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The evidence of radiation effects in embryos and fetuses exposed to Chernobyl fallout and the question of dose response

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Current legal frameworks for radiation exposure limits are based on the risk models of the International Commission on Radiological Protection (ICRP). In Publication 90 (2003), ICRP presents a safe (threshold) dose range of up to 100 mSv for radiogenic effects resulting from *in utero* exposure and bases this conclusion on the findings in Hiroshima and Nagasaki. However, a variety of observations of congenital malformations, fetal loss, stillbirths and infant deaths, as well as of Down's syndrome and other health defects in children after the Chernobyl accident exposures suggest that the A-bomb survivor data are incomplete. The Chernobyl findings are generally marginalized or even denied because of the low values of the estimated human exposures and the inconsistency of the results with the accepted risk models. One explanation for the observations is that physical dosimetric models have underestimated the effective exposure. This possibility is supported by biological dosimetry in the contaminated regions. The assumptions about effects after *in utero* exposure by incorporated radionuclides need to be revised.

Keywords: radiation-induced malformations; perinatal mortality; Down's syndrome; Chernobyl effects in children; dosimetry of incorporated radioactivity; biological dosimetry; infant leukaemia

Introduction

The evaluation of radiation risks by international radiation protection committees is largely based on findings in the Japanese A-bomb survivors, the Lifespan Study (LSS). In the LSS, the only effects observed in those exposed *in utero* were mental retardation and reduced head size; no other significant detriment was reported. Only the time between the 8th and 15th week of gestation was thought to be the period of risk for radiation exposure effects.

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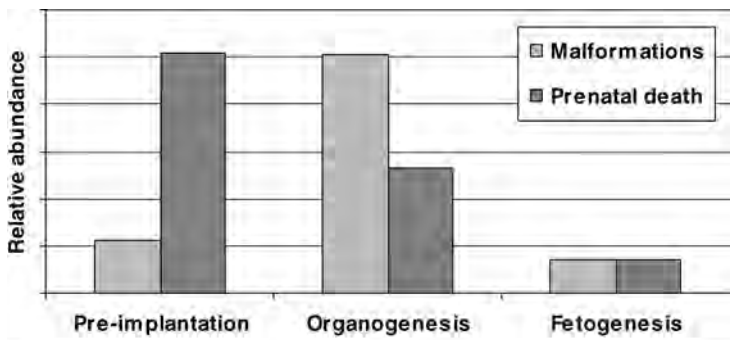
It has been remarked that the Japanese data suffer from several restrictions which limit their suitability as a general base for deriving radiation risks[1]. One point is a proven selection bias caused by the catastrophic situation after the bombing. Another objection which must be stressed especially when considering perinatal effects is the fact that the investigations of the Atomic Bomb Casualty Committee, later the Radiation Effects Research Foundation (RERF), in Hiroshima did not begin earlier than 5 years after the catastrophe when the research institute was established there. The completeness of the data must, therefore, be suspect.

It has been shown in experimental studies in rodents that low dose irradiation in all developmental stages can lead to death of the unborn as well as to morphological anomalies (Figure 1 shows the ratio of malformation to prenatal death in such studies). Further consequences are biochemical and functional distortions where the severity is also dependent on the stage of development at the time of exposure[2].

While radiation-induced cancer is regarded as a 'stochastic' effect – i.e. initiated by mutation of a single cell – developmental distortions will generally appear only after a certain level of exposure which can affect larger cell assemblies. For stochastic effects, the dose dependency after irradiation of tissues shows itself as an increase in effect from zero dose up. In contrast, developmental distortions are described as radiation effects occurring above some 'threshold' dose. An exception is assumed for the pre-implantation phase when the embryo consists of only a few cells (Figure 1). Here, the death or mutation of one cell may also lead to a development error and a dose–effect relationship without threshold[3,4].



Length of the different stages in three species (in days after conception):			
Man	0 – 9 d	10 – 60 d	61 – 270 d
Rat	0 – 7 d	8 – 15 d	16 – 21.5 d
Mouse	0 – 5 d	6 – 13 d	14 – 19.5 d

Figure 1. Developmental effects after low dose exposure *in utero* from [2].

