each miner consists of a sequence of time periods and corresponding cigarette consumption levels. Several studies have been published from this cohort focusing on groups of special interest such as Native American miners [Roscoe et al., 1995] and nonsmokers [Roscoe et al., 1989], and there are recently updated analyses for the white miners [Roscoe, 1997; Hornung et al., 1998; Langholz et al., 1999]. Among the 3,347 white men hired before 1964 and followed 1950–1990, there were 1,698 deaths including 378 from lung cancer. Based on U.S. reference rates, this lung cancer represented a four-fold excess (SMR = 4.2; 95% CI = 3.8–4.6).

Because followup began with a miner's first medical exam (sometime during or after 1950), we focused on those hired on or after January 1, 1950 in order to observe most mining injury deaths including those early in employment. We wanted to be able to compare injury deaths with later excess chronic disease deaths. In the cohort of 2,721 white men hired during or after 1950, there were 1,271 deaths and 267 lung cancer deaths; about 18% of these miners had never smoked and, at the end of followup, 33 percent were ex-smokers. The average radon exposure intensity for the miners studied was 8.0 "working levels" (WL; a standard measure of radon or other airborne radioactivity) [Lundin et al., 1971; Roscoe, 1997]. The median radon cumulative exposure was 333 working-level months (WLM) in a right-skewed distribution for which the maximum was 10,000 WLM. In characterizing risk among these uranium miners, we focused on: (1) lung cancer (International Classification of Diseases-9th Revision (ICD-9): 162), (2) "other-accidents" (hereafter "other-fatal injuries"), thought to encompass most non-transportation work-related fatalities (ICD-9: E890–928; E929.4–929.9), and (3) all lung disease other than cancer (LDOC; ICD–9: 460–519; also known as "nonmalignant respiratory disease").

SMR-Based Calculations

The basic data configuration used to construct many of the risk measures consisted of observed (O) and expected (E) deaths together with years of life lost per death (life expectancy, YL) arrayed across 15 age strata. We constructed two traditional summary measures for cause of death, g, in uranium mining: the *standardized mortality ratio*, $SMR_g = O_g/E_g$, and the *attributable risk fraction*, $ARF_g = (O_g - E_g)/O_g = (SMR_g - 1)/SMR_g$ (Table I). In addition, we calculated summaries based upon *years of potential life lost* [Haenszel, 1950; Gardner and Sanborn, 1990] and *lifetime risk* [BEIR, 1988]. Years of potential life lost due to cause g is defined as $YPLL_g = \Sigma_i\, O_{gi}\, YL_i$ where, for the i^{th} age stratum, O_{gi} is the observed number of deaths from cause g, YL_i is the life expectancy for individuals in the i^{th} age stratum, and the summation ranges over 15 age strata (i.e., 15–19, 20–24, 25–29, ..., 85 +). The *excess* YPLL for each cause of death studied was constructed by multiplying in each age stratum the years lost per death by the excess number of deaths (observed minus expected), and then summing across age strata (Table I). To place this excess on a scale that would be more meaningful, we created the measure *excess YPLL per year of employment* by dividing the excess YPLL in the study population by the total number of years worked in uranium mines (person-years employed). As a final YPLL endpoint, we considered the *excess YPLL per excess case*, which provides the expected loss of life (yrs) resulting from a work-attributable death due to a particular cause.

Expected deaths and SMRs were calculated for each age group using the NIOSH Life table Analysis System [Steenland et al., 1998]. This analysis system classifies followup experience on gender and race and in 5-yr intervals of calendar time and age and applies the appropriate U.S. cause-specific mortality rates. Subsequent calculations for years of life lost were performed within age strata collapsing over calendar time because the life-expectancy information was not calendar-time specific. For the mid-point of each 5-yr age interval, the years of potential life lost per death were calculated by linear interpolation between life

TABLE I. Candidate Measures for Characterizing Population Risk

Risk measure[a]	Formulaic representation	Units
Standardized mortality ratio	$SMR_g = [\Sigma_i O_{gi}]/\Sigma_i E_{gi}]$	Unitless
Attributable risk fraction	$ARF_g = (SMR_g - 1)/SMR_g$	Unitless
YPLL due to cause of death, g[b]	$YPLL_g = \Sigma (O_{gi}) YL_i$	Years
Excess YPLL due to cause of death, g	$e\text{-}YPLL_g = \Sigma (O_{gi} - E_{gi}) YL_i$	Years
Attributable YPLL fraction	$e\text{-}YPLL_g/YPLL_g$	Unitless
Excess YPLL per excess death, g	$e\text{-}YPLL_g/[\Sigma_i (O_{gi} - E_{gi})]$	Years
Excess YPLL per year employed	$e\text{-}YPLL_g$/(total yrs employment)	Unitless
Lifetime risk	$LTR = \Sigma_i \{[R_{gi}/R_{+i}] S(1,i) [q_i]\}$[c]	Probability
Excess lifetime risk	LTR (exposed) − LTR (unexposed)	Probability

