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Chernobyl fallout and cancer incidence in Finland 1988–2007

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Twenty-five years have passed since the Chernobyl accident, but its health consequences remain to be well established. Finland was one of the most heavily affected countries by the radioactive fallout outside the former Soviet Union. We analyzed the relation of the estimated external radiation exposure from the fallout to cancer incidence in Finland in 1988–2007. The study cohort comprised all ~3.8 million Finns who had lived in the same dwelling for 12 months following the accident (May 1986–April 1987). Radiation exposure was estimated using data from an extensive mobile dose rate survey. Cancer incidence data were obtained for the cohort divided into four exposure categories (the lowest with the first-year committed dose <0.1 mSv and the highest ≥0.5 mSv) allowing for a latency of 5 years for leukemia and thyroid cancer, and 10 years for other cancers. Of the eight predefined cancer sites regarded as radiation-related from earlier studies, only colon cancer among women showed an association with exposure from fallout [excess rate ratio per increment in exposure category 0.06, 95% confidence interval (CI) 0.02–0.11]. No such effect was observed for men, or other cancer sites. Our analysis of a large cohort over two decades did not reveal an increase in cancer incidence following the Chernobyl accident, with the possible exception of colon cancer among women. The largely null findings are consistent with extrapolation from previous studies suggesting that the effect is likely to remain too small to be empirically detectable and of little public health impact.

The nuclear power plant accident in Chernobyl on April 26, 1986, resulted in radiation exposure of several hundreds of thousands of people of Belarus, Ukraine and South-Western Russia in the vicinity of the reactor.[1] In public health terms, this is the largest radiation accident ever and the major challenge for radiation epidemiology in past decades. Approximately 115,000 people were evacuated from areas in the vicinity of the plant with an average effective dose 31 mSv during the 20 years after the accident.[1] A total of 6.4 million people residing in the contaminated areas (defined as ^{137}Cs deposition >555 kBq/m^2) were estimated to receive a mean effective dose of 9 mSv during the same period.[1] Notably, the radiation dose to the thyroid from internal exposure to radioiodine was substantially higher, particularly among children. The most important health effect of the accident has been the dramatic, up to 5–10 fold increase in thyroid cancer in the exposed population, primarily those exposed in childhood.[1,2] More than 6,800 thyroid cancer cases have been diagnosed in 1991–2005 among people aged <18 years at the time of the accident in Belarus, Ukraine and south-western parts of Russia.[1] No consistent evidence of increases in other cancer types has been found, though some reports have suggested increases in leukemia and breast cancer.[3–5] Among the nearly 0.5 million clean-up workers involved in the recovery operations, some suggestions of increased leukemia and thyroid cancer have been reported, but the evidence is not conclusive.[6–9] The recent nuclear power plant accident in Fukushima in Japan has heightened the interest on the issue again.[10]

The research efforts have understandably concentrated on the most heavily exposed populations including several hundred thousand clean-up and recovery workers dispatched to the area around the reactor in 1996–2001 (with mean doses of the order of magnitude of 0.1 Sv), as well as the ~5 million residents of the contaminated areas (mean doses from external radiation during the first postaccident year close to 1 mSv) adjacent to the site in Belarus, Ukraine and some parts of Russia. However, practically no systematic large scale studies with long-term follow-up have evaluated the cancer risk

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What's new?

While radiation exposure from the 1986 Chernobyl accident led to dramatic increases in thyroid cancer among people living within the vicinity of the reactor, health consequences for populations outside the former Soviet Union remain unclear. Here, the effect of Chernobyl fallout over a period of nearly 20 years in Finland was investigated using individual-level residential and cancer data. Only a single malignancy, colon cancer in women, was found to be associated with radiation exposure from the accident. The findings support current thinking that the possible impact on cancer incidence at the population level is likely to remain small.

resulting from the low-level exposures outside the former USSR.[11] One should bear in mind that it also took several decades to establish the excess risk of solid cancers among Hiroshima and Nagasaki survivors.

No consistent evidence has been found for an increase in cancer in more distant populations (outside Belarus, Ukraine and south-west Russia) in the early studies.[1] Some studies have suggested increased cancer rates, but in most studies the results have been negative.[12–16] However, projections based on theoretical calculations using the dose information, population size and radiation risk coefficients have suggested that within the first 20 years since the accident, besides the thyroid cancers, about 1800 other solid cancer cases could be anticipated in the three most heavily exposed countries.[17] Furthermore, in the other European countries with only minor doses (<1 mSv), a similar number of excess cases would be expected, due to the very large population exposed (approximately 550 million). Yet, the attributable fraction would remain very small (of the order of 0.01% for solid cancers and 0.05% for leukemia).[17] However, as the expected effects are very small, a large study population and accurate exposure estimates would be needed even to exclude a risk substantially larger than that predicted. Nevertheless, surveillance of cancer incidence trends is warranted to assess the full public health impact of the Chernobyl accident. For that purpose, the absence of a detectable increase is informative, even if the resolution would not be sufficient for demonstrating very small effects.

The mean Cs-137 deposition from the Chernobyl fallout in Finland was 12 kBq/m^2, which is the highest outside the former Soviet Union together with Austria and Slovenia.[18] There was also variation within Finland, with about 12,000 km^2 receiving deposition in excess of 37 kBq/m^2.

