

Leukemia incidence among people exposed to chronic radiation from the contaminated Techa River, 1953–2005

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Abstract Beginning in 1950, people living on the banks of the Techa River received chronic low-dose-rate internal and external radiation exposures as a result of releases from the Mayak nuclear weapons plutonium production facility in the Southern Urals region of the Russian Federation. The Techa River cohort includes about 30,000 people who resided in riverside villages sometime between 1950 and 1960. Cumulative red bone marrow doses range up to 2 Gy with a mean of 0.3 Gy and a median of 0.2 Gy. Between 1953 and 2005, 93 first primary cases of leukemia, including 23 cases of chronic lymphatic leukemia (CLL), were ascertained among the cohort members. A significant linear dose–response relationship was seen for leukemias other than CLL ($P < 0.001$), but not for CLL. The estimated excess relative risk per Gy is 4.9 (95% confidence interval (CI): 1.6; 14.3) for leukemias other than CLL and less than 0 (95% upper bound 1.4) for CLL.

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Introduction

This paper is one of a series of publications describing the Techa River cohort (TRC), which was established in 1967 by the Urals Center for Radiation Medicine (URCRM), to study the long-term health effects of chronic environmental radiation exposure to the riverside villages (Akleyev and Lyubchansky 1994; Kossenko et al. 1997, 2005; Degteva et al. 2000a, b; Krestinina et al. 2007). The population living near the Techa River was exposed to low to moderate radiation doses at low-dose rates from radioactive releases from the Mayak plutonium production facility into the Techa River between 1950 and 1956 (Akleyev and Lyubchansky 1994). The TRC is comprised an unselected general population of men and women of all ages from two different ethnic groups who lived in similar conditions and have received comparable levels of health care. Cohort members were exposed to protracted external and internal ionizing radiation. Internal exposure was from radioisotopes including the bone-seeking radionuclide ^{90}Sr incorporated through contaminated food and drinking water. Because ^{90}Sr was the primary source of exposure to the bone marrow, quantifying the risk of leukemia is a prime interest for this study cohort.

Radiation is a well-established leukemia risk factor as indicated by acute or protracted external exposures among atomic bomb survivors (Preston et al. 1994, 2004), Chernobyl cleanup workers (Kesminiene et al. 2008; Romanenko et al. 2008), patients with endometrial cancer (Curtis et al. 1994), and Mayak workers (Shilnikova et al. 2003), among others. The TRC is one of the few populations that can provide quantitative estimates of the risk of leukemia following low to moderate doses at low-dose rates of environmental radiation exposures. Analyses of mortality in the TRC have revealed a dose–response

relationship for leukemias other than CLL (non-CLL) (Krestinina et al. 2005). The analyses described in this paper differ from previous analyses in that they make use of data on the incidence of non-CLL and CLL and involve an extended follow-up from 1953 through 2005 compared to 1950–1999 in the earlier follow-up. Because of the unique characteristics of the population, the nature of the exposures, and the length of follow-up, risk estimates from the TRC can play an important role in the development of radiation protection standards for the general public.

Materials and methods

Cohort definition, catchment area, and follow-up period

The Techa River, which is part of the Ob river system, originates near the Mayak complex and flows about 240 km until it merges with the Iset River. In the 1950s, there were 41 villages along the river. The TRC includes 29,756 persons born before January 1, 1950, who lived in any of the Techa riverside villages during the period from 1950 to 1960. More than 80% of the cohort members lived near the river between 1950 and 1953, the period of highest radiation exposure. The cohort includes individuals of all ages, consists of about 58% female, and is comprised of two major ethnic groups, with 80% being of Slav origin and 20% being Tatar or Bashkir origin. About 40% of the cohort members were less than 20 years old when first exposed.

Follow-up begins in 1953 (there are no reported leukemia cases in the cohort prior to 1953, but as discussed in the following paragraphs, ascertainment is unlikely to be complete during 1950–1952) and cohort members are considered to be at risk only for periods of time when they are known to have lived in Chelyabinsk or Kurgan Oblast. Cohort members are considered at risk from the first date after 1952 that they lived in either Oblast, and they are considered lost to follow-up when their vital status becomes unknown or when they have emigrated from the study catchment area. Cohort members for whom information is not available at the Chelyabinsk and Kurgan Oblast address bureaus and who do not currently live at their last known address are classified as having an unknown vital status. Because information on people who have left an area is retained for only 6 years at the address bureau, the probability of tracing an individual lost to follow-up more than 6 years ago is very low. When cohort members are known to have left the study catchment area, they are classified as migrants (Kossenko et al. 2005).