[a]Cause of death (g): lung cancer (g = 1); fatal injury (g = 2); lung disease other than cancer (g = 3).
[b]YPLL: years of potential life lost, where YL_i is life expectancy at i^{th} age category.
[c]R_{+i} = all cause age-specific mortality rate; q_i = Pr (death in interval i given alive at the start of interval i); $S(1,i) = (1 - q_1) \times (1 - q_2) \times \cdots \times (1 - q_{i-1})$.

expectancies for white men at ages 25, 45, 65, and 85 that were derived elsewhere [Gilbert et al., 1998].

The estimate of *excess lifetime risk of death* [BEIR, 1988] was based on two sets of age-specific rates, one for mortality from the cause of interest (R_{gi}) and the other for mortality from all causes. The all-causes rates (adjusted to account for excesses in the cause of interest due to uranium mining exposures) are used to calculate the probability of surviving to age i [S(1,i) in Table I], the probability of dying during the i^{th} interval given survival to age i (q_i) and the probability of dying from the specific cause of interest in the i^{th} interval. This leads to the lifetime risk estimate which accounts for competing causes of death: $\Sigma_i \{[R_{gi}/R_{+i}] S(1,i) [q_i]\}$. By calculating lifetime risk up to age 85 using age-specific rates derived from the SMR analysis for the uranium miner population and then subtracting the corresponding quantity using U.S. expected rates, we derived the excess lifetime risk attributable to being a uranium miner.

Poisson Regression Analysis

We used Poisson regression analysis in order to describe the exposure response for both radon and smoking but also to estimate the background lung cancer rate (in nonsmokers unexposed to radon) for the purpose of deriving excess deaths. Age- and calendar time-specific U.S. rates for lung cancer mortality [Monson, 1997] were incorporated as a multiplier of person-years in the Poisson regression model which produced models of rate *ratios* where the intercept was an estimate of the standardized rate ratio (or SMR) for the unexposed population. This approach was particularly appropriate when examining age-effects because external population rates are available for young ages where observed study deaths were sparse. Analyzing smoking history was important due to possible exposure confounding and also because previous investigators had observed a strong radon-smoking interaction. But because the uranium miners had prior exposure to radon during most time under observation the background estimate (from the model intercept) was potentially unreliable. The approach of including U.S. rates permitted placing a requirement on final models that the attained background rate ratios be plausible for an industrial population without radon or smoking exposures. Plausible background rates were derived using a lung cancer SMR estimate from cohorts with minimal exposure to industrial lung carcinogens (0.9 [Park et al., 1991]) and using an estimate of the smoking-attributable fraction for lung cancer in the U.S. male population (0.86, State of Kentucky [Finger and Schultz, 1990]). With the SMR among nonsmokers in the U.S. population expected to be approximately $0.14 = 1.0 - 0.86$, a plausible expected SMR for the unexposed, nonsmoking uranium miners would be about $0.14 \times 0.9 = 0.126$, corresponding to a regression model intercept of $\ln (0.126) = -2.07$.

Analyses of lung cancer mortality in the full Colorado uranium miners cohort by Hornung et al. [1998], Langholz et al. [1999], and Luebeck et al. [1999] have revealed a complex radon exposure-response. It depended on age, smoking, and level of radon exposure intensity, or dose rate, in addition to cumulative dose, and apparently declines after cessation of exposure. In our analysis, we choose the following linear relative rate model which incorporated the previously observed age-dependence of the radon effect (in three cumulative exposure strata) as well as a smoking interaction with the age-specific radon cumulative exposures: $RR = [\exp(a_0 + a_1 (Age - 50)/5)] \times [1 + b_0 CumSmoking + b_1 CumRadon(Age < 60) + b_2 CumRadon(Age\ 60-69) + b_3 CumRadon(Age > 69) + b_4 CumRadon(Age < 60) CumSmoking + b_5 CumRadon(Age\ 60-69) \times Cum Smoking + b_6 CumRadon(Age > 69) \times CumSmoking]$

where the cumulative radon and smoking measures pertained to specific age-intervals of observation. Models were fit using the AMFIT module of Epicure [Preston et al., 1993]. Loglinear models of the form $RR = \exp(b_0 \times \exp(b_1X_1 + b_2X_2 + b_3X_3 + \cdots)$ fit less well than the linear relative rate model. Exposures in this model were continuous variables. Latency in the calculation of cumulative exposure was examined several ways using latency-weighting functions, but a traditional lagging by 5 yr, as in previous analyses [Hornung et al., 1998], performed about as well as any. Smoking exposures was similarly lagged.