The aim of our study was to perform cancer risk assessment of the effect of radiation from Chernobyl fallout in Finland, with estimation of the size of the possible effect. Specifically, we divided the Finnish population into four exposure strata and estimated cancer incidence trends during 1988–2007 in relation to radiation exposure from the Chernobyl fallout. Exposure assessment was based on deposition in the country divided into four regions, separately for houses and blocks of flats. To enhance the validity of exposure estimates, we restricted the analysis to those 3.8 million subjects who had lived in the same residence in the first post-Chernobyl year (May 1986–April 1987), that is, during the period when the most intensive radiation exposure occurred.

Material and Methods

We conducted a cohort study of the residentially stable Finnish population (~3.8 million people, 90% of the total) identified from Statistics Finland, with internal comparisons based on subdivision of the population into four exposure groups. The study included all Finns, who resided in the same dwelling between May 1986 and April 1987, that is, did not move during the first year after the Chernobyl accident. This restriction was used to improve exposure assessment, that is, maximize the variation between strata and minimize it within them. No other inclusion or exclusion criteria were applied.

Exposure assessment was based on Geiger-Müller tube and spectrometric measurements of dose rates from radioactive caesium (^{134}Cs and ^{137}Cs). The measurements in 1,050 locations were performed in a mobile survey between May 1986 and August 1987.[19] The contribution of all relevant short-lived nuclides was determined from spectrometric results. The effect of delay in the measurements was eliminated by using a back-calculation technique that takes into account both the radioactive decay and the washout effect. The influence of fallout from atomic bomb tests in the 1950s and 1960s was eliminated by calculating the calibration factor from the ^{134}Cs deposition. The population doses were mainly caused by ^{137}Cs and other volatile nuclides. Effective doses from external radiation were estimated for each 8 × 8 km square taking into account the deposition and the shielding of buildings against radiation [depending on house type: 0.47 for houses (including detached, semidetached and terraced houses) and 0.18 for blocks of flats]. Four zones with contrasting patterns of deposition were defined, to form dose bands with equidistant cut-points (<0.10, 0.10–0.29, 0.30–0.49 and ≥0.50 mSv during the first postaccident year; Table 1). Other sources of radiation exposure (mainly from natural sources and medical uses of radiation) were assumed to have changed in a similar fashion in each area after the accident (*i.e.*, result in nondifferential misclassification).

Aggregate data on cancer incidence for small area units consisting of 250 × 250 m and covering all of Finland were used. The numbers of person-years (denominator data) were obtained for each square from Statistics Finland by 5-year age group, sex, house type and socioeconomic status (SES)

based on occupation and education from the Population Census 1985 (classified as: farmers, foresters, fishermen; other employers and self-employed persons; upper clerical workers; lower clerical workers; skilled manual workers; unskilled manual workers and others).

The 459,622 cancer cases that 1987–2007 in the study cohort (numerator data) from the Finnish Cancer Registry were linked in the Statistics Finland by personal identity code with the data on sex, age, place of residence (in May 1986), house type and SES. An anonymized database with numbers of cases for 250 × 250 m map squares was generated for the analysis.

The exposure strata were of uneven size, the lowest comprising more than half of the study population (59%) and the highest only 4% (Table 2 and appendix Tables A1–A3). The numbers of person-years for the three lowest exposure strata in each five-year period ranged from 11 million to 1.7 million (men and women, houses and blocks of flats combined), but were substantially smaller for the highest exposure group (640,000–540,000 both sexes, houses and blocks of flats combined, Table 2). Due to the lower exposure levels in blocks of flats, the highest exposure group included practically only people living in houses (Figs. 1*a* and 1*b*). Consequently, no cancer cases were observed in the highest exposure group residing in blocks of flats (Table 3). For the other strata, the median number of cases for the second highest exposure group was ∼10 for blocks of flats for both men and women and was substantially larger for the strata with lower exposure. Therefore, analyses were conducted for houses and flats combined and separately for houses.

Table 1. Exposure categories used in the analysis (dose conversion shown for houses)

Exposure category	Cs-137 deposition (kBq/m^2)	Effective dose[1] (mSv)	Population size[2]
1	<9	<0.1	2,255,716
2	9–27	0.1–0.29	1,070,873
3	28–45	0.3–0.49	350,584
4	≥46	≥0.5	132,551

[1]Dose from external radiation for inhabitants of houses during the first postaccident year (May 1986–April 1987).
[2]Numbers of subjects at start of follow-up (houses and blocks of flats, men and women, all age groups combined).

In the analysis by cancer site, we concentrated on cancer types strongly associated with radiation in previous studies (leukemia, cancers of the ovary, oesophagus, colon, breast, bladder, stomach and thyroid cancers), but also included some "control" sites not consistently associated with radiation in earlier studies (lymphomas, rectal and pancreatic cancer). A meaningful analysis of childhood leukemia was not possible due to the small numbers of cases at follow-up (18 cases in 1998–2002 and two in 2003–2007).