Michel and Fritz-Niggli proved the induction of malformations in mice by only 10 mSv of X-ray exposure[5] and referred to other experimental findings in the literature below 100 mSv (Table 1)[6–10]. In contrast, the International Commission on Radiological Protection (ICRP) postulates 100 mSv as a threshold for effects after *in utero* exposure, including the induction of cancer.

Effects after in utero exposure caused by Chernobyl fallout

The Chernobyl reactor 4 exploded on April 26, 1986. This was followed by a 10-day release of radioactivity while a fire burned in the graphite moderator. The long period of atmospheric release led to an extremely inhomogeneous deposition of caesium isotopes in Europe (Figure 2). Certain areas at great distances from the source (as for example in Bavaria (Germany) and parts of Austria) showed the same surface activity as contaminated regions in Belarus[11].

International committees claim that almost no radiation effects – except thyroid cancer – were induced in the contaminated populations. Developmental effects, in particular, were not taken into consideration because the ICRP-assumed threshold dose for *in utero* effects was not reached in their estimates.

A compilation of data from the literature reporting effects following *in utero* exposure to Chernobyl fallout is given in Tables 2–5. The increase in developmental birth effects was generally studied by time series and by comparison of incidence before and after the accident.

Malformations (Table 2)

Congenital malformations were registered in a variety of European countries[12–28]. In addition, there had been early observations of anencephaly

Table 1. Minimum doses below 100 mSv showing significant effects after *in utero* x-ray exposure in experimental studies; data taken from Fritz-Niggli [2].

	Dose (mSv)	Days after conception	Effects	Source
Mice	10	8	Cumulated developmental defects	[6]
	50	0.5	Death of the embryo	[6]
	50	0.5; 1.5	Death of the embryo, polydactyly	[7]
	50	7.5	Death of the embryo, skeletal malformations	[8]
Rats	10	18	Reflex distortions	[9]
	50	0.4; 0.7	Fetal death	[10]