Table 1 summarizes vital status and follow-up data for TRC members through December 31, 2005. At this time, 21% of the cohort members were alive and living in the

Table 1 Vital status for TRC members as of December 31, 2005

Vital Status	<i>N</i>	%
Alive	6,363	21.4
Deceased	16,619	55.9
Cause of death known	15,025	90.4
Cause of death unknown	1,594	9.6
Vital status unknown	2,219	7.5
Distal migrants	4,555	15.3
Total	29,756	100.0

catchment area. Among the 56% of cohort members identified as deceased, the cause of death was established for 90%. Vital status at the end of follow-up was unknown for only 7.5% of cohort members remaining in the study catchment area, but follow-up was censored for about 15% of the cohort when they emigrated from the catchment area (referred to as *distal migrants*). Neither migration nor having unknown vital status as of the end of 2005 was associated with dose. Thus, while these losses reduce the power to detect radiation effects, they should not bias the risk estimates.

Sources of information

Leukemia cases were ascertained from four main sources: URCRM medical records, regional oncology clinics, regional health centers, and death certificates. Since 1950, leukemias were ascertained from death certificates stored at the government Civil Registration offices (ZAGS) of Chelyabinsk and Kurgan Oblasts (19% of all cases). In 1955, Dispensary No. 1 of the Institute of Biophysics (subsequently Branch 4 of the Biophysics Institute and currently the URCRM) was given responsibility for the systematic observation and treatment of radiation-exposed individuals with hematologic disorders. The medical records of patients with leukemia diagnosed between 1953 and 1955 were obtained by URCRM from the Chelyabinsk region hospital. The URCRM was a key source of information on leukemia cases in the first two decades of follow-up, providing data on 39% of all leukemia cases. Since 1956, leukemia notification forms from the Chelyabinsk and Kurgan Oblast oncology dispensaries have been linked to the TRC cohort roster, and about 37% of all cases were identified from this source. Since the dispensaries are the major cancer treatment facilities in the study catchment area, they have become the primary source for newly diagnosed cases. The remaining cases (about 6% of the total) were ascertained from the records of health centers in Chelyabinsk and Kurgan Oblasts. With these data sources, the ascertainment of leukemia cases is largely complete by 1953 for cohort members residing in Chelyabinsk Oblast or

Kurgan Oblast. Description of information sources in greater detail is provided by Kossenko et al. (2005). For cases treated and followed at the URCRM clinic, cytological and histological examinations are available. Diagnoses for 81% of the leukemia cases were made by qualified hematologists. For cases identified through death certificates only, we lack verification if no autopsy was performed. The details of leukemia case confirmation were given by Ostroumova et al. (2006). For study analyses, all leukemia cases, like all causes of death and all cancer cases, were coded using ICD-9.

Dosimetry

The population of the Techa riverside villages was exposed to external and internal radiation. External radiation exposure from gamma-emitting radionuclides (^{137}Cs , ^{95}Zr , ^{95}Nb , ^{103}Ru , ^{106}Ru , and others) was a consequence of the contamination of river water and floodplains. Internal exposure resulted from consumption of water, milk, and other food products containing radionuclides (primarily ^{89}Sr , ^{90}Sr , and ^{137}Cs) (Degteva et al. 2000a). Internal exposure was primarily a result of ^{90}Sr incorporation into the bone structure, for which the bone marrow was the main target organ. About 80% of the total red bone marrow (RBM) dose was accumulated during the first decade of exposure. The mean (maximum) total dose rate in the study decreased from 40 mGy/year (240 mGy/year) in 1950–1951 to 8 mGy/year (60 mGy/year) in 1960.

Red bone marrow doses were estimated using the TRDS-2000 dosimetry system (Degteva et al. 2000a, b; Kossenko et al. 2005; Krestinina et al. 2005; 2007). The system provides individualized internal and external organ dose estimates and information on the uncertainty of these estimates. Estimates of annual total doses were derived from annual village mean dose estimates that allow for the nature of the releases (source term), distance from the release point, distance of the typical residence to the river flood plain, source of drinking water, and other factors. These were then individualized to account for gender, age, residence history, and other factors. Dates of residency in the contaminated area were abstracted from the tax books for each study settlement, and additional information was obtained from individual interviews using a specially developed questionnaire. Since the 1970s, information on current residence and migration has been obtained from routine address bureau queries used as the primary source for vital status follow-up.

