

**Expert Witness Statement for Case
Don Battersby and Anna Smith
vs.
Secretary of State for Defence**

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1. I am a physicist and am now retired. One of my research interests is the health effects of ionizing radiation. I have done research on this issue and have carried out investigations into the way in which radiation is quantified and related to biological effects like chromosome aberrations. I am the Chair of the independent European Committee on Radiation Risk (ECRR).

My CV and publications I add at the end.

Dr Busby has asked me to act as an expert witness in a case where he is representing some Atomic Test Veterans who are claiming pensions for effects of radiation exposures at the test sites. I am told that these pensions were refused because the radiation risk model of the International Commission on Radiological Protection does not predict cancer or other measurable harmful effects at the low doses that these test veterans apparently received, according to film badges, and in the case of one of the veterans in this case, Barry Smith, he was not at the test sites when there was a detonation.

I have been asked to briefly explain what is a risk model for radiation, how it has been developed and how in the cases of the kind of internal exposures received by the test veterans, the present ICRP model is wrong.

2. A radiation risk model is a way of telling how much cancer, genetic disease or other illnesses are created by exposure to radiation. To do this you need to have studies of people who have received a radiation exposure and to then know how much cancer or other ill health, or genetic effects in their offspring occur. The ICRP method that has been developed uses a measure of radiation called “absorbed dose” or “dose”. The units are Grays (Gy) or for very strong ionizing types of radiation (alpha particles, neutrons) the modified version of dose is called “equivalent dose” and is calculated in Sieverts (Sv). “Dose” is a simple physics based idea. It is energy per unit mass. So an energy of 1 Joule absorbed by 1 kilogram of tissue (assumed to be water) is a dose of 1 Gray. For alpha particle radiation, such as that delivered by Uranium or Plutonium, the model defines a new “equivalent dose” in Sieverts by multiplying the “dose” in Grays by a factor considering the relative biological effectiveness $w_R = 20$ (ICRP 2007). This use of a “radiation weighting factor” was a development to allow for the high degree of ionization density (also called Linear Energy Transfer or LET) caused by alpha particles.

The European Committee on Radiation Risk (ECRR 2010) has extended this idea of using a weighting factor to other situations where the ionisation density is high. By this approach certain internal exposures would carry a higher weighting and so therefore would deliver a

higher equivalent dose. The main target for radiation induced effects is the DNA. The proportion by mass of DNA in a cell is about 1/100th. So for radioactive elements bound by chemical affinity to DNA (for example Strontium-90, Uranium) the “dose” at the DNA could be about 100 times the average dose used by the ICRP approach and this would be one reason for applying a weighting factor of 100 to ICRP calculated doses from Uranium.

3. The ICRP model predictions for cancer are based on external radiation exposures. The cancer and exposure data that are used come from the Life Span study of the survivors of the Hiroshima and Nagasaki bombing in 1945. This study is on-going. The basic method of the study is to group individuals by categories of external dose according to their distance from the location of the detonation. These distances (between one and several kilometres) were converted into external acute doses following calculations and experiments in the USA with similar bombs and structures meant to represent Japanese houses. Then the doses were correlated with the cancer yield.

The radiation risk is expressed in the literature in terms of the Relative Risk per Sv or as the Absolute Risk per Sv. The relative risk refers to the spontaneous or “background” risk and is given by the ratio of the radiation-induced rate of diseases in a population compared to the rate of diseases which spontaneously will occur. The absolute risk will name the rate of diseases which are to be expected by radiation.

The current estimate of the ICRP for the absolute risk per Sv after exposure of a population is presented in the upper lines of Table 1.

The lower lines give a survey about the state of knowledge how it is seen by many scientific critics of the ICRP, as e.g. the members of the ECRR.

Table 1 Health effects by low-dose irradiation of a population

	Cancer mortality	Hereditary diseases	In utero exposure	Morbidity except from cancer
ICRP Risk estimate (2007; 2012)	Exposed as a whole adults 5.5 % per Sv 4.1 % per Sv	0.2 % per Sv 0.1 % per Sv	No effect below 0.1 Sv	No effect below 0.5 Sv
Evaluation by critical analysis	Underestimated by at least about 10-fold	Underestimated severely: Cancer Malformations Mental disorders Down’s syndrome Childhood morbidity Stillbirths etc.	Congenital anomalies	Circulatory and organ diseases; mental distortions etc.