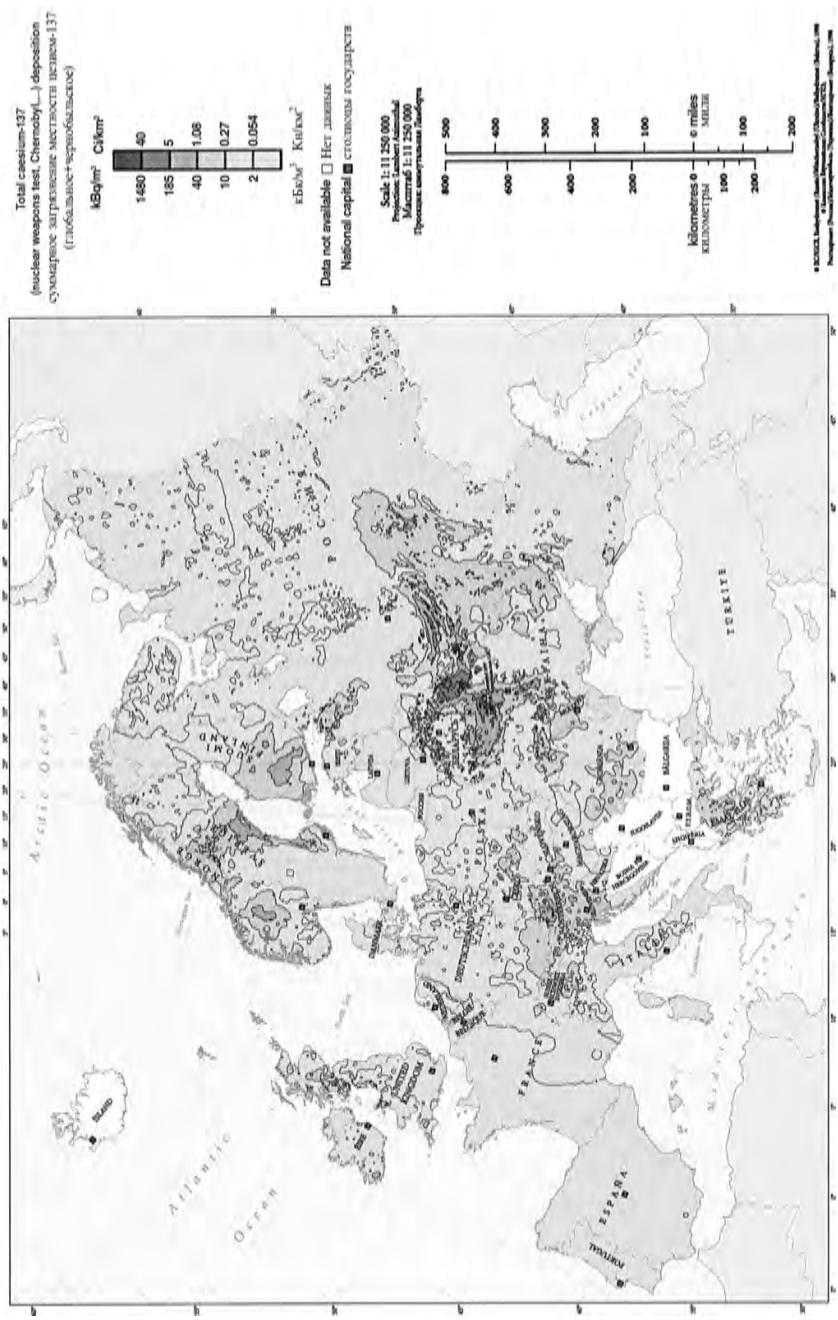


Figure 2. Caesium deposition on Europe after the Chernobyl accident from [11].

Table 2. Observed increase of congenital malformations after *in utero* exposure following the Chernobyl accident.

Country	Effects	Source
Belarus, National genetic monitoring registry	Anencephaly, spina bifida, cleft lip and/or palate, polydactyly, limb reduction defects, esophageal atresia, anorectal atresia, multiple malformations	[12,13]
Belarus		
Highly contaminated region of Gomel	Congenital malformations	[14–16]
Chechersky district (Gomel region)	Congenital malformations	[17]
Mogilev region	Congenital malformations	[16]
Brest region	Congenital malformations	[18]
Ukraine		
Polessky district (Kiev region)	Congenital malformations	[17]
Lugyny region	Congenital malformations	[19]
Evacuees from Prypiat and highly contaminated zone	Congenital malformations	[20]
Bulgaria, region of Pleven	Malformations of heart and central nervous system, multiple malformations	[21]
Croatia	Malformations by autopsy of stillborns and cases of early death	[22]
Germany		
German Democratic Republic, Central registry	Cleft lip and/or palate	[23]
Bavaria	Cleft lip and/or palate, congenital malformations	[24–26]
Annual health report of West Berlin 1987	Malformations in stillborns	[27]
City of Jena (Registry of congenital malformations)	Isolated malformations	[28]
Turkey	Anencephaly, spina bifida	[29–33]

and other neural tube defects in parts of Turkey which were highly contaminated[29–33].

The findings confirm the high radiation-sensitivity of the developing central nervous system known from the A-bomb survivors. Moreover – although the findings in Table 2 refer partly to different effects because there is no internationally agreed classification scheme used for malformations – a broad spectrum of skeletal and other morphological anomalies was found quite similar to the phenomena seen in experimental work[3,65].

In humans, the induction of malformations by low dose *in utero* exposure had been already shown for diagnostic X-rays. Besides leukaemia, prenatal and perinatal deaths, Diamond et al. registered anatomical defects in children after prenatal X-ray diagnostics[66]. German authors investigated 73 liveborn children of mothers who had been examined by X-rays or received nuclear medicine during pregnancy. They found that 7% of the children suffered from anomalies of the eyes[67].

Stillbirth, infant death, spontaneous abortion and low birth weight (Table 3)

Intra-uterine death, premature birth, low birth weight and perinatal deaths were registered in many European countries[16,17,19,26,34–46]. Such effects had been also registered in children who had been X-rayed *in utero* in the period when obstetric X-raying was common[65].