Classification tables were generated for Poisson regression analysis using a Fortran program previously developed. Age was classified in 16 strata (starting at 15, in 5 yr intervals) and calendar time in 9 (starting at 1950 in 5 yr intervals). This program classified the exposure measure of interest in 50 levels, and the smoking cumulative exposure in 10 levels. The continuous exposure measures used in models were the person-years weighted stratum means generated while creating the classification table. Previous work had demonstrated that fine stratification of the exposure variable considerably improved fit, especially in the low dose range which has considerable influence in the intercept estimate [Rice et al., 2001]. The unit of followup was one month, which was the minimum interval specified in the work history.

Hornung et al. [1998], Langholz et al. [1999], and Luebeck et al. [1999] observed enhanced risk with low exposure intensity levels. The specification of parameters for average level is problematic because the prior time course may be important but is lost in averaging. We chose to examine whether there was an important nonlinear short-term dose response for radon-induced damage by using a cumulative measure based on the *square root* of current exposure ($WL^{0.5}$), thereby giving lower exposure levels more relative weight than in the usual time-integration of exposure level.

The importance of time-since-last-exposed was examined and an indicator of recent termination was found to be a strong, positive predictor. However, interpretation was complicated by the possibility that workers had terminated employment because of disease, particularly lung cancer. Therefore, these terms were not included in the final model.

The independent age effect estimated with this model (in the first product term) allowed for departure of the background rate away from the external reference rate as a (linear) function of age. This was intended to address two issues: (a) that the radon effects are being estimated relative to a background rate which is age dependent (effects of the same cumulative exposure occurring later in life otherwise would be compared to a higher expected rate), and (b) the healthy worker survivor effect [Arrighi and Hertz-Picciotto, 1994]. The age stratification of cumulative exposures allowed for the possibility that the biological effects of radiation are inherently age-dependent.

RESULTS

Simple Life Table Analysis

The mortality experience of white uranium miners hired after 1950 reveals a 50 percent excess for all-causes (SMR = 1.51; 95% CI = 1.44–1.58) and a more than doubling of cancer mortality (SMR = 2.06; 95% CI = 1.90–2.24) (Table II). There were highly elevated excesses for: (1) Lung cancer (SMR = 267/70.0 = 3.8; 95% CI = 3.4–4.2), (2) Tuberculosis (SMR = 12/2.6 = 4.53; 95% CI = 2.6–7.3), (3) Lung disease other than cancer, LDOC, (SMR = 163/55.7 = 2.9; 95% CI = 2.6–3.3) consisting largely of emphysema (n = 41) and pneumoconioses and "other-respiratory" disease (n = 91 which, since 1960, included 20 silicosis deaths), and (4) Accidental death (SMR = 153/49.9 = 3.1; 95% CI = 2.7–3.5) for which other-fatal injuries ("other accidents") contributed the largest group (n = 81, SMR = 5.87, 95% CI = 4.8–7.1).

The age-specific lung cancer mortality experience is displayed in Table III including age-specific SMRs and YPLLs. Although the highest relative risks for lung cancer death occurred in the younger workers, as others have observed [Hornung et al., 1998; Langholz et al., 1999], particularly in the 40–44 yr age group (SMR = 9/1.7 = 5.5), the largest excess of lung cancer deaths occurred in the age range 55–59: about 44 deaths. In this cohort of 2,721 white miners, lung cancer resulted in the loss of 5,300 yrs of potential life of which 4,007 yrs, or 76 percent, were excess, presumably attributable to mining exposures (Tables III and IV).

