

Cancer risk analysis of low-dose radiation exposure

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Abstract. Cancer risk assessment was conducted in a population that received prolonged low dose-rate γ -irradiation for about 10 years as a result of occupying buildings containing ⁶⁰Co-contaminated steel in Taiwan. The cancer risks were compared with those populations with the same temporal and geographic characteristics in Taiwan by standardized incidence ratios, adjusted for age and gender. The standardized incidence ratios were significantly higher for all leukemia except chronic lymphocytic leukemia in men, and marginally significant for thyroid cancers in women. All cancers combined were shown to exhibit significant exposure-dependent increased risks in individuals with the initial exposure before 30 years of age, but not beyond this age. Excess relative risk at 1 Sv by Cox proportional hazard model was significantly high in all cancers combined, breast cancers and leukemia, while it was marginally significant in solid cancers combined, all solid cancers combined, stomach cancers and lung cancers. The results indicated that protracted low-dose radiation could induce higher cancer risks in the general public, especially for leukemia and the risks were comparable with those of acute radiation exposure. © 2006 Elsevier B.V. All rights reserved.

Keywords: ⁶⁰Co; Low dose-rate; Radiation; Cancer risk; Human population

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1. Introduction

There are few studies concerning exposure of the general public, particularly including women and children at different ages and various exposure. In late 1982, several ^{60}Co orphan sources were recycled into the steel scrap industry in northern Taiwan resulting in more than 20,000 tons of various contaminated products employed in over 200 residential, industrial, and school buildings [1,2]. It was not until August 1992 that these contaminated buildings started to be identified. The rates of exposure (0.5–270 $\mu\text{Sv/h}$) in them, measured in 1994, have been estimated to be several to >1000 times the background radioactivity (around 0.1 $\mu\text{Sv/h}$) in the general Taiwanese construction. A follow-up study was designed to evaluate risks of developing cancers in these radio-contaminated buildings' exposed population more than 10 years after their initial exposure.

2. Materials and methods

2.1. Study population

The household registration system was established by the Ministry of Interior (MOI) in the 1950s and further computerized in the 1990s. We were then able to trace all individuals with official residential history in these buildings since 1982 when they first moved in. Moreover, individuals identified in the home-owner records or reported by other registrants, but not in MOI's registration, were then evaluated for details of their occupancy through extensive contacts and interviews. Those who left before the registration in 1992 were also traced by household registration, police records and other approaches. If the individuals moved out of these buildings or when the contaminated steel was removed or shielded, they were designated as not having additional exposure. Systematic questionnaire-based interviews were administered to each registered individual to obtain medical history, occupation, education, and detailed exposure history including dates moved in and out and lifestyles in these buildings. Excess exposure to other radiation sources, such as occupational, medical or radiotherapy, was also evaluated. If they were deceased, interviews were conducted with their families, as well as teachers at school or employers when necessary. They were further evaluated periodically after they relocated to non-contaminated buildings. At the end of 2002, 7271 members (including 3461 men and 3810 women) were registered and most of them were followed up closely.

2.2. Ascertainment of cancer cases

Cancer cases were identified through the National Cancer Registry of Taiwan (NCRT), which was established in 1979 by the Department of Health. The cases identified through the NCRT were coded by the International Classification of Disease for Oncology (ICD-O, first edition).

2.3. Exposure evaluation

Exposure assessment by the Taiwan Cumulative Dose (TCD) was established in the beginning of the cohort registration and applied to exposure assessment on an individual basis. The TCD integrated the time-activity analysis [3] and the highly occupied zone (HOZ) model

Table 1
Distribution of person–years at risk^a and cancer cases by age at initial exposure, cumulative exposure and gender