The numbers of cancers and person-years at risk were stratified by gender, age attained, SES and house type. A minimum latency period of five years was assumed for leukemia and thyroid cancer and 10 years for other sites. Thus, the calendar period 1988–1997 was considered the latency period unaffected by the fallout (and used only in the baseline trend and level evaluation) for most cancer types, with

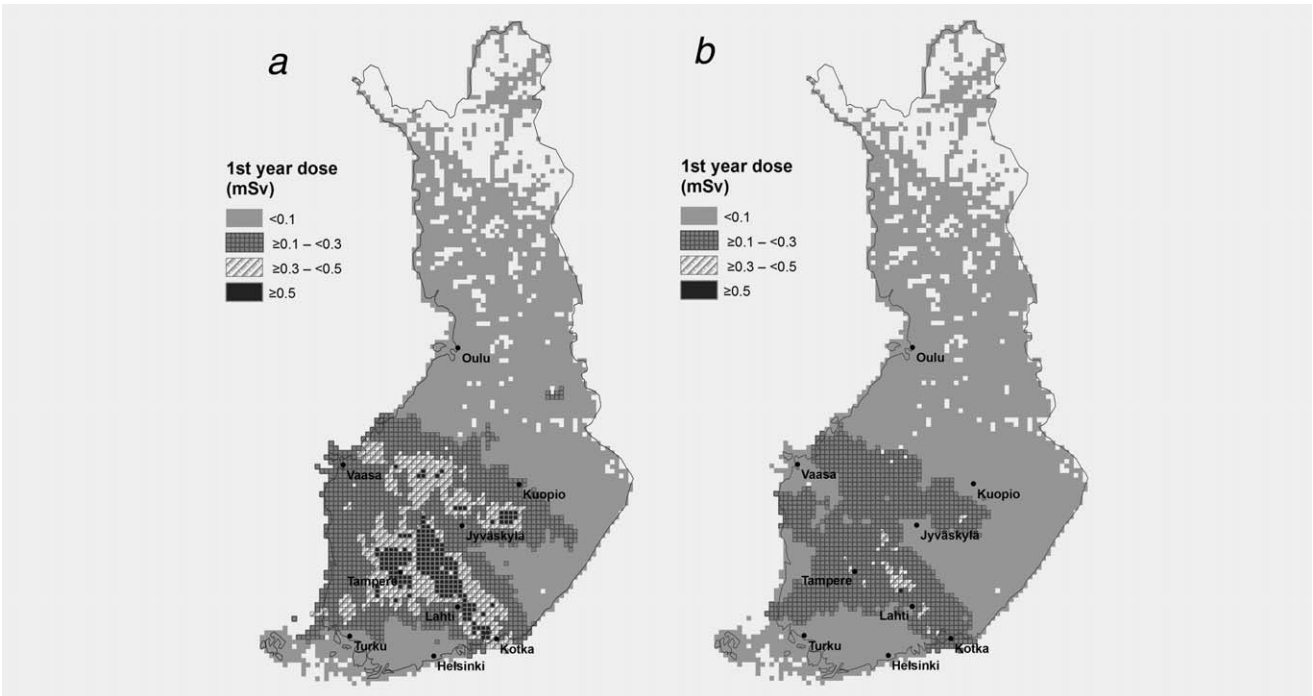

Figure 1. Dose distribution from the Chernobyl fallout in Finland for people residing in (a) houses and (b) blocks of flats.

Table 2. Numbers of person-years by sex, period, house type and exposure category (first year dose, mSv)

	Men				Women			
	1988–1992	1993–1997	1998–2002	2003–2007	1988–1992	1993–1997	1998–2002	2003–2007
Houses								
<0.1 mSv	2,994,300	2,839,864	2,677,365	2,521,231	2,909,381	2,781,300	2,640,070	2,501,507
0.1–0.29 mSv	1,974,224	1,863,738	1,752,072	1,645,296	1,951,142	1,853,280	1,748,575	1,646,706
0.3–0.49 mSv	797,780	750,157	701,830	656,443	785,416	744,161	700,046	656,780
≥0.5 mSv	325,330	307,374	288,876	270,741	322,566	306,625	289,488	272,674
Blocks of flats								
<0.1 mSv	2,273,939	2,136,768	2,000,659	1,887,609	2,833,619	2,651,250	2,458,032	2,313,824
0.1–0.29 mSv	573,176	534,008	496,088	464,356	721,682	670,017	614,803	573,926
0.3–0.49 mSv	58,836	55,696	52,454	49,581	68,652	64,979	61,099	57,602
≥0.5 mSv	25	25	21	20	15	15	15	15

the postlatency period starting from 1998, but a shorter latency period (1988–1992) was applied for leukemia and thyroid cancer.

The number of cancer cases was described by the Poisson distribution, where the logarithm of the incidence rate λ was modelled as a linear function of attained age (i, five-year age groups up to 84 and ≥85 years), SES (s), calendar year (v) and exposure group (e, $e = 1$, if exposure < 0.1 mSv; $e = 2$, if $0.1 \leq$ exposure < 0.3; $e = 3$, if $0.3 \leq$ exposure < 0.5; and $e = 4$, if exposure ≥ 0.5). In the main analysis, also house type (h, houses and blocks of flats) was used, but an additional analysis included only houses. The main model can be written as:

$$\log \lambda_{\text{ishev}} = \alpha_i + \eta_s + \delta_h + \varepsilon_e + \beta v + \tau_e I_v$$

