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Increased Incidence of Malignancies in Sweden After the Chernobyl Accident—A Promoting Effect?

Martin Tondel,[1]* Peter Lindgren,[1] Peter Hjalmarsson,[2] Lennart Hardell,[3] and Bodil Persson[4]

Background *After the Chernobyl accident in 1986, as much as 5% of the released caesium-137 was deposited in Sweden due to a heavy rainfall 2 days after the event. A study of increased incidence of malignancies was initiated after the accident.*
Methods *The cohort included 1,137,106 inhabitants who were 0–60 years old in 1986 and lived in 8 counties of Sweden with the highest fallout of caesium-137. With the dwelling coordinate, GIS-technique and a digital map on caesium-137, each individual was matched for the exposure. Adjustments were made for several potential confounding factors. During the follow-up 33,851 malignancies was recorded 1988–1999.*
Results *Exposure categories were: 0–8 (reference), 9–23, 24–43, 44–66, 67–84, and ≥85 nGy/hr. The corresponding adjusted Mantel-Haenszel incidence rate ratios for total malignancies during follow-up amounted to 1.000, 0.997, 1.072, 1.114, 1.068, 1.125, respectively. The excess relative risk per 100 nGy/hr with the same adjustments and time period was 0.042 95% confidence limit 0.001;0.084. An excess for thyroid cancer or leukemia could not be ruled out.*
Conclusion *Increased incidence of total malignancies possibly related to the fallout from the Chernobyl accident is seen.* Am. J. Ind. Med. 49:159–168, 2006. © *2006 Wiley-Liss, Inc.*

KEY WORDS: caesium-137; ionising radiation; nuclear power; low dose; epidemiology; environment; background radiation; cancer; GIS; cohort

INTRODUCTION

The effect of low dose ionizing radiation is unclear and controversial. The highest terrestrial gamma radiation in the world has been measured in Kerala, India up to 70 mGy/y [Nair et al., 1999] and in Yangjiang, China, 6.4 mSv/y [Tao et al., 2000]. In Sweden the terrestrial gamma radiation, mainly due to granites, contributes to the population's total radiation dose.

After the Chernobyl nuclear power plant accident on April 26, 1986, additional radiation was added to the human dose, especially in the northern part of Sweden. Two days after the Chernobyl nuclear power plant accident in Ukraine airborne ionizing radiation reached Sweden. As much as 5% of the total released caesium-137 from the reactor was deposited in Sweden due to heavy rainfall on April 28–29, 1986, and unequally distributed in the eastern coastal regions from Stockholm in the south to Umeå in the north [Mattsson and Moberg, 1991]. The main contributors to the dose rate in the first weeks were short-lived nuclides replaced by the long-lived caesium-134 and caesium-137 [Edvarson, 1991]. The fallout of caesium-137 reached a maximum ground deposition of about 120 kBq/m^2 which equals about a maximum dose of 4 mSv the first year, with some 20% of the dose for a 50-year period received during the first 2 years after the accident [Edvarson, 1991].

[1]Department of Molecular and Clinical Medicine, Division of Occupational and Environmental Medicine, Linkoping University, Linkoping, Sweden
[2]Department of Health and Society, Division of Social Medicine and Public Health Science, Linkoping University, Linkoping, Sweden
[3]Department of Oncology, University Hospital, Örebro, Linkoping, Sweden
[4]Department of Occupational and Environmental Medicine, University Hospital, Linkoping, Sweden
*Correspondence to: Martin Tondel, Department of Molecular and Clinical Medicine, Division of Occupational and Environmental Medicine, Linkoping University, Linkoping SE 581-85, Sweden. E-mail: Martin.Tondel@lio.se

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In Kerala, there is still no evidence that the incidence of total malignancies is consistently higher because of the high radiation [Nair et al., 1999]. The mortality of malignancies in Yangjiang has not been increased, including leukemia mortality [Tao et al., 2000]. However, other epidemiological studies on adult populations have shown significant increase in the incidence of malignancies or mortality due to the terrestrial gamma radiation [Ujeno, 1987; Tirmarche et al., 1988], but other studies have also failed to show such relationship [Ujeno, 1978; Hickey et al., 1981; Allwright et al., 1983; Noguchi et al., 1986; Walter et al., 1986]. For leukemia the results have been similar with both positive [Edling et al., 1982; Ujeno, 1987; Tirmarche et al., 1988; Hatch and Susser, 1990] and negative studies [Court Brown et al., 1960; Segall, 1964; Jacobson et al., 1976; Hickey et al., 1981; Noguchi et al., 1986].