The estimated maximum cumulative RBM dose (external and internal) was 2 Gy, the mean dose was 0.3 Gy, and the median was 0.2 Gy. Total cumulative RBM dose estimates are between 0.01 and 0.5 Gy for two-thirds of the cohort members, while 19% had estimated RBM doses in

excess of 0.5 Gy. On average, 92% of the total RBM dose was due to internal exposure. A more detailed description of the contribution made by external and internal exposure to dose is presented in Degteva et al. 2000a, b, Krestinina et al. 2005, Ostroumova et al. 2006). The current analyses were based on two-year-lagged cumulative RBM dose.

Data organization and statistical methods

Analyses of background rates and excess risks were carried out using Poisson regression methods (Clayton and Hills 1993; Preston et al. 1993) in a highly stratified table of cases and person-years. The data were stratified on gender, ethnicity, oblast at time of initial exposure, age at entry (five-year groups to age 70 and 70+), attained age (five-year groups to age 80 and 80+), period of entry in the catchment area (pre-1953 and 1953–1960), time since initial exposure, calendar time, and two-year-lagged cumulative RBM dose. Cohort members were considered to be at risk from the earliest of January 1, 1953, or the date at which they first moved into one of the affected villages until their earliest of the date of any cancer diagnosis, death, migration from the catchment area, or the end of 2005. Person-years were accumulated only during periods in which a person was known to be living in Chelyabinsk or Kurgan Oblasts. The radiation effect was described using excess relative risk models (Preston et al. 1993). Similar models have been used to describe radiation effects on cancer risks in the Life Span Study of atomic bomb survivors (Preston et al. 1994, 2004, 1993), in the Mayak worker cohort (Shilnikova et al. 2003) and in the Extended Techa River Cohort (ETRC) (Krestinina et al. 2005). Baseline rates were allowed to vary with attained age, birth cohort, gender, and ethnicity. Linear, linear-quadratic, and pure-quadratic dose–response models were considered. Gender, age at entry, attained age, and ethnicity were considered as potential dose–effect modifiers. Hypothesis tests were carried out using likelihood ratio tests, and confidence intervals were based on the profile likelihood.

Results

More than 830,000 person-years have accumulated for cohort members residing in Chelyabinsk and Kurgan Oblasts over the 53-year follow-up period. During this time, 93 first primary leukemia cases were identified, including 75 for which an incidence date could be determined and 18 which were identified solely from death certificates.

Table 2 provides summary information on the distribution of cohort members, person-years, and leukemia cases by gender, ethnicity, age at exposure, and time since

Table 2 Selected characteristics of leukemia cases and TRC members:1953–2005

Category	People	PY	Leukemia cases			Rate (per 10,000 PY)	
			Any	DCO ^a (%)	Non-CLL ^b	Any	Non-CLL
Gender							
Male	12,565	336,660	42	26	30	1.25	0.89
Female	17,191	495,821	51	14	40	1.03	0.81
Ethnicity							
Tatar & Bashkir	5,950	200,594	31	6	26	1.55	1.30
Slav	23,806	631,887	62	26	44	0.98	0.70
Age at exposure							
<20	11,769	397,045	40	20	34	1.01	0.86
20–40	9,640	296,550	31	16	20	1.05	0.67
>=40	8,347	138,886	22	23	10	1.58	1.15
Years since first exposure (average age)							
0–5 (32) ^c	^d	68,684	5	0	5	0.73	0.73
–10 (35)		119,439	6	0	5	0.50	0.42
–20 (41)		206,818	23	39	18	1.11	0.87
–30 (48)		171,488	18	22	15	1.05	0.87
–40 (55)		133,216	12	17	5	0.90	0.38
40+ (63)		132,836	29	10	22	2.18	1.66
Total	29,756	832,482	93	19	70	1.12	0.84

Catchment area residents only

PY person-years, DCO death certificate only

^a % death certificate only

^b Leukemias other than chronic lymphocytic leukemia (CLL)

^c Average attained age for this time-since-exposure category

^d Number of people not shown since this is a time-dependent factor

exposure. The crude rates in this table, which do not make allowance for possible dose effects, suggest that rates increase with time or age and are slightly higher for men than for women. Cohort members identified as Tatar or Bashkir ethnicity appear to have higher rates than those identified as Slavs. No leukemia cases were reported among cohort members prior to age 10, and only three cases (one acute and one chronic myeloid leukemia, and one acute leukemia of unspecified cell type) were identified among those aged 10–20 years (data not shown in Table 2).

