

CLARIFYING THE PARADIGM FOR PROTECTION AGAINST LOW RADIATION DOSES: RETROSPECTIVE ATTRIBUTION OF EFFECTS *VIS-À-VIS* PROSPECTIVE INFERENCE OF RISK

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AIM

The aim of this paper is to describe a relatively recent international agreement on the vastly debated concepts of:

- (i) attributing health effects to low-dose radiation exposure situations that have occurred in the past; and,
- (ii) inferring radiation risk to low-dose radiation exposure situations that are planned to occur in the future.

An important global consensus has been recently achieved on these fundamental issues at the level of the highest international intergovernmental body, the United Nations. The General Assembly of the United Nations has welcomed with appreciation a scientific report on attributing health effects to radiation exposure and inferring risks that had been prepared by its United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). This paper presents the author's personal views on this extraordinary development.

EPISTEMOLOGICAL BACKGROUND

Before the consensus described in this paper was achieved, the epistemology on radiation risks and effects to low-dose radiation exposure situations had been amply discussed in the peer reviewed literature (González, 2011), and this part of the paper intends to enhance such review. However, it is to be underlined that this is a paper on radiation, not on philosophy. Its references to epistemology will perhaps be elementary for epistemologists and rather tedious for radiation scientists. But they are fundamental for understanding the primary issues behind attribution of radiation effects and inference of radiation risks.

Epistemology relates to the theories of knowledge especially with regard to their methods, validity

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and scope of such theories. Within the context of this paper these are the theories applied for attributing effects and inferring risks from radiation exposure situations. The underlying epistemological questions to be answered are the following (Steup, 2014):

- What are the necessary and sufficient conditions of knowledge for
 - attributing radiation effects and
 - inferring radiation risk?
- What are the sources of such knowledge?
- What is the structure and limits of such knowledge?

Understood more broadly, the epistemology of interest here is about issues having to do with the creation and dissemination of knowledge related to the attribution of radiation effects and the inference of radiation risks. This part is aiming to provide a systematic overview of the problems that the questions above raise and focus on issues relating to the limits of knowledge about radiation effects.

Attribution and Inference

In the context of this paper, *attribution* refers to the knowledge required for assigning health outcomes to past radiation exposure situations. Namely, it refers to revealing radiation health effects from precedent radiation exposure situations and therefore assigning such effects unequivocally to the situation.

Conversely, *inference* refers to the prospective process of reaching a judgment on the plausibility of harm arising from a future planned exposure situation, i.e., by deducing and conjecturing from indirect facts and reasoning, rather than revealing from explicit evidence, that the situation might be harmful.

Provability and probability

It is to be noted that the epistemology of attribution and inference is associated, respectively, with the subtly distinct concepts of *provability* and *probability*. These crucially different notions are expressed in most languages with homonym terms, because they derive from a common root – the Latin word *probare*, which means both and ‘to prove’ and ‘to probe’. This akin terminology is a recipe for confusion and requires an adequate semantics for its disambiguation.

The most common term *probability* may be used to describe how plausible would be that a planned radiation exposure be hazardous. As such, it can measure *risk* quantitatively. In fact, probability is the mean for quantifying the prospective inference of radiation risks. If previous epidemiological information on the incidence of the effect is available, such probability can be derived ‘frequentistically’, namely as the limit of the relative frequency of the effect occurrence in a large number of previous cases. If only indirect information on the plausibility of effect occurrence is available, e.g. from radiobiology or epidemiological data from no comparable situations, an ‘evidential’ probability can still be estimated as a ‘Bayesian’ inference through experts’ judgments. The latter is usually the case for prospective low-dose planned exposure situations for which factual data on frequency does not exist.

Distinctly, *provability* describes capability to demonstrate retrospectively and by evidence the actual occurrence of radiation effects. While probability is restricted to quantifying the prospective plausibility of hazardous outcomes, provability aims at demonstrating the genuineness

and validity of the causality of radiation effects and therefore it is a precondition for attesting the existence of such effects unequivocally and unambiguously. If radiation effects are retrospectively provable (namely their occurrence has been demonstrated), then actual effects can be attributed to the past radiation exposure situation. Demonstrability implies that the effect occurrence is clearly apparent and even obvious, i.e. capable of being logically revealed by showing the steps taken to create a logical argument of occurrence. Conversely, if the occurrence becomes indemonstrable it can be concluded that it is axiomatic; namely it might seemingly be obvious and manifest, a proposition regarded as being self-evidently true, but not able to be proved.