The risk estimate by the ICRP for cancer mortality in column 2 means, that 5.5 % = 550 additional cases of cancer deaths are expected if a population of 10.000 persons is exposed by 1 Sv , or a population of 10 million people by 1 mSv (1 Sv= 1000 mSv). Such relation can also

be considered as an individual risk. It corresponds to a probability of 5 % for suffering from cancer death after exposure by 1 Sv.

Children are assumed to be of higher sensitivity than adults at exposure by factor 3, the risk estimate for adults in Table 1 is therefore only 4.1 % per Sv. The ICRP has derived these figures from the Japanese data of the 1990-ties, but then divided to the half, because they apply the so-called DDREF (Dose and Dose-Rate Effectiveness Factor) of 2. In former times of radiation research scientists believed, that a radiation exposure of high dose-rate (dose per time) as valid in the case of the A-bomb explosion would be much more effective than a low dose-rate exposure of the same accumulated dose.

It was shown in the meantime by many studies in occupationally exposed persons that the opposite may be true – irradiation of low dose-rate is of higher effectiveness than high dose-rate also in case of pure external exposure (Jacob et al. 2009). Many scientists and also German radiation protection boards have therefore abandoned the use of a DDREF (SSK 2007; BfS 2005), while the ICRP is insisting up to now.

Another item is the lower biological effectiveness of high energetic gamma-rays in comparison to that of lower energies. Straume (1995) has drawn attention to the fact that the gamma-rays following the atomic explosion are extremely high-energetic, up to 20 MeV= 20.000 keV. X-rays of 250-kVp (corresponding to an energy range of 15 to 250 keV) are normally taken as a reference source for the equivalent dose in Sv of low LET radiation (X-rays, gammas, betas). It is basic knowledge in radiation biology that the Relative Biological Effectiveness of these radiations also depends on the energy. The ICRP, however, uses a Radiation weighting factor $w_R=1$ for all photons and electrons to derive the dose in Sv from the energy dose in Gy.

Occupational or environmental exposures by natural radioactivity or fission nuclides are normally of substantially lower energy than the Hiroshima radiations which lay predominantly between 2 and 5 MeV, with a mean of about 3 MeV. If the latter is used as a standard as done by the ICRP, the risk factors in other situations should be **multiplied by 2-4**, as is shown in the data compilation of Straume.

The ICRP assume that there is a linear relation between dose and cancer probability right down to zero dose, with no threshold. This is termed the Linear No Threshold assumption. There are various arguments in the literature about the true form of the dose response but I will not discuss them here.

4. There are some other major problems with the ICRP model and its use of the Japanese data. The first one was pointed out by the eminent epidemiologist Alice Stewart (who was the first Chair of the ECRR). It is that the population from which the groups were chosen in 1950, five years after the bombing, excluded all those who had died in the interim as a result of their radiation injuries. Therefore the study groups were healthy survivors and not representative of a normal population exposed to radiation. The second one was that there was significant rainout of radioactivity in the form of nanoparticles of contaminated Uranium (and in Nagasaki, Plutonium). These contaminated parts of the city quite far from the epicenter, and this contamination remained for a long time. Uranium from this contamina-

tion was measured in 1983 (Takada et al 1983). Thus all the groups that were compared in order to establish a risk factor for external dose, were exposed additionally to the residual radioactivity, but at different levels of dose. This leads to an erroneous result in deriving a dose-effect relationship in that direction as if one would use an exposed control group, which therefore corresponds to an relevant underestimation of the effect (Schmitz-Feuerhake 1983).

5. Taken together, these criticisms seriously question the credibility of the Japanese LSS studies even for external acute exposures. Regarding a factor 2 because of cancelling the DDERF, a further factor 3 for the less effect of high-energetic gamma-rays there appears already an underestimation by 6. Moreover, recent analysis of the Japanese data after a longer period of observation lead to higher risks than adopted by the ICRP. Ozasa et al. (2012) investigated the mortality by solid cancer in the A-bomb survivors considering the period 1950-2003 and derived an absolute risk of 26.4 % per Sv. If one adds the risk of 0.7 % per Sv for leukaemia and lymphomas (Preston et al. 2004), a value of 27.1 % per Sv is received for all cancer mortality. The half of this is 13.5 % after dividing by DDREF after ICRP, and this is higher by factor 2.4 compared to the figure given in Table 1 for cancer mortality. Therefore we have – cautiously estimated – at least a 10-fold underestimation by ICRP for external exposure by occupational and environmental radiations.