For other-fatal injuries, risks were even more concentrated among young workers with the 25–29 yr group having the greatest risk (SMR = 10/0.77 = 13.0) (data not shown). At those ages, 92 percent of lost years of life due to these injuries appeared to be work-related. Overall, injuries associated with mining hazards resulted in 2,534 excess years of life lost (Table IV). For lung diseases other than cancer, the age group with the highest risk was 50–54 corresponding to a four-fold risk (SMR = 17/4.0 = 4.27) (data not shown). LDOC accounted for 2,796 yrs of life lost of which 1,890 yr, or 68 percent, were in excess of expected, based on national comparison rates (Table IV).

The SMRs and attributable risk fractions were highest for other-fatal injuries followed by lung cancer and LDOC (Table IV). The attributable YPLL fraction associated with mining was also highest for other-fatal injuries (86%) followed by lung cancer (76%) and LDOC (68%) and all were similar but slightly larger than the respective attributable risk fractions. The excess YPLLs per cohort member related to mining were 1.5 yr for lung cancer, 0.93 for other-fatal injuries and 0.70 for LDOC, or a total of 3.1 yr. The average years of potential life lost for the victims (excess cases) were 38 yr for other-fatal injuries, 20 yr for lung

TABLE II. Colorado Plateau Uranium Miners 1950 Cohort: Mortality Experience in White Men Hired After 1950 (n = 2721) as Standardized Mortality Ratios (SMR)

	Obs	Exp[b]	SMR	95% CI
All deaths[a]	1,271	842.6	1.51	1.44–1.58
All cancers	405	196.2	2.06	1.90–2.24
Cancer of trachea, bronchus and lung	267	70.0	3.82	3.44–4.22
Leukemia	13	7.3	1.79	1.06–2.85
Tuberculosis	12	2.6	4.53	2.61–7.34
Alcoholism	10	3.4	2.95	1.60–5.01
Ischemic heart disease	220	280.9	0.78	0.70–0.88
Diseases of the respiratory system	163	55.7	2.93	2.56–3.33
Emphysema	41	10.7	3.83	2.90–4.97
Pneumoconioses, other resp. dis.	91	23.0	3.96	3.30–4.71
Silicosis (1960–90 only)	20	0.19	105.5	69.9–153.3
Accidents	153	49.9	3.06	2.67–3.50
Transportation accidents	49	27.5	1.78	1.38–2.26
Accidental falls	15	5.4	2.78	1.71–4.28
Other accidents	81	13.8	5.87	4.84–7.06
Suicide	36	18.9	1.90	1.41–2.51

[a]Cohort: all white men hired during 1950–1963 and followed from first U.S. PHS examination until 1990.
[b]Based on U.S. rates for white men, using NIOSH Life Table Analysis System [Steenland et al., 1998].

cancer, and, 18 yr for LDOC, reflecting the differing age-dependencies of risk. In this cohort, with an average duration of employment in uranium mining of 6.31 yr, for every year worked miners lost on average 0.23 yr (almost 3 months or 85 days) of life because of mining-related lung cancer, 0.15 yr (1.8 months or 54 days) from other-fatal injuries, and 0.11 yr (1.3 months or 40 days) from mining-related LDOC (Table IV). Thus, a miner on average lost 5.9 months of life for every year worked in uranium mining, considering only excess deaths from lung cancer, LDOC

TABLE III. Colorado Plateau Uranium Miners 1950 Cohort: Data Used to Construct Various Risk Measures for Lung Cancer

Age	Person years (PY)	Potential yrs lost[a] (YL)	Observed cases (O)	Expected[b] cases (E)	SMR	Observed YPLL[c]	Expected YPLL	Excess YPLL
15–19	238	57.2	0	0.00	0.00	0.00	0.01	−0.01
20–24	2,232	52.4	0	0.00	0.00	0.00	0.24	−0.24
25–29	4,677	47.7	0	0.02	0.00	0.00	1.11	−1.11
30–34	6,593	43.1	1	0.13	7.61	43.10	5.66	37.43
35–39	8,076	38.5	2	0.53	3.80	77.04	20.25	56.79
40–44	9,413	33.9	9	1.65	5.46	305.51	55.91	249.59
45–49	10,472	29.6	23	4.28	5.38	681.26	126.71	554.55
50–54	10,093	25.7	45	8.46	5.32	1155.15	217.06	938.09
55–59	8,051	21.7	56	12.18	4.60	1216.32	264.48	951.84
60–64	5,630	17.8	37	13.70	2.70	657.49	243.38	414.11
65–69	3,688	14.5	33	12.49	2.64	477.18	180.63	296.55
70–74	2,153	12.1	43	9.41	4.57	520.73	114.00	406.73
75–79	989	9.8	14	5.00	2.80	136.64	48.84	87.80
80–84	327	7.4	4	1.72	2.32	29.64	12.77	16.87
85 +	86	5.1	0	0.41	0.00	0.00	2.05	−2.05
Total	72,727	na	267	69.98	3.82	5300.05	1293.11	4006.95

[a]Life-expectancy at mid-point of age interval, based upon Gilbert et al. [1998]; na = not applicable.
[b]Based upon U.S. rates 1960–1994 for lung cancer, classified on age and calendar time.
[c]YPLL, years of potential life lost.