Table 3. Observed increase of stillbirths, infant deaths, spontaneous abortions and low birth weight after *in utero* exposure by the Chernobyl accident.

Country	Effects	Source
Belarus		
Selected regions	Perinatal deaths*	[16]
Chechersky district near Gomel	Perinatal deaths	[17]
Gomel region	Perinatal deaths	[34]
Ukraine		
Polessky district near Kiev	Perinatal deaths, reduced birth rate†, premature births	[17]
Lugyny region	Early neonatal deaths	[19]
Zhitomir oblast, Kiev region, Kiev city	Perinatal deaths, reduced birth rate	[34]
Kiev region	Spontaneous abortions	[35]
Europe		
Greece, Hungary, Poland, Sweden	Stillbirths	[26,36]
Poland	Infant mortality	[37]
Norway	Spontaneous abortions	[38]
Hungary	Low birth weight	[39]
Finland	Premature births among malformed children	[40]
	Reduced birth rate	[41]
	Stillbirths	[26]
Germany		
Total (FRG + GDR)	Perinatal deaths, stillbirths	[26,42,43]
Southern Germany	Infant mortality	[44]
Bavaria	Perinatal deaths, stillbirths	[26,43,45]
	Reduced birth rate	[46]

*Perinatal deaths summarize stillbirths and deaths in the first 7 days from birth.
†Reduced birth rate is considered as a measure for spontaneous abortions.

One example is a study which analysed mortality data from several European countries shown in Figure 3[26]. In this case, the elevations must be interpreted against decreasing prevalences because stillbirths and other perinatal deaths do not show constant rates over the period.

The results are supported by other low dose findings following X-ray diagnostic examination; a survey in 1980 found that 50% of U.S. women suffering from a stillbirth had been X-rayed during pregnancy[68].

Down's syndrome (Table 4)

The relationship between the incidence of Down's syndrome in several European countries and the Chernobyl accident is shown in Table 4[12,47–53]. It is well known that Down's syndrome is connected to the trisomy of chromosome 21 which results from non-dysjunction of this chromosome to the daughter cells of the egg cell. This effect can be only induced by radiation therefore *in utero* during the short period of the first division 'before' or the second division 'after' conception in the phase of pre-implantation. This effect was also observed after X-ray therapy of pregnant women[65]. An elevation of the effect was also registered in the Indian state of Kerala where the population is living on ground with high thorium concentrations[69,70] and also in regions of China with elevated background radiation[71].

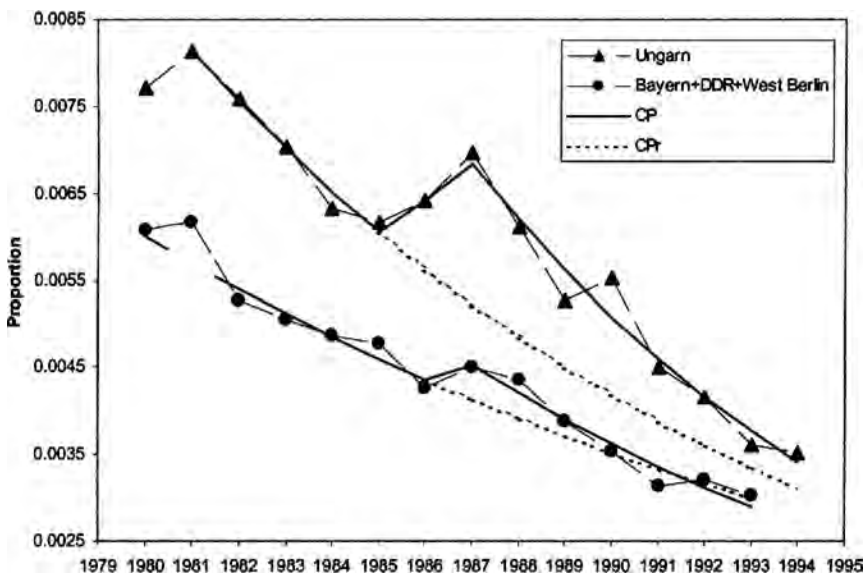


Figure 3. Stillbirth prevalence in Hungary, Bavaria + GDR + West Berlin [26].