Since the Japanese studies are unable to inform us about effects from internal radiation, we should move to examine situations where there are data from internal exposures. Such an analysis has been begun by the ECRR. There are a large number of published studies now, of childhood leukemia and adult cancer near nuclear sites, and effects especially after Chernobyl, where it is clear that the errors in the ICRP model for internal exposures are extremely high, of the order of between 100 and 10,000-times. A review of the issue of internal radiation and health, explaining the problems and listing some of the evidence for the errors in the ICRP approach has been given by Busby 2013. I will copy these below since it saves me having to repeat what has already been done.

6. The issue of internal exposures was the subject of the 2009 International conference of the ECRR held in Lesbos, Greece, where the effects of the Chernobyl accident exposures on the populations of Belarus, Ukraine and the Russian Republic were discussed. At the end of the conference a document: the Lesvos Declaration was prepared by the delegates and signed by all of them. It was agreed that the situation with radiation protection was serious. The Declaration (which I attach) called for governments to set aside the ICRP model and develop a proper and accurate understanding of the effects of internal radiation exposures.

7. I list now evidence for the failure of the ICRP model.

7.1. Childhood cancer near nuclear installations

There have been reports in peer reviewed journals of increased risk of childhood leukemia and non-Hodgkin lymphoma near many nuclear sites in Europe. A list and discussion may be found in ECRR 2010. Child leukemia excesses are found near nearly all the sites that have been examined (ECRR 2010) e.g the reprocessing sites at Sellafield (Beral et al 1993) Dounreay UK (Urquhart et al 1991) and La Hague (France) (Viel et al 1995)] near the Atomic Weapons Establishment Aldermaston (UK) (Roman et al 1987) , the Atomic Energy Research

Establishment Harwell (UK) (Busby and Scott Cato 1997), near Hinkley Point nuclear power station (UK) (Bowie and Ewings 1988) and recently near all the combined nuclear sites in Germany (KiKK study) (Kaatsch et al 2008) and near all the combined nuclear sites in France GB, and Switzerland (Sermage-Faure et al 2012). I myself have made a study of the leukemia cluster near Elbmarsch in Germany which is close to a nuclear plant (Schmitz-Feuerhake et al 2005).

The radiation risk community (Comare 1986, NRPB 1995) basing calculations on the ICRP risk model have worked out the dose ranges and say they cannot be more than a few microSieverts, well below Natural Background.

The ICRP does not give a risk factor for childhood leukaemia but to define a difference between external and internal exposure we can employ the Excess Relative Risk based on the obstetric X-ray studies analysed by Wakeford and Little (2003). This gives an Excess Relative Risk of 50/Sv and based on the Obstetric X-rays results of Alice Stewart.

That would suggest a 4% increase after 1 mSv, 0.2% after 50 μ Sv which we can assume is the highest mean doses received by those in the 5 km distance region. But we are seeing a 100% increase at this level. The error even in using the Wakeford and Little Excess Relative Risk is thus at least 500-fold. If we use the ICRP ERR of 0.45/Sv for all cancers the error is greater than 1000-fold, and this number has been conceded by the authors of the KiKK study and others who argue that because of the enormous disparity between the observers and the predicted on the basis of the ICRP model, the leukemias cannot have been caused by the radiation. There is, however, no other plausible explanation.

7.2 Infant leukemia after Chernobyl

Five different groups (Michaelis et al 1997, Petridou et al 1996, Gibson et al 1988, Busby and Scott Cato 2000, 2001, Busby 2009) reported a statistically significant increase in infant leukemia in 5 different countries of Europe in those children who were in the womb at the time of the Chernobyl Caesium-137 fallout as measured by whole body monitoring. The effect was also reported from the USA (Mangano 1997). Thus the Chernobyl exposure is the only explanation for the increase. This occurred and was reported from Greece, Germany, Scotland, Wales, Belarus, USA and the error this shows in the ICRP model was the subject of two peer reviewed papers (Busby and Scott Cato 2001, Busby 2009). Using the Alice Stewart relation between dose and leukemia above, the error is about 400-fold (depending on the country) (Busby 2009). Using the ICRP model it is upwards of 1000-fold. This analysis is most relevant since it unequivocally supports the causal relation revealed by the nuclear site child leukemias yet in this case fission product internal radiation can be the only cause.