TABLE IV. Colorado Plateau Uranium Miners 1950 Cohort: Summary of Risk Measures for Lung Cancer, Other-Fatal Injury and Lung Disease Other Than Cancer

		SMR analyses				Poisson regression[c]
Risk metric		**Lung cancer**	**Other-fatal injury**	**LDOC[b]**	**Total**	**Lung cancer**
Standardized mortality ratio		3.82	5.87	2.93	—	5.32[d]
Attributable deaths		197.0	67.2	107.3	—	216.8
Attributable risk fraction, %		74	83	66	—	81
Total YPLL for cause g in cohort, yr		5300	2932	2796	11,028	5296
Excess YPLL , cause g in cohort, yr		4007	2534	1890	8431	4338
Attributable YPLL fraction, %		76	86	68	76	82
Excess YPLL per cohort member, yr		1.47	0.93	0.70	3.1	1.59
Excess YPLL per excess case, yr		20.3	37.7	17.6	—	20.0
Excess YPLL per year employed[a]	Yr	0.23	0.15	0.11	0.49	0.25
(in equivalent years, months, or days)	Mo	2.8	1.8	1.3	5.9	3.0
	Da	85	54	40	179	92
Excess YPLL per year underground[a]	Yr	0.34	0.22	0.16	0.72	0.37
(in equivalent years, months, or days)	Mo	4.1	2.6	1.9	8.6	4.4
	Da	124	79	59	261	135
Excess lifetime risk, this cohort[e]		0.145	0.045	0.088	0.278	—

[a]Cohort size: 2,721; average duration of employment (including gaps): 6.31 yr; total years of employment: 17,170; average duration underground: 4.32 yr; total yrs underground: 11,755.
[b]LDOC, lung disease other than cancer.
[c]Model including radon and smoking (Table V): deaths or YPLLs attributable to radon.
[d]SMR derived from model: observed deaths/(observed—radon-attributable deaths).
[e]Excess lifetime risk not calculated from Poisson regression model.

and other-injuries. Due to gaps in mining employment and above-ground assignments, the miners in this cohort averaged only 4.32 yr actually working underground. For every year spent underground, the uranium miners lost 8.6 months of potential life.

Applying the age-specific relative rates of the uranium miner population to a theoretical lifetime, excess lifetime risk was calculated taking into account competing causes of death. For lung cancer, other-fatal injuries, and LDOC the excess lifetime risks were 0.145, 0.045, and 0.088, respectively, for a total of 0.28 (Table IV). Thus, based on their mortality experience through 1990, more than one quarter of workers hired after 1950 and working a lifetime in uranium mining would be expected to die prematurely from mining-related exposures.

The age pattern of years of potential life lost from work-related chronic diseases was very different than that for fatal injury (Fig. 1). Injury dominated below age 40 where 30 percent of cohort followup occurred. Beyond retirement age only the chronic lung diseases contributed excess YPLLs, cancer and non-cancer diseases about equally.

Poisson Regression Models

The final model for lung cancer analyzed by Poisson regression produced a standardized rate ratio estimate for background lung cancer mortality (at age 50) of 0.132 (Table V), very close to the plausible value expected (for all ages combined) from the general population (0.126). The estimated rate ratio for mean cumulative smoking (among lung cancer decedents) was 9.14; rate ratios for radon or radon-smoking cumulative exposures in various age groups ranged 15.9–50.7 (at mean cumulative exposures of lung cancer cases) relative to a non-smoking, non-exposed population. In the final model, use of the cumulative radon exposure measure based on square root of exposure intensity ($WL^{0.5}$), rather than on WL itself, produced a considerably better fit (change in -2 ln[Likelihood] for the six radon terms was 177.2 vs. 158.4), confirming a dose-rate effect: relatively higher impact of low exposures. Using the model estimates to calculate YPLLs (i.e., applying the model to predict deaths in the study population with and without radon exposures set to zero and applying the age-appropriate YL_i to each period of observation) yielded a somewhat higher proportion of excess YPLLs (82 vs. 76) compared to the SMR/life table result and a slightly higher estimate of years of life lost per year employed: 0.25 vs. 0.23 (Table IV). LDOC and injury were not analyzed with Poisson regression because important exposure information was lacking (silica levels for LDOC, physical hazards for injuries). Also based on the Poisson regression model, the decline with age in the excess YPLL from lung cancer per year employed was described (Table VI). Among those at