A highly significant increase of Down's syndrome cases in West Berlin was observed exactly 9 months after the Chernobyl accident (Figure 4)[53]. At that time, this part of Berlin was a kind of closed island. The finding is confirmed by other studies after Chernobyl. A similar peak of cases after 9 months was seen in Belarus[48].

Childhood morbidity (Table 5)

Several authors reported elevated diseases other than malformations and Down's syndrome in children who were exposed *in utero* at the time of the

Table 4. Increase of Down's syndrome after *in utero* exposure by the Chernobyl accident.

Region	Results	Source
Belarus/National genetic monitoring registry	Excess 1987–1994 ca. 17% Excess peak in January 1987	[12,47] [48]
Western Europe	Beginning 1 year after the accident, reaching 22% within 3 years	[49]
Sweden	Slight excess in the most exposed areas (30%)	[50]
Scotland, Lothian region (0.74 million inhabitants)	Excess peak in January 1987 (2-fold significant)	[51]
South Germany	Elevation found by investigations of amniotic fluid	[52]
West Berlin	Excess peak in January 1987	[52,53]

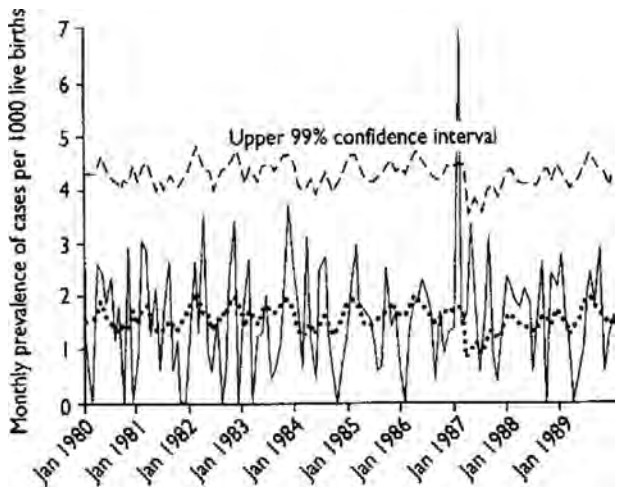


Figure 4. Increase of Down's syndrome cases in West Berlin 9 months after the Chernobyl event [53].

Table 5. Observed health defects in children after *in utero* exposure by the Chernobyl accident except malformations, Down's syndrome and cancer.

Region	Results	Source
Belarus		
Selected regions	Mental disorders	[54]
	Speech–language disorders, mental retardation	[55,56]
Chechersky district near Gomel	Diseases of respiratory organs, blood, circulation, etc.	[17]
Stolin district in Brest region	Diseases of respiratory organs, glands, blood, circulation and digestive organs	[57,58]
Belarus, Ukraine, Russia	Mental retardation and other mental disorders	[59]
Ukraine		
Selected regions	Mental retardation and other mental disorders	[60]
Evacuees from Prypiat	Mental retardation and other mental disorders	[61]
Polessky district near Kiev	Diseases of respiratory organs, blood, circulation, etc.	[17]
Evacuees from Prypiat and highly contaminated zone	Childhood morbidity	[62]
Rovno province	Childhood morbidity	[63]
Immigrants to Israel from contaminated areas	Asthma	[64]

accident[17,54–64]. Predominantly, they seem to be a consequence of the CNS distortions during development.

Elevated cancer rates in children after Chernobyl have also been reported, but are not fully reviewed in this survey. As well as inducing developmental damage, radiation exposure to the fetus increases the risk of cancer in the child. The effect was detected nearly 50 years ago by Alice Stewart through a study of diagnostic X-ray examinations in pregnancy[72]. Her conclusion – although manifold repeated and confirmed by other scientists in growing case numbers – had been denied for decades by the mainstream researchers. Such children are now generally conceded by the risk community to have suffered an excess risk of cancer in the age group 0–14 with a relative increase of 50 per Sievert[72,73].