In Europe concerns about the consequences of the Chernobyl accident have focused on childhood malignancies, especially leukemia, as it is assumed to have a short latency period after irradiation. Several studies on childhood leukemia have been performed outside the former USSR [Auvinen et al., 1994; Hjalmars et al., 1994; Petridou et al., 1994; Parkin et al., 1996; Tondel et al., 1996], but none has shown any clear relationship to the fallout from the Chernobyl accident. However, exposure in utero has shown to increase the risk of leukemia significantly in Greece, Germany, and Ukraine [Petridou et al., 1996; Michaelis et al., 1997; Steiner et al., 1998; Noshchenko et al., 2001], but not in Belarus [Ivanov et al., 1998].

In Belarus, Ukraine and the western part of Russia there has been a dramatic increase in the thyroid cancer incidence in children, but not leukemia or other malignancies, in relation to the accident [World Health Organization, 1995; United Nations Scientific Committee on the Effects of Atomic Radiation, 2000; Gapanovich et al., 2001]. However, a recent study from the Ukraine is the first one indicating an increased risk for acute leukemia in the general population related to the Chernobyl accident [Noshchenko et al., 2002]. Recent studies outside the former USSR have also shown an increase in the thyroid cancer incidence suggested to be related to the accident [Cotterill et al., 2001; Gomez Segovia et al., 2004; Murbeth et al., 2004; Niedziela et al., 2004], but such studies have also failed to link an increase to the Chernobyl accident [Pillon et al., 1999; Szybinski et al., 2003].

The study base of our investigation includes the population of eight counties with the highest fallout after the Chernobyl accident, but also with unaffected areas in these counties serving as reference. The aim of this study was to investigate if the radioactive fallout from the Chernobyl accident has caused a detectable increase of malignancies in Northern Sweden, after adjustment by terrestrial gamma radiation exposure and other potential confounders.

MATERIALS AND METHODS

This cohort is essentially the same as in our earlier study [Tondel et al., 2004], but the population of one additional county (Södermanland) has been included due to another technique for classifying caesium-137 exposure. It was now also possible to include exposure from terrestrial gamma radiation. Finally a longer follow-up enabled us to analyze data in three time-periods. By including Södermanland county 162,757 individuals were added; a total of 1,305,939 inhabitants in the following counties: Norrbotten, Västerbotten, Västernorrland, Jämtland, Gävleborg, Uppsala, Västmanland, and Södermanland. Persons from 0–60 years of age living in the same parish December 31, 1985 and December 31, 1987 were included. To detect even a small risk in relation to the Chernobyl fallout, we included as many exposed counties as possible, used a 2-year residence inclusion criteria, classified exposure by dwelling, created maximum contrast in the radiation exposures without reducing the power, and applied an age restriction, that is, an age span with expected low incidence of malignancies in general.

The malignancies and deaths for the cohort were updated to December 31, 1999 using data from the Swedish Cancer Registry and the individual identity number for the cohort members. If an individual had more than one malignancy during the follow-up, we considered only the first one as the malignancies might not be independent events. Since the start of the Cancer Registry in 1960, all malignancies are coded according to the International Classification of Diseases (ICD) version 7 to allow for comparison over time. Thyroid cancer has the ICD-code 194 and leukemia were defined as ICD 7 204.0, 205.0, and 205.1 taken together. Because of the cohort definition a kind of short latency was allowed as the cohort start was set to January 1, 1988.

To meet the requirement of informed consent, the responsible authority, the Swedish Data Inspection Board, for practical reasons advertised the study in the two largest national newspapers. Local newspapers, radio and television covered these advertisements and there were no refusals to participate in our study, as described in detail elsewhere [Tondel and Axelson, 1999].

Each dwelling in Sweden has been appointed a coordinate by the National Land Survey of Sweden and made available from the Statistics Sweden. We got a match for 1,293,670 individuals in the cohort (99.1%). The accuracy of the coordinate has been calculated to 100 m.