More than half of the cases (48 cases) were classified as chronic leukemias, 45% (42 cases) as acute or sub-acute leukemias, while the type of the leukemia could not be determined for 3% (3 cases) of the cases (Table 3). Five-year absolute survival rates were low, but this is partly due to the cases diagnosed many years ago when treatment for leukemia was not very sophisticated.

Baseline risks

Log baseline rates for non-CLL were well described using gender-dependent quadratic functions of log-attained age.

After allowing for a linear dose response, there was no significant effect of ethnicity ($P > 0.5$), nor was there any evidence for a log-linear birth cohort effect ($P > 0.5$). Non-CLL baseline rates for men rose fairly rapid with age increasing from about 0.1 cases per 10,000 person-years at age 30 (95% CI 0.04; 0.3) to 0.9 (95% CI 0.4; 1.8) cases at age 70. At these ages, the corresponding baseline rate estimates for women were 0.3 (95% CI 0.1; 0.6) and 0.4 (95% CI 0.2; 0.7), respectively. This gender difference in the temporal pattern of the non-CLL baseline rates was statistically significant ($P = 0.01$).

The log baseline CLL rates could be described by a quadratic function of log-attained age with male rates being 2.1 times the female rates (95% CI 0.9; 4.9), $P = 0.08$). The fitted rates were essentially 0 up to age 35 and peaked around age 70 when the fitted rates were 1.1 for men (95% CI 0.5; 2.0) and 0.5 for women (95% CI 0.2; 0.9), and decreased slightly at older ages. There was no evidence of ethnic differences ($P > 0.5$) or a log-linear birth cohort effect ($P = 0.3$) on the CLL baseline rates. Note that CLL cases make up more than half of the cases diagnosed among cohort members in their 50s and 60s.

Table 3 Distribution of leukemias by histological type: Techa River Cohort 1953–2005

Type	Incident cases	DCO	Total	Mean age at diagnosis	Five-year absolute survival (%) ^a
Acute leukemias					
Myeloid	8	0	8	48	12
Other specified types ^b	3	0	3	63	– ^c
Unspecified cell types	19	12	31	52	6
Chronic leukemias					
Lymphocytic	22	1	23	62	56
Myeloid	22	3	25	57	37
Other or unspecified leukemias					
Myeloid	1	2	3	56	– ^c
Total	75	18	93	55	30

^a Based on non-DCO cases

^b Includes one acute monocytic leukemia and two acute erythremic myelosis

^c Not computed due to small number of cases

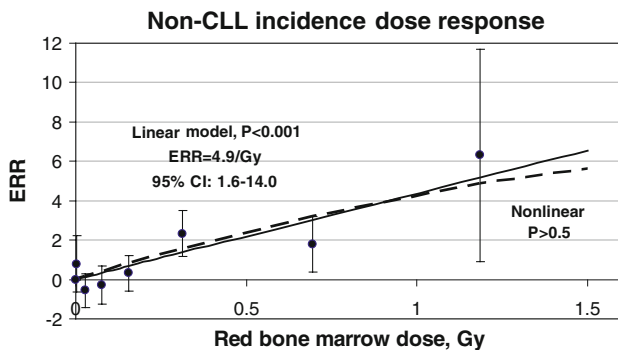


Fig. 1 Non-CLL dose–response function: linear model—solid line, linear-quadratic model—dashed line, non-parametric model—symbols with 95% confidence intervals as error bars

Radiation risk estimates

There was a statistically significant linear dependence on dose for non-CLL ($P < 0.001$). The ERR per Gy estimate was 4.9 with a 95% CI of 1.6 to 14. There was no indication of non-linearity in the non-CLL dose response ($P > 0.5$), and the estimated curvature was close to 0 (-0.12 with a 95% CI $< -0.5; 17$) (Fig. 1). Tests for gender or ethnic differences in the dose response provided no indication of effects, $P > 0.5$. Allowing the dose response to depend on attained age, age at exposure, or time since first exposure did not improve the fit of the model ($P > 0.5$), and the estimates of the changes were small. In contrast, we found no indication of a dose response for CLL ($P > 0.5$) and the ERR per Gy estimate was negative (< -0.2 per Gy) with an upper 95% confidence bound of 1.4.