Of interest, in this regard, is the increase in infant leukaemia after Chernobyl reported in several studies. Because the *in utero* doses were fairly well defined in the different countries, these observations also enable some

idea of the differences between predictions of current risk models and the observations. The matter was discussed in the UK Committee Examining Radiation Risk from Internal Emitters (CERRIE) but the issue was not fully resolved[74,75]. Following Chernobyl, there were statistically significant increases in infant leukaemia reported from Greece[76], Germany[77], Scotland[78], Wales and Scotland[79] and Belarus[80] for those children who were *in utero* over the peak period of fallout in the respective countries. Researchers of the International Agency for Research on Cancer in Lyon wrote to the CERRIE committee in 2004 stating that the 'European Childhood Leukaemia and Lymphoma Incidence Study' was analysing the infant leukaemias in Europe after Chernobyl but so far there has been no published report of any conclusions.

Leukaemia must be considered as a stochastic effect, the less one can follow the general threshold dose concept of the ICRP for exposure *in utero*.

The dose argument

International radiation protection committees assume that the exposures of the population after Chernobyl are much too low to generate teratogenic and genetic effects. Indeed, their physical dose estimates resulted in mean effective life-time exposures in large regions of Europe and in Turkey below 1.2 mSv[81]. The highest average dose for a subregion in the first year after the accident is derived to 2 mSv in Belarus. Therefore, even assuming an extremely high radiosensitivity of the embryo and fetus, the observed effects are not explainable by such low exposures near or below background estimates.

The derived dose values may, however, underestimate the real situation for several reasons:

(1) The assumptions about the transport and distribution of radionuclides after the accident are erroneous. Although fuel hot particles were found thousands of kilometres from the source[82] the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) researchers supposed that relevant radionuclides as Sr-90 and Pu-239 were deposited within 100 kilometres of distance to the Chernobyl plant and the exposure outside – except by iodine in the thyroid – was only generated by the Caesium isotopes 134 and 137.
(2) The dose factors of the ICRP to calculate the dose of an individual after inhalation and ingestion of radionuclides are inadequate, especially in embryos and fetuses[75,83] because they depend on average values of energy per unit mass. Where the mass is small, as with the early fetus or implant, inhomogeneities of energy deposition, so-called anisotropy, can result in very high local doses to

developing tissue from internal radionuclides or particles. There is also a concern about effects of radionuclides which because of their chemical nature (Sr-90, Ba-140 and Uranyl ion) bind to DNA and may, therefore, deposit more energy into the critical target than would be calculated on the basis of the tissue organ approach employed by ICRP. As long ago as 1963, Luning et al. showed significant fetal death in mice whose fathers were exposed to Sr-90 and Cs-137. The authors argued that the clear differential effects of the Strontium isotope compared with Caesium was due to its binding to the DNA[84].

(3) The dose–effect relationships for the developing stages are unknown in the case of incorporated radioactivity. They cannot be derived from study groups who had been exposed to external X-rays or gamma irradiation[75,85]. The ICRP concept to make the effects of different radiation qualities compatible by introducing 'radiation weighting factors' does not effectively deal with this problem. Moreover, it is clear that the dose–effect relationship for the early fetus is unlikely to be linear, because beyond a certain level of radiation injury to any tissue which is critical to the survival of the fetus, there will be a reduction in the end point being considered even though the exposure is increasing, due to death of the fetus and loss as miscarriage. This is the biphasic dose response. Therefore, to argue that effects seen in countries where the doses are low (e.g. Germany, Greece) cannot be caused by radiation because such effects are not seen in countries or areas where the doses are high (e.g. Belarus) is an invalid argument because in the high dose regions, early fetal death may have removed potential cases.

The discrepancy between observed effects and UNSCEAR dose estimates, adopted by the WHO, is confirmed by 'biological' dosimetry. Investigations of unstable and stable chromosome aberrations in the lymphocytes of persons in the contaminated regions have been done by a variety of research groups in rather large collectives directly after the accident or some years later. Dicentric chromosomes and centric rings can be considered as radiation specific. They are a very sensitive indicator for radiation because of their very low and nearly constant rate in unexposed persons which is a consequence of the instability of the dicentrics[86]. The half-life is about 1.5 years in adults. Accumulation of background exposure will, therefore, not lead to continuous elevation of the rate of dicentrics (dic) in an individual. Centric rings (cr) are stable but much less frequent than dicentrics. In case of an acute and homogeneous whole body exposure by gamma or X-rays, the doubling dose for the effect (dic + cr) is only about 10 mSv (2-fold relative elevation after 10 mSv).