The Geological Survey of Sweden performs regular aerial geophysical measurements that are collected in a database with coordinates from the Swedish national system (RT90). The measurements with gamma spectrometry cover potassium, thorium, and uranium. Potassium is given a radiation value in percent (%). For thorium and uranium the value is given in parts per million (ppm). With a conversion

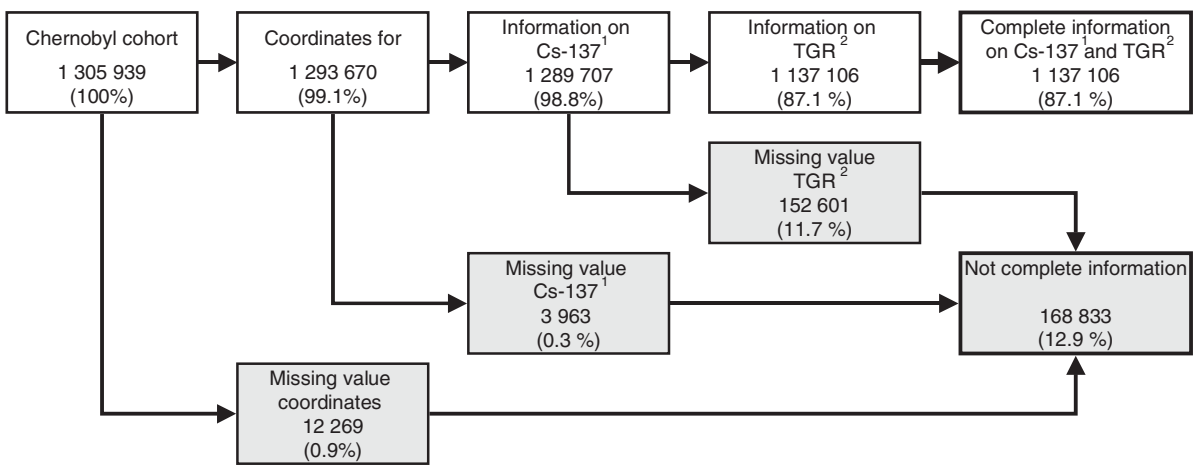
factor from the Geological Survey of Sweden, all the radiation values from the isotopes could be calculated in nGy/hr and subsequently added to a sum of terrestrial gamma radiation. The Swedish Radiation Protection Authority has, with similar technique, released a digital map on the caesium-137 fallout over Sweden after the Chernobyl accident, but given in kBq/m^2. Radiation for both maps is presented in a 200 × 200 m grid. By using a conversion factor given by Finck [1992], the radiation from the Chernobyl fallout to the individuals could be calculated in nGy/hr. Now, each individual in the cohort with a coordinate could be assigned an exposure for terrestrial gamma radiation with the help of the digital map from the Geological Survey of Sweden and for caesium radiation exposure by using the digital map from the Swedish Radiation Protection Authority. This match between coordinates and the exposures was made with GIS-technique in ArcView 3.2, including the spatial module from ESRI. The result was that 1,137,106 individuals could be appointed both a value of terrestrial gamma radiation and caesium-137 (87.1% of the original cohort of 1,305,939 individuals) (Fig. 1). The exposure categories for radiation were created before the analyses and had the same proportion of the population in each category for caesium and terrestrial radiation, respectively, (Table I).

The risk estimates in our study were, apart from age, adjusted by four potential confounding factors. The strength of these four potential confounders is shown in the Tables I and II, that is, terrestrial gamma radiation (Table I), population density, lung cancer incidence 1988–1999, and incidence of malignancies 1986–1987 (Table IIA–D).

To control for the potential confounding of population density we used two models. The first model was based on the number of individuals per square kilometer in each parish. The categories in Table II were found to have the strongest relation to the total cancer incidence and thus best serving to control confounding in this respect. The second model was the official classification of municipalities in H-regions (homogeneity), hence the municipalities of Sweden are classified into six such H-regions depending on the population density, and the number of inhabitants in the nearest vicinity of the main city in that municipality [Statistics Sweden, 1998]. The two highest density categories (H1 and H2) include only the three largest cities of Sweden, Stockholm, Göteborg, and Malmö, which all are outside our study area, leaving H3 (most dense) to H6 (less dense) for classifying the municipality in the eight studied counties.

The age-standardized lung cancer (ICD 7, 162.1) incidence by the municipality for the period 1988–1999 was taken as a proxy indicator for an aggregate of risk factors, that is, smoking habits, industrial and environmental exposures, and ill-defined other risk factors, usually subsumed as socio-economic risk factors. The incidence of malignancies 1986–1987 (i.e., before any expected effect of the fallout) was considered a proxy determinant for the incidence of malignancies in the follow-up period 1988–1999.

The categories of the potential confounding factors in Table II were kept the same as in our earlier publication to allow comparison [Tondel et al., 2004]. However, terrestrial gamma radiation has been added to these potential confounders (Table I). To decide in which order these factors should be included in the stepwise Poisson regression we used the age adjusted excess relative risk for total malignancies during follow-up 1988–1999, that is, lung



[1] Caesium exposure due to Chernobyl accident

[2] Terrestrial gamma radiation

FIGURE 1. Flow chart on the Swedish Chernobyl cohort study. Number of individuals and percent (%) of total cohort in each cell.