Age at initial exposure (years)	Cumulative exposure (TCD ^b , mSv)	Men		Women		Total		Dose rate (mSv/year)	Average follow-up time (years)
		Person–years at risk	Number of cancers ^a	Person–years at risk	Number of cancers	Person–years at risk	Number of cancers		
≤ 30	<1	10,851	1	12,404	2	23,255	3	11.0 (<1, 1413)	15.8 (<1, 20)
	1–50	17,366	3	19,473	12	36,839	15		
	>50	4598	2	4569	5	9167	7		
	40.0 (<1, 2206) ^c	32,815	6	36,446	19	69,261	25		
	Missing	5804	1	6147	0	11,951	1		
Total		38,619	7	42,593	19	81,212	26		
>30	<1	1914	7	2755	9	4669	16	8.5 (<1, 469.5)	17.2 (2, 20)
	1–50	4941	19	4780	17	9721	36		
	>50	1683	5	1810	5	3493	10		
	80.0 (<1, 2363)	8538	31	9345	31	17,883	62		
	Missing	962	4	1503	3	2465	7		
Total		9500	35	10,848	34	20,348	69		
All age	<1	12,765	8	15,159	11	27,924	19	10.5 (<1, 1413)	16.1 (<1, 20)
	1–50	22,307	22	24,253	29	46,560	51		
	>50	6281	7	6379	10	12,660	17		
	47.8 (<1, 2363)	41,353	37	45,791	50	87,144	87		
	Missing	6766	5	7650	3	14,416	8		
Total		48,119	42	53,441	53	101,560	95		

^a Latent period considered, 2 years for leukemia and 10 years for other cancers.

^b Taiwan Cumulative Dose.

^c Mean (minimum, maximum).

Table 2
Standardized incidence ratios for the exposed population^a

Cancer site	Men			
	Observed	Expected	SIR ^b	(95% CI) ^c
All cancers	42	53.8	0.8	(0.5, 1.0)
All cancers except leukemia	36	52.0	0.7 ^e	(0.5, 0.9)
Solid cancers	32	50.9	0.6 ^e	(0.4, 0.8)
Tongue	0	1.1	NA ^f	NA
Oral	1	1.4	0.7	(0.02, 4.0)
Nasopharynx	1	2.0	0.5	(0.01, 2.7)
Esophagus	1	1.8	0.6	(0.01, 3.2)
Stomach	5	4.9	1.0	(0.33, 2.4)
Colon	2	4.0	0.5	(0.1, 1.8)
Rectum	3	3.1	1.0	(0.2, 2.8)
Liver	5	8.9	0.6	(0.2, 1.3)
Lung	7	7.6	0.9	(0.4, 1.9)
Connective	1	0.5	2.1	(0.1, 11.9)
Skin	2	1.5	1.4	(0.2, 4.9)
Melanoma skin	0	0.2	NA	NA
Non-melanoma skin	2	1.4	1.5	(0.2, 5.3)
Breast	0	0.0	NA	NA
Cervix uteri	0	0.0	NA	NA
Corpus uteri	0	0.0	NA	NA
Prostate gland	1	3.4	0.3	(0.01, 1.7)
Kidney	2	1.3	1.5	(0.2, 5.5)
Thyroid glands	1	0.5	2.0	(0.1, 11.1)
Leukemia (all types)	6	1.8	3.4 ^e	(1.2, 7.4)
Leukemia except for CLL ^g	6	1.7	3.6 ^e	(1.3, 7.8)
Acute lymphocytic leukemia (ALL)	3	0.4	6.8 ^e	(1.4, 19.8)
Acute myelocytic leukemia (AML)	2	0.6	3.3	(0.4, 11.8)
Chronic myelocytic leukemia (CML)	1	0.3	3.9	(0.1, 21.9)
Multiple myeloma	1	0.3	3.9	(0.1, 21.5)
Malignant lymphoma	3	0.9	3.3	(0.7, 9.7)
Non-Hodgkin's lymphoma	3	0.5	6.3 ^e	(1.3, 18.4)

^a Latent period considered.

^b Standardized incidence ratio.

^c CI=confidence interval.

^d $0.05 \leq p < 0.1$.

^e $p < 0.05$.

^f NA=not available.

^g Chronic lymphocytic leukemia.