It is a general experience that the observed rates of dicentric chromosomes and centric rings after Chernobyl are considerably higher – by 1 or 2 orders of magnitude – than would be expected from physically derived dose estimates[87].

A remarkable finding in many of the chromosome studies is that they report an overdispersion of the dicentrics and the occurrence of multi-aberrant cells[88–94]. This is a reliable indication for a relevant contribution of incorporated α-activity or of hot particles.

Examples of the exposure of populations by Chernobyl fallout are given in Table 6[95–97]. In Austria and Germany, the Alps regions were predominantly affected by Chernobyl fallout which was washed out there by rainfall. Some chromosome studies were, therefore, also carried out in these regions. In a study of 16 adults in Salzburg city, Austria, in 1987 (June–August), the physical dose estimate was derived by the authors using UNSCEAR modelling[95]. Two of the citizens had been studied already in 1984/1985, before the accident. They were also followed up in 1988 and 1990 (Figure 5).

In a study of 29 persons in Berchtesgaden, Germany, only 20 kilometres away from Salzburg, two areas with low contamination in southern Germany, Baden-Baden and Tirschenreuth (near to the Czech frontier), were selected for controls (Table 6)[96]. The physical dose estimates were taken by the authors from German authorities. The elevation factors given for the dic + cr rate in Table 6 were derived by using the former published control value 0.9×10^{-3} of the authors gained from 26 unexposed adults[93].

Both studies in the Alps region lead to elevations of dic + cr which are far above the equivalent calculated excess exposures. Although the Salzburg investigators found a correlation between aberration rate and measured Chernobyl deposition, the German investigators doubted the causation by radiation because of the high aberration rates in their controls. In contrast to this, they found a significant decrease with time in a subgroup of the Berchtesgaden sample (Table 6) as would be expected after a Chernobyl contamination. Further, there were several cells showing an overdispersion of aberrations and therefore an incorporation of alpha radioactivity.

Norway was contaminated in spots up to 600 kBq/m^2 of Cs-137; chromosome studies were done in three such regions and a 10-fold elevation of dic + cr still 5 years after the accident was found[97]. The doses were calculated based on whole body counter measurement of Cs-134 and Cs-137 using dose conversion factors of the ICRP. The authors interpreted the enormous discrepancy in the aberration findings as due to a biphasic dose response. It is reported that radioactive particles from Chernobyl were released predominantly by the fire after the explosion and this contributed significantly to the population exposure even in Norway[82]. The contamination contained fission products but also heavy fuel and breeding products such as U and Pu.

Table 6. Biological dosimetry in persons living in West European regions contaminated by Chernobyl releases.

Region	Sample	Date of study	Result of chromosome study			Physical excess dose estimate	Source
			dic + cr	Derived dose*	Overdispersion		
Austria							
Salzburg	16 adults	1987	6-fold	30 mSv		0.1–0.5 mSv	[95]
Germany							
Berchtesgaden	27 adults and two children	1987–1991	3–2 fold	15–10 mSv	Six cells with two dic	≤1.6 mSv	[96]
Baden–Baden	20 adults	1987–1991	3-fold	15 mSv	In one person three cells with two dic	<0.14 mSv	
Tirschenreuth	11 adults	1987–1991	2-fold	10 mSv	one multiaberrant cell	< 0.14 mSv	
Berchtesgaden							
Subgroup	5	1987/1988	3-fold	15 mSv			
Subgroup	5	1990/1991	1.6-fold				
Norway							
Selected regions	44 reindeer samples and 12 sheep farmers	1991	10-fold	50 mSv		5.5 mSv	[97]

*Minimum dose (decline of dicentrics not considered) if homogeneous whole body dose is assumed.