Table 4 presents the number of person-years and cases in categories of cumulative 2-year-lagged RBM dose. The table also includes estimates of the number of radiation-associated non-CLL cases. It was estimated that 59% (95% CI 32%; 80%) of the non-CLL cases were associated with the radiation exposure. The proportion of radiation-associ-

Table 4 Distribution of observed and expected leukemia cases by red bone marrow dose category

Dose category (Gy)	PY	Non-CLL ^a		CLL cases ^c
		Observed cases	Fitted excess ^b	
<0.01	103,499	6	0	5
–0.1	137,364	3	1	3
–0.2	171,109	8	4	5
–0.5	254,262	31	14	5
–1	140,848	15	17	5
≥ 1	25,399	7	5	0
Total	832,482	70	41	23

CLL chronic lymphatic leukemia, PYR person-years

^a Leukemias other than CLL

^b Excess case estimate from a linear dose–response model with no effect modification. The model does not constrain the fitted number of cases in a given dose category to be equal to the observed number; so the fitted excess can exceed the number of observed cases. For example, the fitted number of cases in the 0.5–1 Gy dose category is 21.5 with 4.5 background cases and 17 excess cases

^c Excess case estimates not shown as there was no evidence of a dose response ($P > 0.5$)

ated cases was higher for those exposed to more than 0.5 Gy.

Discussion

For over 50 years, the TRC of about 30,000 people with low to moderate doses of low-dose-rate to moderate-dose-rate environmental radiation exposure has been followed up to evaluate radiation-related health effects. Our current results demonstrate a statistically significant linear dependence ($P < 0.001$) of the excess relative risk of non-CLL incidence on cumulative dose to RBM with almost 60% of the non-CLL cases in the cohort attributable to radiation exposure. The ERR per Gy of 4.9 for non-CLL in this

analysis was lower, albeit still statistically compatible with the estimate (6.5; 95% CI 1.8; 24) from an early mortality analyses (Krestinina et al. 2005). Baseline rates, both for previous leukemia mortality analyses and for current leukemia incidence analyses in the TRC, have indicated no significant effect of ethnicity ($P > 0.5$), birth cohort ($P > 0.5$), and Kurgan Oblast ($P > 0.5$). Since we are using the same dose estimates and there is little evidence that the ERR is changing with time or age in this cohort, the difference in the ERR estimates is due primarily to the extended follow-up period and the addition of many incident cases. The present analysis includes about 50% more cases than were used in the mortality analyses. The suggestion that the reduction in risk is related to the additional cases is supported by the similar risk estimate (ERR per Gy 4.6; 95% CI: 1.7; 12.3) found in a case–control study of 83 incident leukemia cases conducted in the TRC before TRDS2000 was available (Ostroumova et al. 2006).

Our estimate of the non-CLL ERR at 1 Gy is similar to that seen in the atomic bomb survivors (Preston et al. 1994, Preston et al. 2004) who received acute high-dose-rate exposures. However, the atomic-bomb-survivor dose response has significant upward curvature, and the ERR estimates, based on the survivor data at doses comparable to those received by TRC members, would be somewhat smaller than what is observed in the TRC. These differences can be accounted for by the different nature of the organism's reaction to different exposure doses and dose rates. Thus, at high exposures, the dose–response curve assumes an exponential dependence shape, while at small doses, the response is closer to a linear dependence. In particular, there is practically no difference between leukemia risk values for the TRC calculated using both a linear and those using a linear-quadratic dependence.

Two recent case–control studies of leukemia risk among Chernobyl cleanup workers (Kesminiene et al. 2008; Romanenko et al. 2008) report increased risks for all leukemia as a group with somewhat higher risks for CLL than for non-CLL. The non-CLL ERRs per Gy were 2.73 (95% CI < 0 ; 13.5) for Ukrainian cleanup workers (Romanenko et al. 2008) and 5.0 (90% CI -0.04 ; 57) for workers from Belarus, Russia, and the Baltic countries, which are consistent with what we find for non-CLL in the TRC. However, in contrast to the Chernobyl studies that report ERR/Gy estimates for CLL of 4–5, there is no suggestion of a dose–response both for CLL incidence and for CLL mortality (Krestinina et al. 2005) in the TRC.

Conclusion

For more than a decade, we have been focusing on improving the completeness of follow-up and quality of cause of

death information. Thus, the proportion of individuals with unknown vital status at the end of the follow-up was reduced to 7.5%, and the proportion of deceased cohort members with unknown cause of death was reduced to 10%. These changes led to an increase in statistical power and narrower confidence intervals around the point estimates of risk. Development of a new Techa River dosimetry system is nearing completion. Once the new dose estimates are available, we will update the current risk estimates and conduct analyses of the contribution of external and internal dose to the leukemia risk.

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