[4], incorporating a detailed history of occupancy in each radioactive area of the buildings and area-specific radiation exposure to the whole-body. Environmental radioactivity of representative spots in each room was measured accordingly. Cumulative excessive exposures were corrected for the half-life of radioactive decay, i.e., 5.27 years for Co-60, and integrated with lifestyle patterns while residing in the contaminated buildings. The TCD was shown to be comparable with biodosimetric analysis by fluorescence in situ hybridization of chromosomal

Women				All			
Cases				Cases			
Observed	Expected	SIR	(95% CI)	Observed	Expected	SIR	(95% CI)
53	60.9	0.9	(0.7, 1.1)	95	114.9	0.8 ^d	(0.7, 1.0)
52	59.3	0.9	(0.7, 1.2)	88	111.6	0.8 ^e	(0.6, 0.9)
50	58.5	0.9	(0.6, 1.1)	82	109.5	0.7 ^e	(0.6, 0.9)
1	0.3	3.7	(0.1, 20.7)	1	1.5	0.7	(0.02, 3.7)
0	0.2	NA	NA	1	1.7	0.6	(0.02, 3.3)
0	1.0	NA	NA	1	3.1	0.3	(0.01, 1.8)
1	0.3	3.6	(0.1, 20.3)	2	2.2	0.9	(0.1, 3.3)
2	3.1	0.6	(0.1, 2.3)	7	8.2	0.8	(0.3, 1.8)
3	3.8	0.8	(0.2, 2.3)	5	7.9	0.6	(0.2, 1.5)
2	2.7	0.7	(0.1, 2.7)	5	5.9	0.8	(0.3, 2.0)
3	3.7	0.8	(0.2, 2.3)	8	13.1	0.6	(0.3, 1.2)
3	4.5	0.7	(0.1, 2.0)	10	12.5	0.8	(0.4, 1.5)
1	0.4	2.3	(0.1, 12.6)	2	0.9	2.2	(0.3, 7.9)
1	1.5	0.7	(0.02, 3.6)	3	3.0	1.0	(0.2, 2.9)
1	0.2	5.4	(0.1, 30.1)	1	0.4	2.8	(0.1, 15.7)
0	1.4	NA	NA	2	2.8	0.7	(0.1, 2.6)
12	12.1	1.0	(0.5, 1.7)	12	11.2	1.1	(0.6, 1.9)
12	12.9	0.9	(0.5, 1.6)	12	11.9	1.0	(0.5, 1.8)
3	1.5	2.0	(0.4, 6.0)	3	1.4	2.2	(0.5, 6.4)
0	0.0	NA	NA	1	3.8	0.3	(0.01, 1.5)
0	1.1	NA	NA	2	2.4	0.8	(0.1, 3.0)
6	2.3	2.6 ^d	(1.0, 5.7)	7	2.7	2.6 ^e	(1.1, 5.4)
1	1.5	0.7	(0.02, 3.7)	7	3.3	2.1 ^d	(0.8, 4.3)
1	1.5	0.7	(0.02, 3.8)	7	3.2	2.2 ^d	(0.9, 4.6)
0	0.4	NA	NA	3	0.8	3.6 ^d	(0.7, 10.4)
1	0.5	1.8	(0.05, 10.1)	3	1.2	2.5	(0.5, 7.4)
0	0.2	NA	NA	1	0.5	2.2	(0.1, 12.1)
0	0.2	NA	NA	1	0.5	2.2	(0.1, 12.3)
2	0.7	2.9	(0.04, 8.1)	5	1.6	3.1 ^e	(1.0, 7.2)
2	0.4	4.6	(0.6, 16.5)	5	0.9	5.4 ^e	(1.8, 12.6)

translocation frequencies [5]. The average excessive cumulative exposure, i.e., above background radiation exposure, was 47.8 mSv (ranging from < 1 mSv to 2363 mSv). The estimated dose-rate of excess exposure was 10.5 mSv/year on average (< 1 to 1413 mSv/year).

2.4. Statistical analysis

The standardized incidence ratio (SIR) adjusted for age and gender was calculated with the observed number of cancer cases as the numerator and the expected number of specific cancer cases as the denominator, compared with a reference population with the same temporal and geographic characteristics. There was no attempt to match economic factors related to residency, although residency was apparently influenced by economic factors.