where the period of observation was divided into the latency and post-latency period using an indicator variable I_v that equals 1 in the period beyond latency time and 0 otherwise. In the main model (illustrated for the two lowest exposure groups in Fig. 2, left panel), the slope β of the logarithm of the incidence rate was assumed to be the same in all the exposure strata. In addition, an alternative model (Fig. 2, right panel) where β_e is estimated separately for each exposure group e (instead of a common β), allowing the slopes of the logarithms of the incidence rates to vary between the exposure groups, was fitted. In both models, rate ratio $(RR_e) = \exp(\tau_e)$ is the RR between the incidence rate in exposure group e in the postlatency period and the expected one that would have been observed, if the intercept of the logarithm of the incidence rate remained unchanged after the latency period. To test for the trend in the RRs the effect of the increasing exposure was modeled using a numerical exposure variable e such that $\tau_e = \tau_1 + \gamma(e - 1)$. In the models with the numerical exposure variable, excess rate ratio $(ERR) = \exp(\gamma) - 1$ is the excess RR per an increasing exposure unit, that is, the RRs in exposure groups 2, 3 and 4 are $\exp(\gamma)$, $\exp(2\gamma)$ and $\exp(3\gamma)$-fold, respectively, as compared with the RR in exposure group 1. The four models (main and alternative model, both with categorical and numerical exposure) were compared, and the heterogeneity and the trend in the RRs were tested using the likelihood ratio test.

A permission to use the cancer registry data was issued by the National Institute for Health and Welfare. The ethical committee at STUK–Radiation and Nuclear Safety Authority was informed of the study protocol. No confidentiality issues arose, as no individual-level data were used and the units of observation were too large for identifying individuals.

Results

The study covered more than 15 million person-years of observation during each 5-year period (Table 2 and appendix Tables A1a–A3b), with more than 400 cancer cases by period for each studied cancer site when all exposure categories, men and women and both house types were combined (Table 3). For the analysis restricted to houses, the overall numbers were smaller by approximately a half, but the numbers in the highest exposure groups were hardly diminished.

Of the six cancer types analyzed in both sexes and the two female cancer sites a priori selected as potentially radiation-induced, only colon cancer among women showed an association with the level of exposure from the Chernobyl fallout (Table 4). In the trend analysis of the entire cohort across the four exposure groups, the ERR for female colon cancer per increment in exposure category was 0.06 (95% CI 0.02–0.11), that is, the RR in the highest exposure group was $1.06^3 - 1 = 19\%$ larger than that in the lowest exposure group. Alternatively, this result across the four exposure groups can be interpreted as 1.19-fold increase in incidence in the highest exposure stratum, with the lowest exposure group as reference, after the latency period. In the highest exposure group, an incidence rate ratio (RR) of 1.11 (95% CI 0.90–1.37) was found for female colon cancer, that is, a 11% increase in the observed relative to the expected incidence rate for the highest exposure group after the latency period. An analysis limited to houses (with higher exposure and less

Table 3. Numbers of cases by period, sex, house type, cancer site and exposure category

	1988–1992				1993–1997				1998–2002				2003–2007			
	<0.1	0.1–0.29	0.3–0.49	≥0.5	<0.1	0.1–0.29	0.3–0.49	≥0.5	<0.1	0.1–0.29	0.3–0.49	≥0.5	<0.1	0.1–0.29	0.3–0.49	≥0.5
Men																
Houses																
Leukemia	318	223	89	38	330	226	97	33	350	238	93	43	388	226	113	35
Esophagus	125	113	47	15	136	101	41	11	191	107	59	19	170	121	70	23
Colon	513	366	161	61	640	499	228	64	726	591	196	84	914	676	286	103
Bladder	628	464	195	72	804	569	225	78	795	549	212	69	850	557	208	87
Stomach	662	535	240	79	642	477	194	68	580	396	169	71	481	431	162	55
Thyroid	88	48	20	8	119	60	34	9	112	66	25	10	133	56	34	10
Hodgkin	81	65	31	13	94	68	26	6	82	72	25	10	83	51	29	13
Non-Hodgkin	401	290	119	57	541	346	137	45	627	419	164	64	732	481	172	53
Rectum	359	277	111	53	434	304	136	72	480	376	150	65	573	397	162	82
Pancreas	362	283	133	45	457	347	117	44	511	330	129	67	593	391	163	58
Blocks of flats																
Leukemia	266	53	11	0	236	74	4	0	271	69	4	0	271	56	5	0
Esophagus	124	38	5	0	142	33	4	0	146	52	8	0	170	44	7	0
Colon	523	138	12	0	628	143	15	0	661	189	12	0	728	199	16	0
Bladder	612	158	11	0	616	163	14	0	637	154	12	0	611	159	11	0
Stomach	536	148	11	0	463	116	7	0	357	106	11	0	365	94	9	0
Thyroid	56	13	2	0	80	14	2	0	76	14	4	0	90	14	3	0
Hodgkin	53	17	1	0	74	19	3	0	61	19	3	0	62	12	1	0
Non-Hodgkin	323	92	11	0	481	110	5	0	448	101	14	0	510	110	11	0
Rectum	360	117	8	0	351	101	8	0	386	104	7	0	474	114	15	0
Pancreas	366	88	6	0	351	92	10	0	369	87	8	0	469	114	7	0
Women																
Houses																
Leukemia	243	162	78	32	262	187	62	29	255	187	78	28	257	154	80	36
Breast	2852	1897	842	313	3389	2310	1008	419	4021	2804	1158	489	4411	3011	1217	540