7.3 Cancer following Chernobyl in Northern Sweden

The study by Martin Tondel found an 11% increase in cancer for every 100 kBq/sq metre of Cs-137 from Chernobyl (Tondel et al 2004). It is possible to calculate that 100 kBq/m² Cs-137 including a further 100kBq/m² of Cs-134 if reduced exponentially due to rain washout to rivers and lakes with half life of 6 months would give a committed effective dose of about

1 mSv. The ICRP model (ICRP 2007) predicts an Excess Relative Risk¹ of 0.45 per Sv, so the ICRP expected excess relative risk, including a DDREF of 2 is 0.0225%. The error in ICRP model defined by Tondel's result is thus 490-fold.

7.4 Cancer in persons exposed in adulthood

Since the 1970-ies, a great variety of studies on nuclear workers have been done. Many of them showed a significant increase of cancer with dose even within the legal limits. This was confirmed in 2007 by the IARC (International Agency for Research on Cancer), a foundation of the WHO. IARC organized the 15-Country Collaborative Study of Cancer Risk among Radiation Workers in the Nuclear Industry (Cardis et al. 2007). The measured doses in the study groups were low, mean values lay below accumulated 50 mSv. The results in the studies – expressed in values of the excess relative risk per Sv – were very different, this may be caused predominantly by the problem of choosing the suitable control group. It is proven that the normal population can not serve for comparison because of the so-called “healthy worker effect”. Persons engaged for such professions are selected to be in good health and show less cancer incidence near zero dose burden. The effect can reach 50 % or more.

Researchers therefore avoided to use an external control group in the meantime. They divide the study group into dose cohorts and construct a dose-effect relationship. From this the risk per Sv is derived, as it is done in the recent work on the A-bomb survivors. Studies using such an intrinsic control and having observed the development of diseases over a long period, show much higher effects as would be predicted by ICRP. Examples are given by the investigations using the Canadian National Dose Registry for occupational workers (Zielinski et al. 2008). The registry “includes information on about 600,000 nuclear, industrial, medical and dental workers exposed to the average cumulative dose of several mSv”. The result for cancer mortality was an excess relative risk ERR of 3.0 per Sv which is 6.7 times higher than the risk estimate of ICRP (2007) with an ERR per Sv of 0.45.

Similar differences are also shown in a study of Richardson et al. (2009) about Non-Hodgkin lymphoma in nuclear weapons workers at the Savannah River Site in South Carolina between 1950 and 1986. They compared their results to those of the ICRP reference collective, the Japanese survivors. The workers showed an ERR = 6.99 per Sv, which is about 9 times the value of ERR = 0.79 per Sv derived from the Japanese cohort.

A further confirmation of the high discrepancy between ICRP estimates and real risks of workplaces with low dose irradiation must be seen in the fact that pilots and flight attendants show increased cancer rates (Table 2). They are exposed by external cosmic radiation which includes a high LET component of neutrons and protons (charged particles). They are treated as radiation workers in most countries. The studies regarded in Table 2 could not reflect to measured individual exposures. The dose-rate for those persons is estimated to be 2-5 mSv per annum above background and to reach about 80 mSv in a lifelong career (Waters et al. 2000). Taking this figure and the relative risk of 1.67 for all cancers of male flight attendants in Table 2 – which means an ERR=0.67 per 0.080 Sv – this

¹ The Excess Relative Risk ERR is defined as Relative Risk minus 1 (ERR = RR-1)

would result an an ERR per Sv of 8.4 compared to 0.45 by ICRP (factor of 17). This raw estimate is suitable to prove again the severe underestimation of risks by ICRP.

Table 2 Meta-Analysis of studies about cancer in flight personnel

Extract of results (Tokumar et al. 2006; Buja et al. 2005)

Kind of disease	Number of regarded studies	Relative	
		Risk	Confidence interval
Female flight attendants			
Breast cancer		1.41	1.22-1.62
Melanoma		2.13	1.58-2.88
Male flight attendants			
All cancers	3	1.67	1.14-2.45
Melanoma	3	3.42	1.94-6.05
Further skin cancer	2	7.47	3.52-15.87
Non-Hodgkin-lymphoma	2	2.49	1.03-6.03
Leukaemia	2	1.67	0.35-7.94

7.4 Human sex ratio at birth perturbed by low doses of internal fission-product ionizing radiation

Studies by Hagen Scherb and Kristina Voigt (2010) show clear and highly statistically significant alterations in the human sex ratio at birth (the number of boys born to girls) after (a) atmospheric bomb testing, (b) Chernobyl and (c) near nuclear facilities. Effects are shown to be local, European (several countries were studied) and global, supporting earlier evidence of increases in infant mortality during the period of atmospheric weapons testing. Sex ratio has been accepted as a measure of genetic damage with the preferential killing of one or other sex depending on the type of exposure (mothers or fathers). According to Scherb and Voigt, millions of babies were killed *in utero* by these effects . A recent re-analysis of the sex ratio effect in Hiroshima reveals the effect in those populations also (Padmanabhan 2011), evidence which was overlooked by the USA researchers through poor epidemiology and questionable decisions. This evidence objectively confirms the serious genotoxic effect of internal ionising radiation on germ cells and the exquisite sensitivity of humans and other living creatures to releases from Uranium fission. The ICRP does not consider such effects nor are they included in any assessment of harm.