TABLE I. Number of Individuals and Total Malignancies by Exposure Category

	Exposure category caesium-137 (nGy/hr)						Exposure category terrestrial gamma radiation (nGy/hr)					
	0–8	**9–23**	**24–43**	**44–66**	**67–84**	**≥85**	**0–52**	**53–67**	**68–78**	**79–93**	**94–101**	**≥102**
Population (n = 1,137,106)	350,387	277,518	216,588	178,875	57,014	56,724	348,938	276,643	222,412	173,410	59,837	55,866
	(30.8%)	(24.4%)	(19.1%)	(15.7%)	(5.0%)	(5.0%)	(30.7%)	(24.3%)	(19.6%)	(15.2%)	(5.3%)	(4.9%)
Total malignancies	10,212	8,164	6,491	5,531	1,735	1,718	10,487	7,932	6,583	5,346	1,799	1,704
1988–1999 (n = 33,851)	(30.2%)	(24.1%)	(19.2%)	(16.3%)	(5.1%)	(5.1%)	(31.0%)	(23.4%)	(19.5%)	(15.8%)	(5.3%)	(5.0%)
MH-IRR 1988–1999 (95% CL)	1.000	1.015	1.060	1.047	1.015	1.034	1.000	0.983	1.007	1.027	1.024	1.028
	reference	(0.985; 1.045)	(1.028; 1.094)	(1.013; 1.082)	(0.964; 1.068)	(0.983; 1.088)	reference	(0.955; 1.012)	(0.977; 1.039)	(0.994; 1.061)	(0.974; 1.077)	(0.977; 1.082)

Mantel-Haenszel-weighted incidence rate ratios (MH-IRR) for total malignancies are adjusted by age with 95% confidence limits (95% CL) within caesium-137 exposure and terrestrial gamma radiation, respectively.

TABLE II. A–D. Strength of Four Identified Risk Factors for Total Malignancies in the Follow-Up Period 1988–1999

A: Population density			B: H-region			C: Lung cancer incidence 1988–1999			D: Incidence of total malignancies 1986–1987		
Density[a]	**No. of cases**	**MH-IRR (95% CL)**	**H-region[b]**	**No. of cases**	**MH-IRR (95% CL)**	**Lung cancer[c]**	**No. of cases**	**MH-IRR (95% CL)**	**Malignancies[d]**	**No. of cases**	**MH-IRR (95% CL)**
≤40	15,494	1.000	H6	4,812	1.000	≤7.99	3,935	1.000	≤82	2,939	1.000
41–199	7,713	1.064 (1.035; 1.093)	H5	7,002	1.010 (0.973; 1.047)	8.00–9.99	12,207	1.045 (1.008; 1.083)	83–106	9,111	1.018 (0.976; 1.061)
200–499	5,786	1.062 (1.030; 1.094)	H4	4,441	1.019 (0.979; 1.062)	10.00–11.99	9,649	1.018 (0.981; 1.057)	107–122	15,964	1.072 (1.031; 1.115)
≥500	4,858	1.083 (1.049; 1.119)	H3	17,596	1.063 (1.030; 1.098)	≥12.00	8,060	1.055 (1.016; 1.096)	≥123	5,837	1.053 (1.007; 1.101)
Total	33,851			33,851			33,851			33,851	

MH-IRR are adjusted by age and with 95% CL. Homogeneity-regions (H-region) go from sparse (H6) to dense (H3) populated regions. Lung cancer incidence 1988–1999 was taken as a proxy determinant for an aggregate of risk factors, including smoking, and similarly, the total incidence of malignancies 1986–1987 was taken as a determinant for the incidence of malignancies 1988–1996.

[a]Number of individuals per square kilometer on parish level.

[b]Population density classified by Statistics Sweden on municipality level.

[c]Lung cancer incidence per 100,000 person-years 1988–1999 on municipality level.

[d]Incidence of total malignancies for ages 5–59 years per 100,000 person-years 1986–1987 on municipality level.