Table 3. Numbers of cases by period, sex, house type, cancer site and exposure category (Continued)

	1988–1992				1993–1997				1998–2002				2003–2007			
	<0.1	0.1–0.29	0.3–0.49	≥0.5	<0.1	0.1–0.29	0.3–0.49	≥0.5	<0.1	0.1–0.29	0.3–0.49	≥0.5	<0.1	0.1–0.29	0.3–0.49	≥0.5
Ovary	474	366	139	56	553	406	163	74	511	360	151	59	519	380	176	71
Esophagus	101	94	25	12	99	83	28	16	92	65	23	5	78	69	23	14
Colon	586	486	193	88	700	574	248	82	754	629	274	109	790	739	315	121
Bladder	168	125	51	15	198	135	52	19	198	176	56	23	225	136	58	20
Stomach	471	402	183	69	422	365	152	47	397	321	136	42	342	283	131	48
Thyroid	321	172	63	28	393	218	83	31	316	220	84	44	373	194	78	35
Hodgkin	56	33	22	9	75	50	13	10	78	64	22	9	56	39	25	7
Non-Hodgkin	381	272	112	41	481	360	137	49	501	402	175	53	566	426	174	55
Rectum	268	249	101	46	354	260	101	39	377	278	124	33	362	320	131	57
Pancreas	396	294	109	47	420	335	140	52	468	374	129	45	579	411	183	56
Blocks of flats																
Leukemia	272	79	3	0	269	78	5	0	310	87	8	0	258	67	8	0
Breast	3816	964	79	0	4247	1082	93	0	4990	1155	102	0	5460	1263	111	0
Ovary	630	149	21	0	600	175	13	0	555	156	14	0	565	158	15	0
Esophagus	132	34	5	0	113	47	3	0	120	34	2	0	96	34	4	0
Colon	913	275	17	0	979	248	21	0	1006	277	14	0	1006	239	17	0
Bladder	282	71	2	0	317	74	4	0	284	69	7	0	287	57	4	0
Stomach	619	211	17	0	579	162	10	0	421	119	8	0	368	111	13	0
Thyroid	267	53	10	0	296	74	8	0	274	64	7	0	273	71	4	0
Hodgkin	64	14	3	0	69	17	1	0	47	16	2	0	40	19	1	0
Non-Hodgkin	514	135	10	0	573	161	10	0	629	148	14	0	653	170	16	0
Rectum	408	122	12	0	437	136	12	0	446	92	8	0	456	115	14	0
Pancreas	600	155	19	0	616	151	7	0	617	140	12	0	658	175	10	0

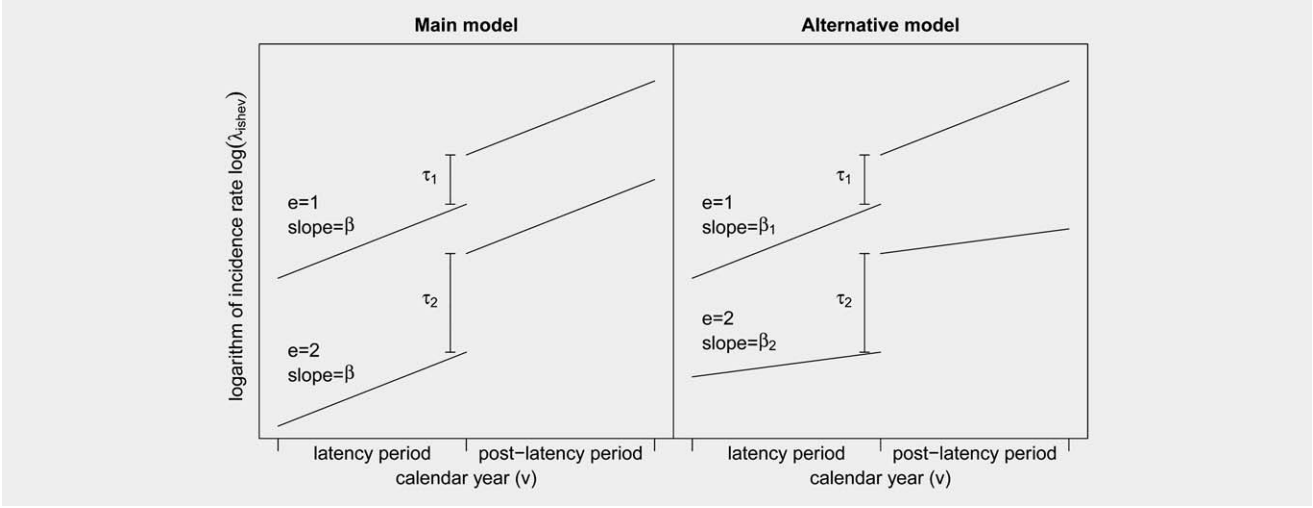

Figure 2. A schematic representation of the statistical models applied in the data analysis for the two lowest exposure groups. e: exposure group, tau: change in log incidence rate from the latency to the post-latency period.

variation than in blocks of flats) showed largely comparable results for female colon cancer (RR = 1.03 for the highest exposure group, CI 0.83–1.28, Table 4). No such association was found among men (RR = 1.03, 95% CI 0.98–1.08 for trend across exposure categories and RR = 1.06 for the highest exposure group). A weak, non-significant positive relation was observed also for female thyroid cancer, with a similar finding observed in the analysis restricted to houses, but little evidence for such relation among men.