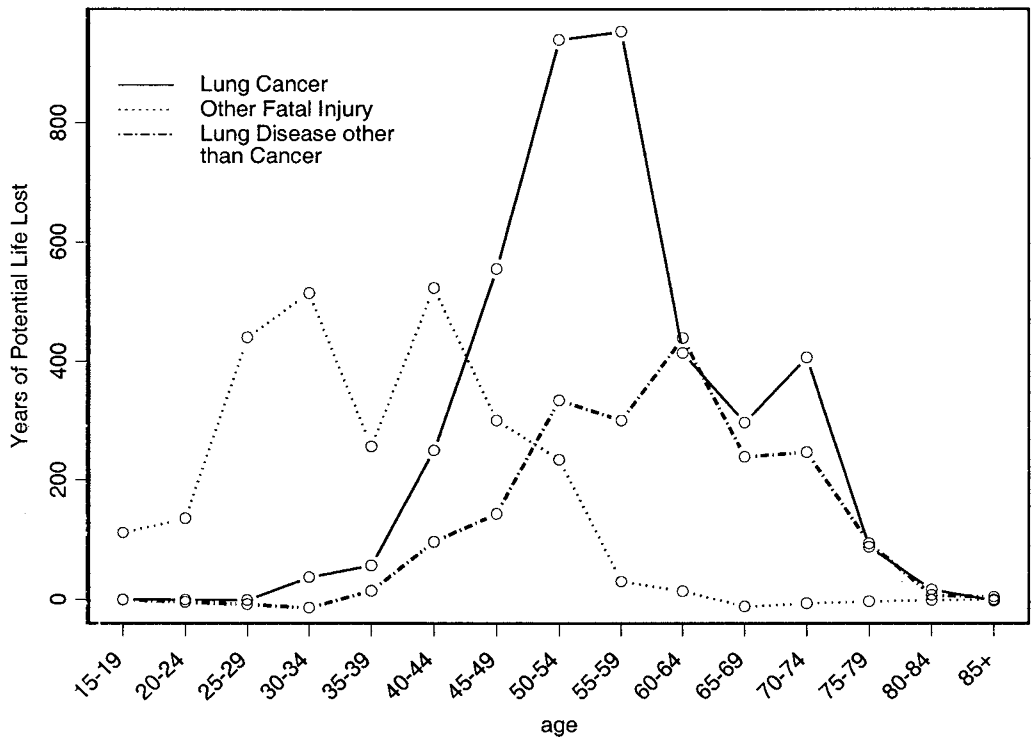

FIGURE 1. Colorado Plateau Uranium Miners Cohort: Excess YPLLs due to uranium mining in cohort hired 1950–63 by age at death, showing relative contributions of lung cancer, other-fatal injuries and lung disease other than cancer.

TABLE V. Colorado Plateau Uranium Miners 1950 Cohort: Rate Ratios for Lung Cancer With Cumulative Exposures to Radon (Square Root WL) and Smoking (Both With 5 yr Lag) by Poisson Regression

Model[a]	**n**[b]	**Estimate**	**Mean cum. exposure**[c]	**RR**[d]
Intercept	267	−2.022	—	0.13
Age (centered at 50; per 5 yr interval)	267	−0.1247	—	0.88
Cum. smoking	247	0.01789	454.9	9.14
Cum. radon (< age 60)	134	12.75	3.28	42.8
Cum. radon (age 60–69)	70	5.586	2.66	15.9
Cum. radon (> age 69)	61	10.19	2.51	26.6
Itr: radon (< age 60) × smoking	126	0.0301	1,114	34.5
Itr: radon (age 60–69) × smoking	65	0.0175	1,411	25.7
Itr: radon (> age 69) × smoking	54	0.0370	1,343	50.7

[a]Radon and smoking cumulative exposures lagged 5 yrs; radon cumulated as square-root of Working Level, in 100 $(WL)^{0.5}$-mos. Cumulative smoking in pack-mos. Age entered as loglinear term; radon and smoking terms entered as linear terms. Itr—interaction term. $-2 \ln(\text{Likelihood}) = 3797.8$; for terms involving radon, change in $-2 \ln L = 177$ for 6 df; $P < .000001$.