TABLE III. MH-IRR for Total Malignancies Adjusted for Age, Lung Cancer Incidence 1988–1999, Incidence of Total Malignancies 1986–1987, Terrestrial Gamma Radiation, Population Density, and Homogeneity-regions According to Tables I, II

	MH-IRR (95% CL)					
	Time periods			Follow-up 1988–1999		
	Total malignancies (n = 33,851)			Total malignancies (n = 33,851)	Thyroid cancer (n = 355)	Leukaemia (n = 426)
Caesium-137 nGy/hr	1988–1991	1992–1995	1996–1999	1988–1999	1988–1999	1988–1999
0–8	1.000 (reference)	1.000 (reference)	1.000 (reference)	1.000 (reference)	1.000 (reference)	1.000 (reference)
9–23	0.944 (0.855; 1.043)	1.042 (0.956; 1.137)	0.997 (0.928; 1.072)	0.997 (0.950; 1.047)	0.986 (0.636; 1.530)	0.823 (0.525; 1.290)
24–43	0.985 (0.846; 1.146)	1.203 (1.057; 1.370)	1.037 (0.929; 1.159)	1.072 (0.996; 1.154)	1.150 (0.612; 2.159)	1.116 (0.668; 1.866)
44–66	1.263 (1.042; 1.531)	0.977 (0.816; 1.169)	1.150 (0.993; 1.331)	1.114 (1.011; 1.228)	0.545 (0.184; 1.614)	1.404 (0.705; 2.794)
67–84	1.243 (0.979; 1.578)	0.897 (0.725; 1.108)	1.110 (0.972; 1.328)	1.068 (0.949; 1.202)	1.447 (0.521; 4.021)	0.729 (0.220; 2.414)
≥85	1.302 (1.026; 1.654)	1.052 (0.856; 1.293)	1.083 (0.901; 1.301)	1.125 (0.999; 1.267)	0.536 (0.113; 2.549)	1.094 (0.448; 2.668)

cancer incidence 1988–1999 (ERR 0.045 per 100 nGy/hr), incidence of malignancies 1986–1987 (0.042), terrestrial gamma radiation (0.042), population density (0.034), and H-region (0.018), respectively.

In the first analysis we followed each individual over time and calculated the number of person-years from January 1, 1988 until December 31, 1999, or until the occurrence of the first malignancy or death, whichever came first. Throughout all the analyses, 5-year age groups were applied. Mantel-Haenszel weighted risk estimates were calculated (Tables I–III), and a log-linear Poisson regression model with maximum likelihood estimates was applied for the trend analyses, that is, excess relative risks (Table IV).

In the second analysis, calculating the excess absolute risk (EAR) in each exposure category, an age restriction was necessary to obtain the same age distribution both at starting of the cohort and at the end of follow-up in 1999, that is, comparable incidence rates over time (Fig. 2). The incidences per 100,000 person-years were directly standardized for age using weights corresponding to the European population, as defined by the World Health Organization, but restricted to ages 10–59 years [World Health Organization. International Agency for Research on Cancer, International Association of Cancer Registries, 1976]. The EAR per 10^5 person-years was based on the standardized incidence rate differences (SIRD per 10^5 person-years), using the following formulas.

$$\mathrm{SIRD}_{ij} = (\mathrm{SIR}_{ij} - \mathrm{SIR}_{jk})$$
$$\mathrm{EAR}_{ij} = (\mathrm{SIRD}_{ij} - \mathrm{SIRD}_{0i})$$

where

i = time period or follow-up,
j = exposure category,
k = 1986–1987,
0 = reference category (0–8 nGy/hr).

By definition SIRD_{0i} is not influenced by the exposure, that is, is an underlying time trend or secular trend.

Both the first and the second analyses were performed in STATA Statistical Software, release 6.0, College Station, TX: Stata Corporation.

RESULTS

During the period of follow-up, 33,851 incident total malignancies were recorded between 1988–1999. Age adjusted MH-IRR for incidence of malignancies 1988–1999 is presented for caesium-137 and terrestrial gamma radiation, respectively (Table I). Table III shows the MH-IRR adjusted for age, and the potential confounding factors shown in Table II together with terrestrial gamma radiation. The most evident dose-response is seen in the first time period 1988–1991, Figure 2, Table III and IV. When comparing ERR per 100 nGy/hr for caesium in the stepwise regression (Table IV) a significant increase is seen for the same time period followed by a decline in 1992–1995 and then a slight, but not complete return in risk 1996–1999. The significant ERR of 0.042 (95% CL 0.001;0.084) per 100 nGy/hr for caesium exposure during the follow-up 1988–1999 is therefore influenced by the first 4 years increase, and to less extent by the following 8 years. This pattern in the Poisson regression is not changed in any of the steps. An increased risk for thyroid cancer or leukemia was in statistical terms less likely in relation to the fallout, but could not be excluded taking the confidence limits into account (Tables III and IV).