None of the sites previously showing no consistent association with radiation exposure ("control sites") was associated with the level of exposure. Very little variation with exposure was found for rectum and pancreas cancers, and Hodgkin lymphoma showed a weak positive association with exposure level.

The main model provided a better fit for all cancer sites except ovary. The alternative model that did not assume proportional incidence trends between the exposure groups during the latency and postlatency periods within each exposure group, that is, did not assume the same β_e for all exposure groups (illustrated in Fig. 2, right panel), provided a better fit for ovarian cancer. The results of these analyses were qualitatively similar in that they did not show an increase in relation to exposure level. The RR point estimates varied more across the exposure groups and point estimates for the ERR deviated more from zero in the analysis based on the alternative model (results not shown).

A population-based colorectal cancer screening program was launched in Finland gradually from 2004 onwards, and the areas first covered where in the regions that were mainly in the highest exposure category. The observed effect for female colon cancer was not substantially affected by limiting the analyses to the period prior to introduction of screening (up to the year 2003) or excluding the target age group or municipalities where screening was introduced (ERR = 0.07, 95% CI 0.02–1.12 for houses and block of flats, ERR = 0.09, 95% CI 0.03–0.15 for houses only).

Analyses with a shorter 5-year latency for solid cancers gave largely consistent results, but the risk related to female colon cancer was smaller and no longer significant (RR across the four categories 1.04, 95% CI 0.98–1.10 and RR in the highest exposure group 0.97, with similar findings in analysis restricted to houses only). In addition, a positive association emerged with female breast cancer (RR = 1.04, 95% CI 1.01–1.07 across the four categories and RR = 1.18 in the highest exposed group) and an inverse association between radiation exposure and non-Hodgkin lymphoma in men (both in the entire material and houses only).

An analysis by age at exposure was also conducted and showed no evidence of effect modification for the sites previously associated with radiation (all likelihood ratio *p*-values >0.2 for an interaction term of age and exposure), though the numbers of cases in the age group 0–19 years were so small that the statistical power was low (<10 cases in the highest exposure group in all 5-year periods in each site, except breast and Hodgkin disease <20 cases).

Discussion

We found little evidence overall for an increased incidence of cancer in relation to Chernobyl fallout in Finland with a study design using individual level residential and cancer data combined with area-level exposure assessment. Even though our results can exclude only an effect several orders of magnitude larger than anticipated, they can be regarded as informative for evaluation of the public health impact of the Chernobyl accident and contributing to knowledge about the health consequences of the Chernobyl accident. The strengths of the study include a reasonably long observation period of 20 years, large study population of nearly four million people

Table 4. Incidence rate ratios (RRs) between the incidence rates in the postlatency period 1998–2007 (1993–2007 for leukemia and thyroid) and the expected ones based on the trends in 1988–1997 (1988–1992 for leukemia and thyroid) by sex, house type, cancer site and exposure category (RR_1 for the lowest exposure category and RR_4 for the highest)

	Men						Women					
	RR_1	RR_2	RR_3	RR_4	ERR, %	95% CI	RR_1	RR_2	RR_3	RR_4	ERR	95% CI
Blocks of flats and houses												
Sites associated with radiation												
Leukemia	0.96	1.01	0.98	0.88	0	(−9, 9)	1.09	1.11	1.02	0.99	−2	(−11, 7)
Breast	–	–	–	–			1.03	1.07	1.04	1.11	2	(−0.4, 4.0)
Ovary	–	–	–	–			0.87	0.91	0.99	0.91	4	(−2, 10)
Esophagus	1.13	1.04	1.35	1.43	6	(−5, 17)	0.98	0.89	0.94	0.73	−7	(−18, 7)
Colon	0.94	1.07	0.90	1.06	3	(−2, 8)	0.96	1.03	1.09	1.11	6	(2, 11)
Bladder	0.96	0.96	0.89	0.91	−2	(−7, 3)	1.00	1.06	1.08	1.17	5	(−4, 16)
Stomach	0.93	1.00	0.95	1.03	3	(−2, 9)	1.02	1.03	1.09	1.04	2	(−4, 9)
Thyroid	1.34	1.18	1.50	1.16	−1	(−17, 18)	1.12	1.30	1.25	1.35	8	(−2, 19)
Sites not associated with radiation												
Hodgkin lymphoma	0.73	0.71	0.74	0.93	3	(−11, 19)	0.97	1.40	1.47	0.95	15	(−2, 35)
Non-Hodgkin lymphoma	0.94	0.97	0.97	0.82	0	(−6, 6)	0.96	1.01	1.13	0.94	4	(−2, 11)
Rectum	0.98	0.99	1.01	0.91	0	(−6, 6)	0.93	0.90	1.02	0.86	0	(−7, 7)
Pancreas	0.98	0.91	0.92	1.09	−1	(−7, 5)	0.95	0.99	0.99	0.81	0	(−6, 6)
Houses only												
Sites associated with radiation												
Leukemia	1.00	0.96	1.07	0.89	−1	(−10, 10)	1.05	1.11	0.96	0.98	−2	(−13, 9)
Breast	–	–	–	–			1.01	1.08	1.02	1.09	2	(−1, 5)
Ovary	–	–	–	–			0.84	0.84	0.97	0.87	4	(−3, 11)
Esophagus	1.24	1.00	1.40	1.50	4	(−8, 17)	0.90	0.84	0.98	0.75	−2	(−17, 15)
Colon	0.94	1.02	0.88	1.03	1	(−5, 7)	0.88	0.99	1.04	1.03	8	(2, 14)
Bladder	0.93	0.91	0.87	0.88	−2	(−8, 3)	1.07	1.17	1.10	1.22	4	(−8, 16)
Stomach	0.93	0.98	0.93	1.02	2	(−4, 8)	1.07	1.06	1.09	1.04	0	(−7, 8)
Thyroid	1.30	1.22	1.52	1.16	1	(−17, 24)	1.13	1.26	1.36	1.35	8	(−3, 21)
Sites not associated with radiation												
Hodgkin lymphoma	0.74	0.73	0.76	0.96	5	(−11, 23)	1.25	1.53	1.67	1.03	5	(−13, 26)
Non-Hodgkin lymphoma	1.02	1.05	0.99	0.84	−3	(−10, 3)	0.91	1.01	1.10	0.92	6	(−2, 14)
Rectum	0.98	1.03	1.00	0.90	−1	(−7, 6)	0.92	0.96	1.05	0.86	2	(−6, 11)
Pancreas	1.01	0.90	0.94	1.09	−1	(−8, 6)	0.94	0.96	0.98	0.78	−1	(−8, 6)