Table 3
Relative risks (RR) of cancer by different categories of Taiwan Cumulative Dose (TCD) and age at initial exposure^a

	Cohort members	All cancers		All solid cancers		Leukemia except for CLL ^b		Thyroid cancers		Breast cancers		Combination of thyroid and breast	
		N ^c	RR (95% CI) ^d	N	RR (95% CI)	N	RR (95% CI)	N	RR (95% CI)	N	RR (95% CI)	N	RR (95% CI)
<i>Age at initial exposure</i>													
<i>≤30 years</i>													
<i>Gender</i>													
Men ^e	2408	6	1	3	1	3	1	0	1				
Women	2633	19	2.8 ^f (1.1, 7.0)	17	4.8 ^f (1.4, 16.4)	0	NA ^g	2	NA	6		8	
<i>TCD (mSv)</i>													
<1 ^e	1962	5	1	9	1	2	1	1	1	4	1	5	1
1–50	2489	14	2.6 ^h (0.9, 7.5)	10	1.3 (0.5, 3.2)	0	NA	1	NA	2	NA	3	0.8 (0.2, 3.3)
>50	590	6	4.7 ⁱ (1.4, 15.7)	1	2.3 (0.3, 18.0)	1	NA	0	NA	0	NA	0	NA
Total	5041	25		20		3		2		6		8	
<i>>30 years</i>													
<i>Gender</i>													
Men	568	31	1	26	1	2	1	1	1				
Women	637	31	0.9 (0.6, 1.5)	30	1.1 (0.6, 1.8)	1	NA	4	3.8 (0.4, 34.2)	6		10	

TCD (mSv)													
<1	323	19	1	28	1	0	1	2	1	4	1	6	1
1–50	648	36	0.9 (0.5, 1.7)	26	1.0 (0.6, 1.6)	2	NA	3	1.7 (0.3, 10.3)	1	0.3 (0.03, 2.5)	3	0.6 (0.1, 2.3)
>50	234	7	0.6 (0.3, 1.5)	2	0.6 (0.2, 2.7)	1	NA	0	NA	1	2.6 (0.3, 23.3)	1	1.7 (0.2, 14.4)
Total	1205	62		56		3		5		6		10	
<i>All age</i>													
Gender													
Men	2976	37	1	29	1	5	1	1	1				
Women	3270	50	1.2 (0.8, 1.8)	47	1.4 (0.9, 2.3)	1	0.2 (0.02, 1.6)	6	NA	12			18
TCD (mSv)													
<1	2285	24	1	37	1	2	1	3	1	8	1	11	1
1–50	3137	50	1.2 (0.7, 2.0)	36	1.0 (0.6, 1.5)	2	0.8 (0.1, 6.2)	4	NA	3	0.4 (0.1, 1.7)	6	0.6 (0.2, 1.7)
>50	824	13	1.2 (0.6, 2.3)	3	1.0 (0.3, 3.0)	2	3.1 (0.4, 24.1)	0	NA	1	1.9 (0.2, 15.5)	1	1.4 (0.2, 10.6)
Total	6246	87		76		6		7		12		18	

^a Latent period considered.

^b Chronic lymphocytic leukemia.

^c Number of cases.

^d CI=confidence interval.

^e Reference group.

^f $p < 0.05$.

^g NA=not available.

^h $0.05 \leq p < 0.1$.

Cancer risks associated with exposure were analyzed by the Poisson regression model. The models were estimated for those with age at initial exposure ≤ 30 or for >30 years. The excess cumulative exposure was lagged 2 years for leukemia and 10 years for other cancers to allow for a minimum latent period of cancer induction. Cox proportional hazard regression models were used to estimate the hazard ratios. The excess relative risk (ERR) was calculated as relative risk minus 1 [6], and the 95% confidence interval for incremental excess relative risk at 1 Sv ($ERR_{1\text{ Sv}}$) was then constructed, with all tests two-tailed.

2.5. Results

These 7271 cohort members were followed up for 16.1 ± 4.0 (mean \pm standard deviation) years on average (<1 to 20 years), with initial exposure at 17.2 ± 16.0 years of age (intrauterine to 80 years old) and a total of 101,560 person–years at risk of follow-up (48,119 for men and 53,441 for women) (Table 1). A total of 141 cohort members were found to have developed cancers, and 46 of these cases (45 solid cancers and 1 multiple myeloma) developed in less than the minimal latent periods; that is, 95 subjects who developed cancers with latent period more than the minimal latent periods were included for analysis.