7.5. Cancer and genotoxic effects in Iraq following DU exposure

A series of studies of the population of Fallujah Iraq (Busby et al 2010, Alaani et al 2011, 2012) shown to have been exposed to Uranium following the 2003-2004 battles have revealed extremely high rates of congenital malformations at birth and cancer and leukemia/lymphoma in adults. The studies also draw attention to significant sex ratio effects at birth beginning after 2004. These results, and the increases in genotoxic effects in the offspring of Gulf veterans support and are supported by the other sets of observations reviewed above which show that inhaled Uranium nanoparticles represent a very serious hazard which was not incorporated into the Life Span study and is entirely overlooked by ICRP.

7.6. Chernobyl effects as reported in the Russian peer-reviewed literature

The effects of the Chernobyl accident exposures have been reported in the Russian language peer review literature since 1996. These results have been reviewed by Busby and Yablokov 2006, 2009, Yablokov et al 2009 and Busby et al 2011 but have been largely ignored by ICRP. They constitute a very large body of peer reviewed work which show that the effects of the Chernobyl accident exposures are massive and extremely serious. They range from cancer and leukemia to heart disease especially in children together with a range of illnesses which can be best described by the term premature ageing (Busby et al 2011). They include congenital transgenerational diseases and are reported in animals and plants which cannot be affected by the kind of psychological processes (radiophobia) which have been employed by the radiation risk establishment to account for the early reports coming out of the affected territories. In addition, there are objective measurements of serious biological harm to humans and other living creatures affected by the exposures. The germline mutations found by minisatellite tests (Dubrova et al 1997) in humans were also associated with real morphological effects and fitness loss in birds (Ellegren et al 1997) and were shown to have caused significant sex ratio changes in the birds and also population loss (Moller et al 2012) which is in agreement with the findings of Scherb and Voigt and the infant mortality findings. The implications for the understanding of the historic effects of the nuclear project on human health are alarming.

7.7 Uranium

The anomalous health effects of exposure to Uranium, especially in the form of particulates, have been increasingly clear in the last 10 years. The radiobiological evidence is reviewed in ECRR2009 and in a recent review (Busby 2015). The issue is relevant to the Test Veterans since the bombs were constructed from Uranium and will have, like in Hiroshima and Nagasaki, produced fallout and rainout of Uranium particles. There is insufficient space here to do more than note that the current risk external radiation based model cannot begin to explain or predict what is found empirically. Despite the massive evidence including studies by nuclear industry and military scientists, the agencies ICRP, UNSCEAR, BEIR et al persist in their assertions that major observed effects, including chromosome aberration increases cannot be due to Uranium. Most recently there have been studies of French Uranium workers showing leukemia and lymphoma excess, lung cancer excess and heart disease at doses which are too low by some 2000-times to explain them on the basis of current risk models (Guseva Canu et al 2012, 2011, 2010). There is an urgent need to carry out research into this issue and indeed the European radiation research group MELODI have recently created a project CURE, CURE: Concerted Uranium Research Europe. In the report launching this development in March 2015 the authors write (CURE 2015)

. . . a large scale integrated collaborative project will be proposed to improve the characterization of the biological and health effects associated with uranium internal contamination in Europe. In the future, it might be envisaged to extend collaborations with other countries outside the European Union, to apply the proposed approach to other internal emitters and other exposure situations of internal contamination, and to open the reflections to other disciplines interested in the effects of internal contaminations by radionuclides.