DISCUSSION

The main finding is an increase in the incidence of total malignancies related to increasing caesium radiation during the follow-up period, 1988–1999. This increase is mainly

TABLE IV. ERR for Total Malignancies per 100 nanoGray per hour (nGy/hr) for Caesium-137 in Three Different Time Periods and Follow-up 1988–1999 Adjusted by Different Confounding Factors in a Stepwise Poisson Regression

	ERR per 100 nGy/hr (95% Confidence Limits)					
	Time periods			Follow-up 1988–1999		
	Total malignancies (n = 33,851)			Total malignancies (n = 33,851)	Thyroid cancer (n = 355)	Leukaemia (n = 426)
Confounding factor	1988–1991	1992–1995	1996–1999	1988–1999	1988–1999	1988–1999
Age[a]	0.114 (0.038; 0.196)	0.026 (−0.035; 0.092)	−0.001 (−0.054; 0.054)	0.036 (0.000; 0.073)	−0.214 (−0.460; 0.142)	−0.077 (−0.334; 0.277)
Age + lung cancer[b]	0.107 (0.028; 0.192)	0.027 (−0.038;0.097)	0.021 (−0.035;0.081)	0.045 (0.007; 0.084)	−0.192 (−0.448; 0.180)	−0.098 (−0.359; 0.267)
Age + lung cancer + cancer 86–87[c]	0.096 (0.015; 0.183)	0.021 (−0.046; 0.092)	0.030 (−0.028; 0.093)	0.045 (0.006; 0.086)	−0.210 (−0.468; 0.172)	−0.072 (−0.347; 0.317)
Age + lung cancer + cancer 86–87 + TGR[d]	0.093 (0.012; 0.182)	0.029 (−0.039; 0.102)	0.035 (−0.024; 0.098)	0.049 (0.009; 0.090)	−0.231 (−0.487; 0.152)	−0.081 (−0.357; 0.312)
Age + lung cancer + cancer 86–87 + TGR + density[e]	0.098 (0.016; 0.188)	0.029 (−0.039; 0.103)	0.034 (−0.026; 0.098)	0.050 (0.010; 0.092)	−0.241 (−0.495; 0.140)	−0.091 (−0.366; 0.300)
Age + lung cancer + cancer 86–87 + TGR + density + H-region[f]	0.101 (0.017; 0.193)	0.012 (−0.056;0.087)	0.028 (−0.032; 0.093)	0.042 (0.001; 0.084)	−0.224 (−0.487; 0.174)	−0.150 (−0.413; 0.229)

The order of factors has been selected in the order from the highest to the lowest in age adjusted ERR per 100 nGy/hr for the period 1988–1999, see Materials and Methods section.

[a]Five years age bands applied.

[b]Lung cancer incidence 1988–1999 in categories on municipality level according to Table II.

[c]Incidence of total malignancies for ages 5–59 years per 100,000 person-years 1986–1987 on municipality level in categories as shown in Table II.

[d]Terrestrial gamma radiation categories as in Table I.

[e]Number of individuals per square kilometer on parish level and categories as Table II.

[f]Population density classified by Statistics Sweden on municipality level, H-region categories as in Table II.