Simple SIR analyses (Table 2) revealed significantly elevated risks for thyroid cancers ($n=7$, SIR=2.6, 95% confidence interval (CI): 1.1–5.4) and non-Hodgkin's lymphoma ($n=5$, SIR=5.4, 95% CI: 1.8–12.6), and marginally significant elevated risk for leukemia except chronic lymphocytic leukemia (leukemia except CLL; $n=7$, SIR=2.2, 95% CI: 0.9–4.6), for both genders combined. SIR was marginally significantly lower in both genders combined for all cancers (SIR=0.8, 95% CI: 0.7–1.0). There were significantly lower risks for all cancers except leukemia (SIR=0.8, 95% CI: 0.6–0.9), and all solid cancers combined (SIR=0.7, 95% CI: 0.6–0.9). In men, there were significantly elevated risks for all leukemia ($n=6$, SIR=3.4, 95% CI: 1.2–7.4) and leukemia except CLL ($n=6$, SIR=3.6, 95% CI: 1.3–7.8). In women, there were marginally significant elevated risks for thyroid cancers ($n=6$, SIR=2.6, 95% CI: 1.0–5.7).

Among those who received initial exposure before 30 years of age, those who were exposed to more than 50 mSv had significantly increased risk for all cancers combined (relative risk (RR)=4.7, 95% CI: 1.4–15.7), as compared with those who had received less than 1 mSv of exposure (Table 3). There was no significant exposure-dependent increase in those with initial exposure after the age of 30 years. For all ages combined, there was no significant exposure-dependent increase in leukemia (except CLL; RR=3.1, 95% CI: 0.4–24.1), breast cancers (RR=1.9, 95% CI: 0.2–15.5), and thyroid/breast cancers combined (RR=1.4, 95% CI: 0.2–10.6) among those with more than 50 mSv as compared with those received less than 1 mSv of exposure.

There were significant increased risks in ERR for all cancers combined ($ERR_{1\text{ Sv}}=1.0$, 95% CI: 0.09–3.0), breast cancers ($ERR_{1\text{ Sv}}=2.4$, 95% CI: 0.03–10.1), leukemia ($ERR_{1\text{ Sv}}=8.1$, 95% CI: 0.4–57.4) and leukemia (except chronic lymphocytic leukemia; $ERR_{1\text{ Sv}}=8.1$, 95% CI: 0.4–57.4) (Table 4). There were marginally significant increased risks for all solid cancers combined (except thyroid cancers; $ERR_{1\text{ Sv}}=1.0$, 95% CI: -0.07 –3.1), all solid cancers combined ($ERR_{1\text{ Sv}}=0.9$, 95% CI: -0.07 –3.0), cancers of stomach ($ERR_{1\text{ Sv}}=4.1$, 95% CI: -0.2 –32.9), and lung ($ERR_{1\text{ Sv}}=2.0$, 95% CI: -0.1 –9.4).

Table 4
Excess relative risk estimates per Sv (ERR_{1 Sv}) in the exposed population^a

Cancers	Number of cases	ERR _{1 Sv}	95% CI
All cancers	87	1.0 ^b	(0.09, 3.0)
All cancers (except leukemia)	81	0.8	(−0.1, 2.8)
Solid cancers (except thyroid cancers)	69	1.0 ^c	(−0.07, 3.1)
Solid cancers	76	0.9 ^c	(−0.07, 3.0)
Stomach	7	4.1 ^c	(−0.2, 32.9)
Liver	6	2.0	(−0.8, 40.0)
Lung	10	2.0 ^c	(−0.1, 9.4)
Breast	12	2.4 ^c	(0.03, 10.1)
Cervix uteri	12	−1.0	(−1.0, 5.3 × 10 ³)
Thyroid glands	7	−0.1	(−1.0, 3.8 × 10 ²)
Leukemia (all types)	6	8.1 ^b	(0.4, 57.4)
Leukemia except CLL ^d	6	8.1 ^b	(0.4, 57.4)

^a Based on Cox proportional hazard model: $h(t) = h_0(t) \exp(\beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_4)$, where $h_0(t)$ is a baseline hazard function, X_1 is gender, X_2 is age at initial exposure, X_3 is attained age and X_4 is excess cumulative exposure.