Excess RRs (ERR, %) per incremental exposure category are also shown.

and consideration of other factors, such as SES, house type and residential stability.

Only the analysis of colon cancer among women showed an association between radiation exposure and incidence in the postlatency period. A similar finding was also seen in the subset of people residing in houses, but no such result was observed for men. A possible confounding factor was colorectal cancer screening with occult fecal blood test introduced in Finland during the study period, and it was first commenced in the area mainly belonging to the highest exposure group, with gradual extension to cover 40% of the country by 2008.[20] Restricting the analysis to the unscreened population (in terms of area or age group, or period) did not materially affect the findings, which indicates that the result was not due to screening. Other potential confounding factors include dietary factors (fruit and vegetables/fiber *vs.* fat), physical exercise (obesity), alcohol, smoking, NSAIDs, menopausal hormone use. We had no information on them, but they would confound the results only if changes occurred in them contemporaneously with the fallout and with a similar geographical pattern.

The effect size corresponds to a 1.19-fold increase in the highest exposure group relative to the lowest. The committed effective doses from external radiation over 20 years can be

roughly estimated as <1 mSv in the lowest versus 5 mSv in the highest exposure category. This corresponds roughly to an ERR of 0.04 per mSv or 40 per Sv, which is clearly inconsistent with for instance the results of the Life Span Study of the atomic bomb survivors (ERR 0.5 per Sv for colon cancer),[21] which is considered the most important source of information regarding long-term health effects of ionizing radiation. Further, colon was one of the few cancer sites with a higher risk coefficient for men than women among the atomic bomb survivors.

This finding could also be attributable to chance, which is also credible given that 14 site and gender strata were analyzed as a priori radiation-related (the probability of detecting at least one statistically significant finding out of 14 tests by chance alone is $1 - 0.95^{14} = 0.51$, provided that the comparisons are independent). Yet, none of the four cancer sites with little previous evidence of being radiation-induced (used as "negative controls") showed an association with the exposure.

A quarter of a century has passed since the Chernobyl accident. Nevertheless, the health effects remain to be characterized in detail and we are only approaching the stage, when large scale data about cancer incidence following the latency period become available. To some extent, this is attributable to the low doses and consequently small increases in morbidity, strongly limiting the possibilities of epidemiological studies to detect an increase—or exclude an effect of the size predicted from other studies. The typical cumulative effective doses (~0.3 mSv for 1986–2005) for the Western European populations were comparable to a single radiographic examination such as two projections in mammography or three chest x-rays with two projections and less than the dose from a single CT scan.[1]

The expected magnitude of effects can be extrapolated from the results of studies on higher doses. Among the atomic bomb survivors, the effect size for solid cancers have been approximately ERR 0.5 per Sv.[21] The mean committed effective dose in 1986–2005 for the Finnish population has been estimated as 1.4 mSv[1], and for the highest exposure group in this study, the committed effective dose is likely to be of the order of magnitude of 5 mSv (given that the first-year dose in this group was 3.5-fold higher than the population-weighted mean dose in the entire population). This is a crude assessment relying on the assumption that due to the lower contribution of short-lived radioisotopes in Finland, the dose committed during the 20-year period was 3–5 times the first-year dose, while the corresponding ratio for the areas close to the accident site estimated as three by UNSCEAR. Such dose would translate into an excess cancer incidence of <0.05% (RR of <1.0005), assuming similar effect per dose unit as among atomic bomb survivors (despite differences in exposure features such as dose rate and contribution of internal exposure). The nominal risk coefficient proposed by the International Commission for Radiological Protection (lifetime absolute risk of 5.5×10^{-2} for cancer death) can also be used to obtain a crude measure of the anticipated effect. Applying this figure to the collective dose in the cohort (~500 person-sievert for the first-year dose, up to 2000 person-sievert taking into account internal exposure and long-term committed dose) would give 30–120 additional cancer deaths over a period of some 50 years. For cancer incidence, the figures would be higher by a factor of three, but still only a tiny fraction of the background risk. Such effects are likely to remain below the threshold of detectability for any epidemiological study.