Provability is the mean for revealing retrospectively the factual occurrence radiation effects. It should be noted, however surprisingly it may be, that provability has not the same quality of quantification than probability. Paraphrasing previous thinking in this epistemological area, neither provable should be taken as a synonym of true nor improvable as a synonym of false (Gödel 1931); rather, provability should be taken as a mean for allowing qualified professionals to attest revealed effects with a high degree of confidence.

Imputation

The technical term attribution has some time been confused with the legal term *imputation*, but attribution is a synonym of imputation. While attributing means regarding *something* (e.g. health effects) as being caused by *something* else (e.g. radiation exposure), diversely, imputing means ascribing to *someone* (e.g., a nuclear employer) to cause something bad to *someone else* (e.g., causing by imprudence radiation effects to an occupationally exposed worker). Imputation is mainly related to occupational compensation claims, for example as part of a multi-stage test for legal liability associated with the causal relationship between the conduct of employers of occupationally exposed workers and the occupational harm that those workers may have experienced. These legal issues have been internationally considered (ILO, 2010) and will not be discussed in this paper.

Epistemological summary

In sum, the attribution of radiation effects is a retrospective notion based on the concept of *provability*, which involves *counterfactual logical analysis*, *demonstration* and, eventually, professional *attestation* for confirming that actual

effects have actually being incurred as a result of extant exposure situations. Conversely, the inference of radiation risk is a prospective notion associated with the concept of *probability*, usually a Bayesian probability, which quantify via experts' judgment the likelihood that harm might occur from planned exposure situations.

Radiation effects can be attributed on the bases of pathological and epidemiological facts from the outcomes of exposure situations. Conversely, radiation risk can be inferred on the basis of information on similar situations or from extrapolated radiobiological knowledge and available epidemiological data from other situations. It should be noted that epidemiological data are usually unavailable for protracted low-dose exposure situations; they are usually obtainable for relatively high doses and high dose rates and are usually expressed "frequentistically".

Under this framework, it has been concluded that there are epistemological limits for attributing actual effects retrospectively to low-dose exposure situations. However, it has also been concluded that under present knowledge, radiation risks are inferable for prospective low-dose radiation exposure situations, however small the expected doses may be, and, therefore, that ascribing nominal radiation risks to planned exposure situations for radiation protection purposes is required for reasons of duty, responsibility, prudence and precaution.

RELEVANT RESOLUTIONS OF THE GENERAL ASSEMBLY OF THE UNITED NATIONS

Following the confusing reporting of casualties attributed to the aftermath of the Chernobyl accident, the General Assembly of the United Nations took particular interest on the attributable health effects to low-dose radiation exposure situations. In its resolution 62/100 of 17 December 2007, the General Assembly encouraged UNSCEAR to submit, at its earliest convenience, a report to clarify further the assessment of potential harm owing to chronic low-level exposures among large populations and also the attributable health effects (UN, 2007a).

One year after, in its resolution 63/89, the General Assembly, endorsed an UNSCEAR's strategic plan for the period 2009–2013 to increase awareness and deepen understanding among authorities, the scientific community and civil society with regard to levels of ionizing radiation and the related health

and environmental effects as a sound basis for informed decision-making on radiation-related issues. Such a strategic objective 'highlighted the need for UNSCEAR to provide information on the strengths and limitations of its evaluations, which are often not fully appreciated. This involves avoiding unjustified causal associations (false positives) as well as unjustified dismissal of real health effects (false negatives). Specifically, there was a need to clarify the degree to which health effects could be attributed to radiation exposure' (UN, 2010). Then, in its resolution 66/70, the General Assembly called upon UNSCEAR to submit to it at its sixty-seventh session the report requested by it on the attributable health effects from radiation exposure (UN, 2011).

As a result of these developments, on 18 December 2012, the General Assembly, during its sixty-seventh session and under Agenda item 50, adopted its Resolution 67/112 on *Effects of Atomic Radiation* (UN, 2012). In this Resolution, the General Assembly commended UNSCEAR's valuable contribution to wider knowledge and understanding of the levels, effects and risks of ionizing radiation with 'scientific authority and independence of judgement' and, significantly and importantly, '**welcomed with appreciation the scientific report on attributing health effects to radiation exposure and inferring risks**' that had been requested by the General Assembly in its resolution 62/100 (UN, 2007b).