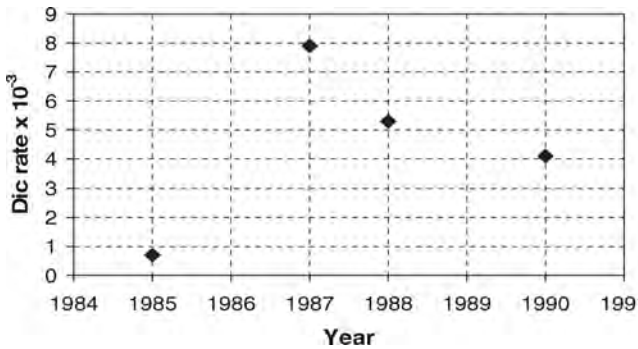


Figure 5. Mean rate of dic + cr in two citizens of Salzburg [95].

If the UNSCEAR assumptions were accurate – i.e. the exposure except iodine outside the 100 kilometre-range is only generated by incorporated and deposited Cs-134 and Cs-137 – then the chromosome information could be used for dose estimation because Caesium distributes uniformly in the body and the external gamma exposure is approximately uniform. The elevated rates of (dic + cr) in Table 6 would then represent between 10 and 50 mSv whole body dose in the sample. These examples show the high grade of error for the physically derived values.

Because there is evidence, however, that the contamination includes other nuclides which distribute selectively to certain tissues as bone and bone marrow, quantitative dose values can not be derived by the cytogenetic parameters.

Discussion

The effects seen in the cytogenetic studies after Chernobyl – significant elevation of the chromosome aberrations in the selected population sample and also in single persons within these samples – are clearly caused by radiation. These changes are objective and physical: they cannot be interpreted by psychological or socioeconomic factors after the catastrophe.

The observations about birth defects after Chernobyl were done in epidemiological studies of an ecological nature. A causal relationship would not be derivable in a single study even if the official dose estimate could be disproved. Because the findings are, however, repeated in numerous different investigations, not only can the kind of detriment be described precisely, but it is also possible to exclude other causes than radiation which could be responsible for regional or genetic reasons. Further, the observed malformations correspond to those which would be expected on the basis of the results of the experimental research after median and low doses. This is also true for intra- and extra-uterine deaths.

The cited studies are, of course, methodically of different quality. The often raised objection that there is only a feigned elevation of the effect because the investigators paid special or higher attention after the event is not valid. Stillbirths and infant deaths are precisely registered. The listed malformations in Table 2 are taken mainly from routine registries. In cases of gross anomalies such as anencephaly and *spina bifida aperta*, misdiagnosis can be excluded. The former effect was so high in Turkey that a real increase after Chernobyl cannot be doubted[98].

In Croatia and the former German Democratic Republic (GDR) (Table 2), the autopsy of all abortions, stillbirths and early infant deaths was laid down by law. Moreover, the GDR had a national registry for congenital malformations, the authors pointed out that the elevated rates occurred in the regions of the highest contaminations in the GDR[23].

It is not possible from the data to derive dose–effect relationships for incorporated radioactivity. Further research efforts are necessary to gain more information about the dose in the contaminated regions.

A further problem for the estimation of dose–effect relationships for chronic exposure of a population must be seen in the fact that it is not generally easy to decide if the observed effects are teratogenic or genetic because malformations are also inducible by exposure of parental germ cells. It was pointed out by Rugh in 1962 that the appearance of certain radiation-induced malformations of the central nervous system is completely similar whether generated *in utero* or preconceptionally[3,65]. Fetal death, premature births and neonatal deaths are also genetically inducible. As reported by the RERF, significant elevations of genetic effects were not observed in the children of the A-bomb survivors. On the other hand, malformations have been reported after preconceptional exposure by diagnostic X-rays[99] and following occupational exposure[100].

What is shown at present by the experience after Chernobyl, is that the early stages of development in human life are highly vulnerable to ionising radiation as was assumed in former times of radiation research. The current concept of dose thresholds – as high as 100 mSv stated in ICRP 90 of 2003 – does not appear to conform to the observational evidence in cases of chronic low-dose exposure.

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