Our results are consistent with the majority of earlier studies assessing the effect of the Chernobyl fallout on cancer incidence in Europe.[12–16] The findings are in contrast with those reported from Sweden suggesting an early increase in cancer incidence[22] (although the study periods do not overlap). That study reported an increase in overall cancer incidence in the area most heavily affected by the Chernobyl fallout in 1988–1999. The authors interpreted the findings as indicating a promotion effect, but the approach has been criticized for methodological shortcomings, including ignoring underlying trends and prior differences in rates. Based on the results of the studies on Hiroshima and Nagasaki atomic bomb survivors, the increase in solid cancer risk is expected to remain elevated for at least 2–3 decades since its first emergence, though with some attenuation.[21]

Our study has notable advantages compared with earlier ones. We were able to establish a nation-wide cohort of population remaining resident in the same dwelling for the time period when the most intensive exposure took place, which improved the validity. The dose rate at 12 months from the accident was only about one tenth of the initial level. In addition, the follow-up for cancer incidence was conducted at individual level and therefore cases (as well as person-years) were allocated to the correct exposure group regardless of subsequent eventual moving, which avoids misclassification and ecological fallacy. The large size of the study population and long follow-up enhanced the statistical power, though in respect to the anticipated effect size it remained modest.

Some obvious limitations, however, restrict the conclusions. Ideally, exposure assessment would be performed using personal dosimeters for external dose and whole body counting for internal exposure. This is, however, clearly not feasible for any population of meaningful size for epidemiological studies. External dose is a function of the deposition, with modification by protection afforded by shielding of the buildings. The deposition can be expected to remain reasonably homogenous within the area units of 8×8 km, compared with large geographical units used in previous studies. Shielding was accounted for by the proportion of single-family houses and other low-rise residential buildings within a square. The shielding factor for radiation due to fallout was defined as the ratio of the dose rate indoors to the outdoor dose rate. This gives typical values for the small area, though with some misclassification due to aggregation to group level. Further variation that we could not account for was due to

occupancy (time spent indoors) and exposure outside the home (both at work and during leisure time, including time spent in other buildings and travel outside the home district). Even if we had to resort to averaging over a small area with group-level data, we were able to use smaller units of observation in exposure assessment than in any earlier epidemiological study on the health effects of Chernobyl fallout. Therefore, dilution owing to non-differential exposure misclassification should have less effect in our study.

We did not have information on internal radiation exposure, which resulted mainly from ingestion of food stuffs contaminated by radionuclides, primarily caesium-137. In the area with the highest fallout, milk products and fish were the most important sources, and elsewhere also meat and eggs.[23] Radioiodine did not contribute substantially, as much of it decayed already before reaching Finland due to its short half-life, and intake through milk was also reduced by the fact that the cows were not yet out on pastures in Finland at the time of the accident.[23] The internal dose was 0.3 mSv for the first year in the entire population (and below 0.5 mSv even in the areas with the highest fallout), and declined rapidly thereafter.[24] The mean committed effective dose from ingestion of radionuclides from the Chernobyl fallout for the Finnish population during 1986–2005 has been estimated as 1 mSv.[23,24] The internal dose was correlated with external radiation dose, but with less marked contrast between areas due to the fact that most consumed food is not produced locally.[12] If incremental dose from internal exposure, which is correlated with the external dose but has less steep gradient across the population, is taken into account in the dose-response analysis, it would increase the constant (exposure level in the reference category), but slightly flatten exposure contrast and hence observed the slope (regression coefficient) could slightly underestimate the true exposure-effect gradient.

Obviously, individual-level information on the major lifestyle determinants of cancer risk would have been desirable for control of confounding, as in any epidemiological study. Such data were unavailable in our study and we are unaware of any cohorts of this size with such information. We were, however, able to control for the effects of age, sex, SES and house type. Besides confounding, other sources of radiation exposure may cause exposure misclassification, which can mask an effect. However, in our longitudinal study, differences between exposure groups that remained unchanged would not bias the results, as they are incorporated in the underlying trend (of the background incidence rates). Therefore, for instance differences in natural background radiation between exposure groups would not affect our findings. Only systematic differences in relation to fallout over time (*i.e.,* diverging trends in exposure from other sources) could bias the findings related to the effects of the fallout.

In conclusion, we were unable to demonstrate an increase in cancer incidence in Finland related to the fallout from Chernobyl. We were able to construct sub-groups with a relative exposure contrast by a factor of five, but the absolute differences in radiation doses were very small and the overall dose level was low. The strengths of the study include larger size and longer follow-up than in earlier studies, as well as restriction to a residentially stable cohort, which improved exposure estimation. However, we could not exclude a small effect of the size expected on the basis of existing risk estimates (of the order of magnitude of 1% increase in cancer incidence), as the statistical power was limited by the low exposure levels.

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