REPORTING FROM THE UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION AND EPISTEMOLOGICAL COMMENTARIES

In its fifty-ninth session on 21–25 May 2012 UNSCEAR approved its customary report to the General Assembly (UNSCEAR, 2012). In this report, UNSCEAR informed the General Assembly that it discussed substantive documents on the attribution of health effects to different levels of exposure to ionizing radiation, and on uncertainties in risk estimates for cancer due to exposure to ionizing radiation and summarizes its findings with the support of a detailed scientific annex that underpin them and that will be published separately (it is in print). UNSCEAR reached a number of conclusions on the attribution of health effects to different levels of exposure to ionizing radiation. These conclusions are quoted in bold italics hereinafter and subsequently commented by the author of this paper.

Attribution of Deterministic Effects

‘An observed health effect in an individual could be unequivocally attributed to radiation exposure if the individual were to experience tissue reactions (often referred to as “deterministic” effects), and differential pathological diagnosis were achievable that eliminated possible alternative causes. Such deterministic effects are experienced as a result of high acute absorbed doses (i.e. about one gray or more), such as might arise following exposures in accidents or in radiotherapy’

It is well known that large radiation doses may cause a substantial amount of cell killing, which may be sufficiently high to counterbalance the replenishing mechanisms and result in recognizable tissue reactions. These reactions are usually manifested a short time after irradiation but can also occur late, causing observable harm to the organ or tissue, eventually to the individual, and, in extreme cases, resulting in death. In order to reach the level of diagnosis, a given proportion of cells must be depleted. These types of effects used to be termed ‘deterministic’ because all of them were assumed to be determined to occur at doses in excess of a ‘threshold’ dose that could produce the necessary number of depleted cells. At present, they are preferably termed tissue reaction effects because they express as a response of the affected tissues and for some of them the existence of a threshold dose at high doses is not evident. Tissue reactions become significant at doses much higher than what is normally termed ‘low-dose’.

The main characteristic of tissue reactions is that they are diagnosable in the exposed individual by an experimented radio-pathologist. Therefore, these effects can be clearly attestable and therefore attributed to the exposure. The only condition for their attribution is the achievement of differential pathological diagnosis that eliminates possible alternative causes.

Attribution of Stochastic Effects

‘Other health effects in an individual that are known to be associated with radiation exposure — such as radiation-inducible malignancies (so-called “stochastic” effects) — cannot be unequivocally attributed to radiation exposure, because radiation exposure is not the only possible cause and there are at present no generally available biomarkers that are specific to radiation exposure. Thus, unequivocal differential pathological diagnosis is not possible

in this case. Only if the spontaneous incidence of a particular type of stochastic effect were low and the radiosensitivity for an effect of that type were high (as is the case with some thyroid cancers in children) would the attribution of an effect in a particular individual to radiation exposure be plausible, particularly if that exposure were high. But even then, the effect in an individual cannot be attributed unequivocally to radiation exposure, owing to competing possible causes’.

It is also well known that if cells are not killed by radiation but remain viable but mutated, i.e., with its genetic information changed, such altered cells may eventually lead to serious health consequences. If the mutations occur in somatic cells and involve modifications in genes related to the formation or prevention of malignancies, certain types of cancers may develop in the exposed individual. If mutations occur in germ cells, namely those concerned with transmitting genetic information to the descendants of the exposed individual, it is reasonable to suspect that hereditary disorders may arise. These types of effects in the individuals or in their descendants are latent in the sense that they may manifest long time after the exposure. Their manifestation is aleatory, occurring at random, and they are therefore termed ‘stochastic’.

A main characteristic of stochastic effects is that it is not feasible to associate a radiation exposure with a particular individual affected by an effect. The reason is that such effects can be originated by many causes and, until now, there is not available any biomarker that permit to associate the effects to radiation exposure. Both cancer and heritable effects are very common and can originate in multiple causes. Therefore, a qualified pathologist can diagnose the effect on the affected individuals but it is unable to associate the effect to the cause.

The demonstrability of stochastic effects can therefore be done only collectively, e.g. demonstrating the increase of the prevalence of such effects on a large exposed population when compared to a non-exposed population, throughout an epidemiological study. Demonstration is therefore collective rather than individual, and can be done by epidemiologists rather than pathologists alone. It includes at least the following distinct situations:

- Situations where a clear increase in the prevalence of some cancers is epidemiologically evident in an exposed cohort, such as that confirmed by the survivors of the Hiroshima and

Nagasaki nuclear bombing. In these situations the effect occurrence is collectively attributable, as they can be logically established with confirmable steps in a logical argument of occurrence and supported by epidemiological evidence.