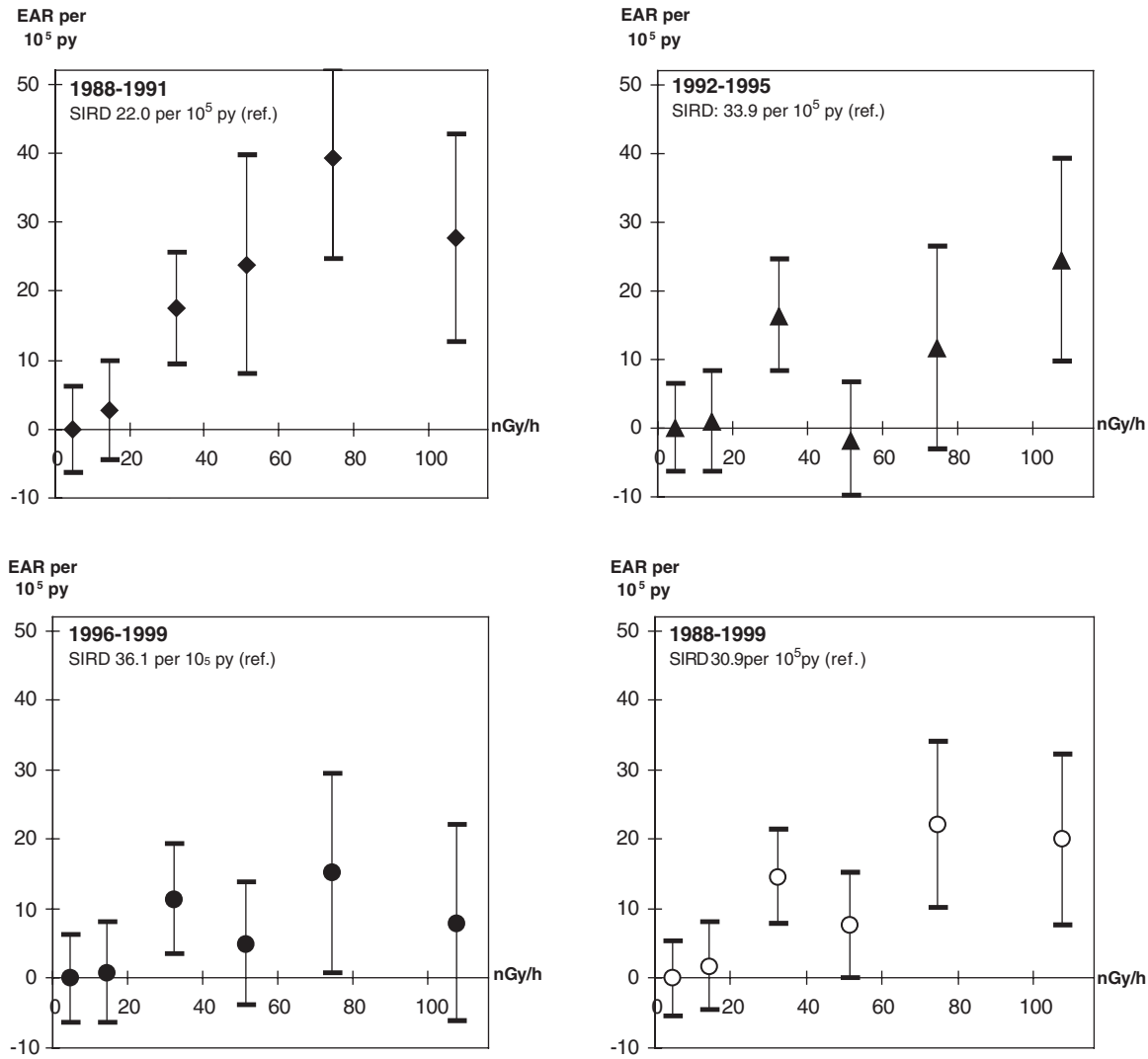


FIGURE 2. Excess absolute risk (EAR) was calculated for each exposure category of caesium-137, and in each time period and follow-up, as the age standardized incidence of total malignancies per 10^5 person years minus the incidence in 1986–1987 (SIRD), and defined as the excess above the reference category (0–8 nGy/hr). Each EAR with 95% confidence limit is placed in the median value of nanoGray per hour (nGy/hr) for that exposure category. European population restricted to ages 10–59 years is used as the standard.

explained by an increase in the first time period 1988–1991 and can be seen in MH-IRR, EAR, and ERR, respectively. The present study and a previous one [Tondel et al., 2004] both suggest a possible increase in malignancies after the Chernobyl accident, let alone marginally increased risks. Our ERR per 100 nGy/hr of 0.042 (95% CL 0.001;0.084) for 1988–1999 is also in accordance with our previous ERR per 100 kBq/m^2 of 0.11 (95% CL 0.03;0.20) in 1988–1996 because it equals to an ERR per 100 nGy/hr of 0.046 (95% CL 0.016;0.077). The slight difference in risk can possibly be explained by a larger influence of the early period in the latter estimate.

The benefit of our study compared to others is the relative accuracy in exposure assessment of ionizing radiation both from the Chernobyl fallout and from the terrestrial gamma radiation. The aim of our study was not to study the effect of low dose radiation as such, but rather to investigate the effect of the Chernobyl fallout in Sweden. The risk estimates for caesium-137 are in the same magnitude as for the terrestrial gamma radiation, however the latter was explored as a confounding factor. However, the main limitation in our estimation of the dose is the lack of individual information regarding food habits like reindeer meat, game meat, wild berries, and mushrooms, the main contributors to the internal dose. This inaccuracy in individual exposure assessment can, however, only act in the direction of lowering the overall risk.