[b]n, number of smoking or radon-exposed lung cancer deaths.

[c]Mean cumulative exposure of lung cancer cases.

[d]RR, rate ratio; evaluated at age 50 (intercept), for 5 yr increase (Age) and at mean cum. exposures of lung cancer decedents.

TABLE VI. Colorado Plateau Uranium Miners 1950 Cohort: Excess Years of Life Lost From Lung Cancer Per Year of Employment in Uranium Mining, by Age of Surviving Population, Based on Poisson Regression Model

Age	Surviving population[a]	Total employment duration[b]	Excess YPLLs[c]	Excess YPLL per year employed
40–44	2,631	15789.3	4233.8	0.268
45–49	2,557	15703.5	3984.4	0.254
50–54	2,456	15750.5	3426.4	0.218
55–59	2,289	15199.4	2478.3	0.163
60–64	2,095	14041.3	1353.5	0.096
65–69	1,907	12517.7	871.4	0.070
70–74	1,756	11320.2	537.7	0.048

[a]To end of age interval.
[b]Person-year-weighted prior employment duration in age interval.
[c]Excess YPLLs predicted for current and subsequent time periods.

risk in the age interval 40–44, 0.27 yr of potential life were lost per year employed, declining to 0.10 yr among those in the 60–64 age interval.

DISCUSSION

Colorado Plateau Uranium Miners Cohort

The excess lifetime risk of dying for the uranium miners was 28 percent, a high occupational burden compared to the ceiling of 0.1 percent suggested by the U.S. Supreme Court in the benzene decision [Infante, 1995]. Exposures other than radon contributed to this excess mortality, including silica (related to the LDOC, lung cancer and TB excesses) [Checkoway et al., 1997] and mining safety hazards. Radon may have itself contributed to the LDOC excess [Archer et al., 1998], but most likely silica and other dusts dominated. Excess mortality in this SMR-based analysis was underestimated for two reasons. The healthy worker effect (HWE), evident in the low SMR for ischemic heart disease in this population (SMR = 0.78), would produce expected SMRs for lung cancer and particularly LDOC that are 0.90 or less [Park et al., 1991]. In the case of LDOC, SMRs in typical industrial cohorts free of respiratory hazards range 0.5–0.8 [Park et al., 1991] so that an unbiased estimate of LDOC relative risk in the uranium miners would be in the range 3.7–5.9, not 2.9. In addition, the YPLL analyses used standard life table-derived values for life expectancies that very likely are underestimates for healthy workers. The nature of the HWE for injuries is less clear, particularly as some driving injuries may relate to employment.

Second, other causes of death had excesses, including six excess leukemia deaths (13 obs, 7.3 exp) and nine excess tuberculosis deaths (12 obs, 2.6 exp), almost certainly due to silicotuberculosis given the number of silicosis deaths (20 after 1960) (Table II). Other silica-related mortality not analyzed could have included deaths from renal and cardiovascular disease. This study did not have the statistical power to detect elevated risks with small excess numbers at other anatomical sites.

On the other hand, some of the excess mortality could have arisen from employment other than in the uranium mines, thereby resulting in over-estimation of uranium mining effects. However, for these miners, estimates of radon exposures in non-uranium hard rock mining were a small part of their total (the mean non-study cum. exposure was 18 WLM compared with 606 WLM in study mines). Silica exposure, not characterized in this study, could have included significant contributions from non-study workplaces. This would affect the LDOC findings and, to a lesser extent, the lung cancer findings. In the case of lung cancer, internal comparisons using Poisson regression modeling would reduce the role of non study exposures.

The injury fatalities are more difficult to interpret in relation to work. We focused on the ‘‘other-fatal injury’’ category assuming most industrial accident cases would be classified there. Observing all 81 other-injury deaths to occur below age 65 supported that assumption. However, there were excesses as well in the categories ‘‘transportation’’ and ‘‘falls’’ (Table II), some of which were possibly work-related. In an attempt to exclude fatal injuries not occurring in study mines, we performed additional SMR analyses restricting the period of followup to include active employment and up to one year post employment in a study mine (to allow for delayed deaths from fatal injuries). With this restriction, the SMR for all accidents rose from 3.1 to 5.2, for other-fatal injury, from 5.9 to 14.1, and for transportation fatalities from 1.8 to 2.3. However, SMRs with followup beginning one year after termination from a study mine were still somewhat elevated for all accidents and other-fatal injury (2.4 and 3.6, respectively) indicating that: (1) part of the excess was not work-related, (2) there was excess risk in subsequent employment, or (3) many injuries in the study mines resulted in deaths delayed more than one year from the time of injury. On the other hand, the reference population rate for other-injuries may also largely represent work-related injuries, so that the SMR analysis is in reality identifying only the excess above this national rate. Thus, probably most of the excess mortality in the other-fatal injury category was attributable to uranium mining.