8. Genetic damage and Chromosome damage in the Test veterans

I have been asked to comment on the discovery of significant chromosome damage in the veterans as shown by the study of Wahab et al 2008. The creation of chromosome damage following exposure to ionizing radiation has been a research interest of mine. The study of Wahab et al showed that the New Zealand Test veterans exhibited a significantly greater level of chromosome damage than controls. Since all that the veterans had in common was their shared experiences at the test sites it is clear that there must have been some common exposures to a genotoxic agent capable of causing chromosome damage. This can only have been exposure to radioactivity. There are some useful other studies which may help in showing what kind of radioactivity. The bombs were made from Uranium and when the bombs exploded, the Uranium was converted into particles contaminated the sites and which could have been inhaled following resuspension, which is a well described phenomenon. In the case of Mr Barry Smith, I understand that he was a camp barber and cut the hair of personnel who may have visited dusty areas which were contaminated with fallout or rainout. Mr Battersby, I am told, cleaned down aircraft which had flown through radioactive clouds, and also therefore would have inhaled the radioactive dust.

Chromosome damage was found in a study of Uranium workers in England (Martin et al 1991), in Gulf War veterans who were exposed to Depleted Uranium particles and where the Uranium was measured in their Urine (Schroeder et al 2003) and in Uranium miners (Zaire et al 1997). In all of these groups, the ICRP calculated dose will have been low, and yet the Uranium exposures were clearly sufficient to cause chromosome damage. This may suggest that it was the Uranium in the fallout and rainout at the test sites that caused the finding in the New Zealand veterans. I have already argued (above) that the genotoxic effects of Uranium are not properly assessed by the ICRP model.

The finding of chromosome damage in the veterans suggests that they suffered genetic or genomic damage. If this were the case, we might expect to find effects in their offspring. There are such effects. The 2007 case control study of the British Nuclear Test Veteran Association (Busby and de Messieres 2015) found a 9-fold excess risk of congenital conditions in the children, 8-fold in the grandchildren. The veterans also reported significantly high levels of miscarriages in their wives. The same levels of congenital effects in offspring were reported in the 1998 Rabbitt Roff study. I understand that one of the veterans in this case, the late Mr Battersby, on his return from the test sites, fathered twins who died shortly after birth.

9. Conclusion

The veterans were exposed to internal radioactivity. The current radiation risk model, that of the ICRP, bases its predictions and explanations on a study of those exposed to acute external radiation. There are good reasons to reject the findings of this Japanese Lifespan Study and the models based on it for internal exposures like those experienced by the test veterans. Published studies of those exposed internally to fission radioisotopes and to Uranium show that serious health effects occur at very low doses as estimated by the ICRP averaging approach to calculating dose. In some cases the differences between the outcomes predicted by ICRP and the observed effects (e.g. Uranium workers, childhood leukemia) are

greater than 1000-fold. A significant number of expert scientists and researchers in the area of radiation risk agree on this. Therefore in my expert opinion it would not be correct to deny causation in the veterans on the basis of the estimates of their doses based on the current risk model, that of the ICRP.

Witness Statement

I understand that my duty is to the Tribunal and the information in this report is truthful and accurate. I do not know any of the clients in this case. I have received no fee for writing this report but was asked to do so by Dr Busby. I reserve the right to alter my opinion following further evidence that may become available.

Schmitz - Feuerhake

Prof. Dr. Inge Schmitz Feuerhake
Hannover, Germany, Oct. 1, 2015

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Curriculum vitae Inge Schmitz-Feuerhake

Sept. 28, 1935	Born in Osnabrück, Germany
1955-1960	University education in physics and mathematics in Hannover and Würzburg, Germany
1960	Graduation in physics and mathematics for teacher's profession at secondary schools
1960-1966	Scientific assistant at the Institute of Applied Physics, Technical University of Hannover, research in the field of radioactivity detection
1966	Doctor degree in physics, thesis about the dosimetry of radioactive fallout
1966-1973	Physicist at the Medical Academy of Hannover in the Institute of Nuclear medicine, research on dosimetry and diagnostic application of radioactive nuclides, manager of a nuclear research reactor
1969	Married to Klaus Schmitz, physicist
1972	Birth of son Robert Schmitz
Since 1973	Professor of experimental physics at the university of Bremen, research on radiation dosimetry, radiation risks and health physics
1987	Death of husband Klaus Schmitz
1990	Founding member of the Society of Radiological Protection, Germany
1996	Married to Günter Helle
2000	Retired
2004	Chair of the European Committee on Radiation Risk ECRR, an independent expert board

International Publications of Inge Schmitz-Feuerhake since 1990

Claus, B., Grahmann, B., Hormann, V., Keuneke, S., Leder, M., Müller, H., Peters, E., Rieger, E.-M., Schmitz-Feuerhake, I., Wagschal, F.: Estimation of Sr-90 transfer factors for rye in podzolic soils after the Chernobyl accident. *Radiat. Environ. Biophys* 29 (1990) 241-245

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