- Situations where the occurrence of the effects is not only collectively attributable with the above process but also where the individual attribution is *ostensible*, namely it is apparent and most likely correct, but formally it is not necessarily true. Individual attribution can be ostensible when the ‘background’ incidence of the effect is low, and the radiosensitivity of the effect is high. A typical case of ostensible individual attribution is given when paediatric follicular thyroid cancers become epidemiologically evident in children exposed to radio-iodine.

- Situations where the occurrence of the effects are not epidemiologically apparent And the effect occurrence become *axiomatic*; this is particularly applicable to situations of low-dose radiation exposure, where attribution becomes a preposition regarded as being self-declared true but not able to be demonstrated.

In sum, stochastic effects:

- can be attributed only collectively rather than individually,
- can be demonstrated through epidemiological studies as increase in prevalence of the effects over the exposed population, and
- in some circumstances individual attribution can be ostensible, namely attribution cannot be individually demonstrated but it can be considered apparently true even if it is not necessarily so.

It shall be emphasized that for all individual cases, including those where attribution is ostensible, the attribution of stochastic effects is formally axiomatic for reasons of *counterfactuality*. Counterfactuality is the distinguishing attribute of being contrary to being actual or based on fact; to be counterfactual is to be going counter to the facts, usually as a hypothesis. The analysis of counterfactuality between radiation exposure and its effects is essential to assess whether the effects are provable at individual level. In this regard, the concept of *subjunctive conditional declaration*, sometimes also termed *remote conditional declaration* and usually termed *counterfactual conditional*, on the facts connecting radiation

exposure and its effects on an individual is particularly important. This type of declaration is particularly relevant to understand the logical links between radiation exposure and health effect.

The crucial question governing the counterfactuality of radiation effects can be formulated as follows:

Can the premise ‘*health effects have been incurred by an individual following radiation exposure*’ be explained in terms of a counterfactual conditional declaration of the form ‘*should the radiation exposure have not occurred, would the health effects have not been incurred?*’

The answer to this critical question is decisively negative for stochastic effects. In fact, if cancer develops in a given individual following a given radiation exposure, it cannot be affirmed with certainty that should the radiation exposure have not occurred, the cancer would not have occurred, because such cancer could have originated by a different cause.

Epistemological limitations of radio-epidemiology

‘An increased incidence of stochastic effects in a population could be attributed to radiation exposure through epidemiological analysis — provided that, inter alia, the increased incidence of cases of the stochastic effect were sufficient to overcome the inherent statistical uncertainties. In this case, an increase in the incidence of stochastic effects in the exposed population could be properly verified and attributed to exposure. If the spontaneous incidence of the effect in a population were low and the radiosensitivity for the relevant stochastic effect were high, an increase in the incidence of stochastic effects could at least be related to radiation, even when the number of cases was small’

Counterfactuality and demonstrability impose *de facto* epistemological thresholds on the provability of stochastic effects. The epistemological constraints are dominated by epidemiological uncertainties. The epidemiological estimates of stochastic effects are by definition uncertain, i.e., particularly indecisive at low doses. Uncertainty is a descriptor and quantifier of the epistemological limits of the epidemiological estimations. It is the concept used to qualify what is not known, reliable or definite and when data are not completely confident or sure. Two types of uncertainties are usually recognized: *aleatory*, which are due to

the stochastic variation of the magnitudes of the estimates; and *epistemic*, which are those due to lack of knowledge founded on an incomplete characterisation, understanding or measurement. Uncertainties dominate the epistemology of the multiple phenomena linking radiation-induced DNA mutations with the expression of stochastic health effects. Knowledge is uncertain simply because many facts are ignored, the available information is not fully dependable, and therefore, the conclusions derived from such information may be doubtful. This is particularly the case when the radiation dose is low and delivered at low rates. The possible physiological mechanisms associated with protracted radiation health effects can be hypothesized, even inferred, but without complete confidence or assurance that the assumptions are correct.

UNSCEAR has identified a large number of sources of uncertainty in epidemiological estimates of radiation effects. They are the cause of the *de facto* thresholds, limits, in knowledge about stochastic effects. For reasons of simplicity, it would be sufficient to analyze one of the more dominant uncertainties and assess its influence on demonstrability. These are the aleatory uncertainties in the epidemiological estimates, which are caused by the statistical power provided by dose level and cohort size.