^b $p < 0.05$.

^c $0.05 \leq p < 0.1$.

^d CLL = chronic lymphocytic leukemia.

For all cancers combined, the ERR_{1 Sv} was 1.1 (95% CI: 0.07–3.1), 1.4 (95% CI: 0.2–3.8), 2.9 (95% CI: 0.8–7.1), and 4.6 (95% CI: 1.3–12.6) for those with equal or more than 0.2, 0.5, 3.0 or 10.0 mSv/year, respectively, while the ERR_{1 Sv} for all the exposed cohort was 1.0 (95% CI: 0.05–2.9). There was a significant trend of increase with excess dose-rate of exposure in average ($p = 0.003$).

3. Discussion

The exposed cohort is unique because it consists of a general population, and has received relatively higher radiation exposure similar to those in the Life Span Study (LSS) cohort. However, the exposed cohort differs from that of the LSS in that the exposure was protracted and primarily from gamma rays. It was different from other occupational radiation exposure cohorts in that it comprised similar numbers of both genders.

This study showed that there were significantly elevated SIRs for leukemia (except CLL) in men and significantly elevated risks for malignant lymphoma (especially for non-Hodgkin's lymphoma) in both genders combined, also reported by the UNSCEAR [7]. Owing to the relatively small number of cancer cases, leukemia except CLL was not shown with significantly elevated risks, with gender considered.

This study did not observe an elevated SIR for breast cancers, but there was a significant exposure-dependent increase in breast cancers in the exposed population. Several other variables for breast cancers, including body mass, reproductive history and hormone use, were not considered in this analysis, and may affect different observations in SIR and RR. A further nested case-control study for this cohort will help clarify the association between exposure and breast cancers.

Significant exposure-dependent increased risks in individuals with initial exposure before age of 30 years, but not beyond, especially for all cancers combined, was noted. According to

the ICRP 60 report [8], cancer risks associated with low-dose or low dose-rate exposures may be less than from acute high exposure. A reduction factor, termed the “dose and dose rate effectiveness factor” (DDREF) is used to allow for a reduced effectiveness of radiation in inducing cancer in human populations at both low doses and low dose rates. Studies on the Japanese atomic bomb survivors indicated DDREF as 2 to 2.5 for leukemia, about 1.4 for solid cancers, and 1.7 for leukemia and solid cancers combined. For solid cancers, DDREF were very similar. In the current study, a combination of thyroid/breast cancers was representative as the target radiation sensitive solid cancer for assessing the excess cumulative exposure-dependent relationship between radiation and cancer incidence. Further follow-up in this cohort population would provide better analysis on the related DDREF.

Compared with the reference population, the study population had lower incidences of all cancers combined, all cancers combined except leukemia, and all solid cancers combined. Most study cohort members have resided in buildings constructed in the early 1980s, a period of rapid economic development in Taiwan. It was likely that the exposed population could have higher socioeconomic status than the general population, with healthier lifestyles and consequently lower cancer risks, a situation that has been described in other population studies. Unfortunately, we were not able to closely match the economic status of these individuals with the reference population in this study. Since the exposure assessment was made without knowledge of the disease status of these exposed subjects, information bias was unlikely, even though there might be random measurement errors.

In this study, we used both external comparison (SIR) and internal comparison (RR) to assess the cancer risks of the exposed population. The relative risk analysis can further help clarify the relationship between excessive cumulative radiation exposure and cancer risks in this population. This study cohort was large enough to detect statistically significant cumulative exposure-dependent increases in various cancers, but the average follow-up period was still too short to observe the development of the whole spectrum of cancers. Further follow-up of the study cohort is necessary to corroborate our findings and identify other types of cancers that may also be related to the protracted and low dose-rate ionizing radiation.

The Tables 1, 2 and 3 were reproduced from S-L. Hwang, et al., Cancer risks in a population with prolonged low dose-rate gamma-radiation exposure in radio-contaminated buildings, 1983–2002, *Int. J. Radiat. Biol.* 82 in press, by the Taylor & Francis Group.

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