Population density seems to be a determinant for total cancer in our study, as reported by another investigation [Schouten et al., 1996], and is probably caused by air pollution, different life styles, and occupational exposures in the cities. Smoking habits may, to some extent, be related to city life style as earlier studies have shown increasing lung cancer incidence with increasing population density [Nasca et al., 1980; Haynes, 1986; Walter et al., 1986]. Moreover, adjusting the risk estimates by using lung cancer incidence is our way of adjusting for occupational risk factors, that is, blue collar workers in Sweden have a higher smoking prevalence than white collar workers. Either smoking-related or smoking non-related malignancies could explain our findings**.** By using both our own population density classification of the parishes and the official H-region classification we control for air pollution and life style risk factors in the cities and adjust for residual confounding left out from either of them. The H-region density takes into account that people may live in a sparsely populated parish and commute to work in the nearest large city.

An early effect of the Chernobyl fallout was detected in our study, that is, the strongest ERR of 0.101 per 100 nGy/hr for the time period 1988–1991, but in contrast to the other time periods it stayed significant in all the adjustments made in the stepwise regression. We have used the strongest available confounders to anhilate this effect, but failed, and therefore we tend to believe it indicates a true dose-response from ceasium-137. However, we cannot completely exclude the possibility of remaining confounding or undetected bias to explain the risk estimates.

Based on the etiologic fractions in Table III, the number of incident malignancies related to the Chernobyl accident in the eight counties of Sweden could formally be calculated to 1,278 cases during the follow-up 1988–1999 (fourth column, Table III). The previous estimate was 849 cases during 1988–1996 [Tondel et al., 2004]. However, using the estimated collective dose in Sweden of about 6,000 manSv during 50 years [Mattsson and Moberg, 1991] and the risk estimates given by The International Commission on Radiological Protection [1990], the number of expected extra deaths in malignancies has been calculated to 300 cases [Moberg and Reizenstein, 1993].

It is not possible to detect any specific site or sites responsible for the increased risk, nor have we any good explanation of the early effect. Interpretation of our results could therefore be that the ionizing radiation might act as a late stage promotor. It is usually proposed that the latency period for solid cancers induced by ionizing radiation is rather long, even longer for low dose. Our study implies, on the contrary, that there might be an increased risk also in a few years after a sudden radiation exposure to a population. However, people in the more highly exposed regions might have been more likely to see a doctor because of a sense of vigilance. As only a small fraction of the malignancies are possible for early diagnosis, it is unlikely to have influenced the incidence of total malignancies on the population level in this study. If such effect has occurred it is impossible to differ from the suggested promotion. Such early effect can have been overlooked in previous studies. The ICRP risk estimate relies to a great extent on the follow-up of the atomic bomb survivors in Hiroshima and Nagasaki, but has been questioned as the cohort was created 5 years after the explosions and, therefore, ignored earlier cases [Stewart and Kneale, 2000].

A short latency period has also been seen in a mortality study on 14,111 patients with ankylosing spondylitis where an increase was noted 0–2 years after the X-ray treatment [Smith and Doll, 1982]. In a follow-up of the Three Mile Island accident in 1979 deaths in total malignancies were non-significantly increased for both men and women in 1979–1984 and followed by a decline. There was an increased standardized mortality ratio again for men in 1990–1998 [Talbott et al., 2003]. Recently, in a report from Belarus, the highest increase in incidence of total malignancies has already been seen in the region with the highest radioactive fallout after the Chernobyl accident [Okeanov et al., 2004]. Our findings of an increase in the incidence of malignancies soon after the Chernobyl accident is therefore not unique. In this context it is interesting that the increased number of thyroid cancers in children of Belarus in 1990, 4 years after the Chernobyl accident, now have been linked to a specific genetic rearrangements caused by radiation resulting in increased growth rate and subsequently a shorter latency period. This rapid form of thyroid cancer was followed by a slow growing form of thyroid cancer with another chromosomal re-arrangement, hence it took a longer time to diagnose [Williams, 2002; Williams et al., 2004].

Increase in thyroid cancer incidence has been seen in Belarus, Ukraine, and the western part of Russia and has been attributed to radioactive iodine through inhalation and through the food chain. This route was never prominent in Sweden and can therefore probably explain why we could not identify any increase in thyroid cancer risk. Leukemia is also a concern, as it is supposed to have a short latency, but no obvious tendency of such effect was seen in our study, nor in the earlier ones [Tondel et al., 1996, 2004]. However, our interpretation of the results regarding thyroid cancer and leukemia has to be cautious as it is based on few cases. Furthermore, taking these confidence limits into account this study can be regarded as non-positive.

The follow-up period is still rather short to evaluate an eventual long-term effect of radioactive fallout in Sweden from the Chernobyl accident. Therefore, a more definite conclusion on time trends in the incidence of malignancies, including a potential late second peak, cannot be made at this stage nor can the overall number of radiation-related malignancies be estimated.

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