The detailed smoking information available for this population permitted a relatively rigorous treatment of smoking in the lung cancer analysis using Poisson regression. The larger excess YPLLs for lung cancer using the Poisson regression analysis compared with SMRs, suggests negative confounding (from smoking or perhaps, a healthy worker effect).

The measures of risk developed here for uranium miners depend on the joint age structure of employment and followup, which are specific to the study population; they could diverge considerably from the predicted risks of individual workers and would not be directly comparable to those derived from other uranium miner populations. Sasieni and Adams [1999] have proposed standardized lifetime risk and YPLL measures that would permit such comparisons.

Risk Characterization

The loss in life expectancy among uranium miners (estimated to be 86 months or 7 yr for 10 yrs of underground work) can be placed in perspective by comparison with gains in life expectancy from common medical interventions [Wright and Weinstein, 1998]. For example: quitting smoking in 35-year-old men is estimated to add 10 months of life from averted heart disease in populations at average risk, or 28 months in populations at high risk; annual fecal occult blood test plus tri-annual X-ray or colonoscopy in 50-year olds adds 2.8 months; chemotherapy in patients with extensive small cell carcinoma of the lung adds about 7 months; implantation of pace-makers in survivors of cardiac arrest with recurrrent arrhythmias adds 36–46 months; bone marrow transplant (compared with chemotherapy) in patients with relapsed non-Hodgkins lymphoma adds 72 mo. The loss of potential years of life from working 10 years in Colorado uranium mines generally greatly exceeds gains resulting from a variety of major medical interventions and preventive strategies, including quitting smoking.

Others have made similar calculations in other contexts. For example an estimated 6.9 min of life lost are lost per cigarette smoked [Centers for Disease Control and Prevention, 1994] which, at one pack per day, corresponds to the loss of 12 months of life for 10 yr of smoking. Ultimately, risk reduction is based on choices by society, employers and employees of acceptable levels of risk. This report demonstrates alternative measures for communicating risk such as the per-person YPLL that are methodologically only slightly more complex than an SMR but may well be more meaningful, especially for individual workers. We believe the perceived significance of a loss of 2 months per year worked from LDOC is quite different from that of an "SMR of 3.0," or "a three-fold excess of respiratory disease deaths." Although cumulative or lifetime risks present a probability that is closer to an intuitive perception of personal risk, they do not at all address the time course of the expected adversity or its impact on one's life expectancy. The approach here addresses that concern and also one recently raised by Greenland [1999] that exposures whose effect is to accelerate the onset of inevitable disease are not adequately described with attributable-risk fraction or cumulative risk.

This relatively simple analysis [Appendix] based on life tables and SMRs, validated in the case of lung cancer by a more sophisticated regression analysis accounting for smoking, illustrates another dimension for the communication of risk of occupational disease and injury. Providing a clearer measure of adverse effect would improve risk communication when, otherwise, insufficient awareness or unintuitive measures can result in widely differing perceptions of personal risk. Occupational injury and disease epidemiologists should be encouraged to present years lost per years worked along with more traditional measures of exposure-associated mortality, particularly for study populations where excess risk has arisen from a relatively short duration of employment.

APPENDIX

For the SMR-based analysis, Table III contains the entire basis for calculating excess years of life lost, various derivatives of which were summarized in Table IV. A Poisson regression modeling approach yields a prediction equation for the number of deaths from the cause of interest in each observation interval of the classification table—$E\ (n) = (\text{P-Yrs}) \cdot \exp(a + b_1X_1 + b_2X_2 + b_3X_3 + \cdots)$. Applying this equation to each observation interval with the measured exposures, and then subtracting the same prediction but with the exposure of interest set to zero, yields the excess deaths in each observation interval. Each observation interval has been classified on age so that a life expectancy can be attached to each excess death. Summing across all observation time excess deaths multiplied by years of potential life lost at that age yields excess years of life lost for the study population.

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