Among the many different types of epidemiological studies, for simplicity and purpose of illustration, the type will be selected generally comparing an exposed group of people against a similar but unexposed group, usually termed 'control' group. In order to quantify the health effect in the exposed group, the difference between the number of such effects in the exposed group (E) and those in the control group (C), $E - C$, must be determined. However, since the effects are stochastic, both, E and C, fluctuate statistically usually following a normal distribution. Thus, for an epidemiologist to attest that effects have actually occurred, the difference $E - C$ should be larger (about twice) than its standard deviation, $\sqrt{E - C}$. Under this condition, the effects can be epidemiologically attested if, and only if, the radiation dose, D, is larger than the inverse square of the number of people in the epidemiological study, N, namely: $D > cN^{-2}$, where 'c' is a constant that depends *inter alia* of the background incidence of the health effects in question (Beninson, 1996). Thus, in a 'log D – log N' coordinate plane, a straight line will demarcate the area where effects can be attestable through epidemiological studies, namely they are *provable*, from that where

the actual occurrence of effects is improvable, namely axiomatic. This is illustrated in Figure 1 which presents the epistemological limitations for attesting the occurrence of stochastic effects due to aleatory uncertainty in the estimates due to the impact of dose level (D) and the cohort size (N) on statistical power.

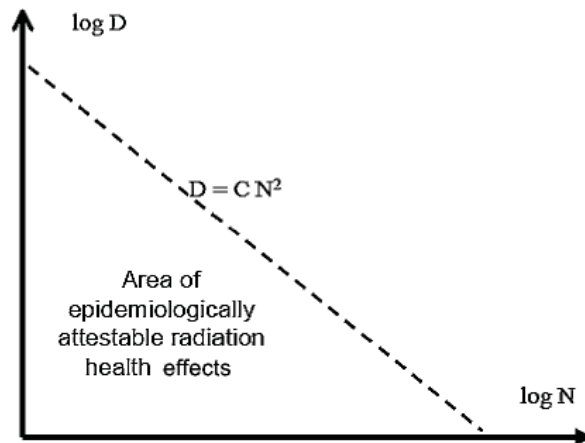


Figure 1

Attributability of Hereditary Effects

‘Although demonstrated in animal studies, an increase in the incidence of hereditary effects in human populations cannot at present be attributed to radiation exposure; one reason for this is the large fluctuation in the spontaneous incidence of these effects’

Taking into account available radio-biological information and studies in animals, UNSCEAR had made extrapolations of excess human heritable diseases in one generation due to low-dose exposure on the relevant population (UNSCEAR, 2001). UNSCEAR concluded that the excesses of heritable diseases in the first generation after a large population exposure of 1 gray are:

- for dominant effects (including X-linked diseases) ~750–1500 per million per Gy *vis-à-vis* a baseline frequency of 16,500 per million
- for chronic multi-factorial diseases ~250–1200 per million per Gy *vis-à-vis* a baseline frequency of 650,000 per million
- for congenital abnormalities ~2000 per million per Gy *vis-à-vis* a baseline frequency of 60,000 per million (chromosomal effects were assumed to be subsumed in part under the risk of autosomal dominant and X-linked diseases and in part under that of congenital abnormalities).

In sum, UNSCEAR considers that for a population exposed to radiation in one generation only, the risk of hereditary effects to the progeny of the first post-irradiation generation should be inferred to be 3000–4700 cases per Gy per one million progeny, which constitutes 0.4–0.6% of the baseline incidence of those disorders in the human population.

But UNSCEAR now emphasizes that there are not any human epidemiological evidence on such numbers. These numbers are Bayesian probabilities, i.e. expert's degrees of believe, which are assigned taking into account the available biological and animal information but cannot be used to attribute hereditary effects. Moreover, it is obvious that being the estimated risk 0.4–0.6% of the baseline incidence of those disorders, it would be statistically impossible to attribute hereditary effects to any exposure situation.

Biological Indicators of Radiation Exposure vis-à-vis Attribution

‘Specialized bioassay specimens (such as some haematological and cytogenetic samples) can be used as biological indicators of radiation exposure even at very low levels of radiation exposure. However, the presence of such biological indicators in samples taken from an individual does not necessarily mean that the individual would experience health effects due to the exposure’.

There is ample evidence that radiation exposure triggers the presence of biological indicators such as cytogenetic samples of chromosomal aberrations. These indicators are very useful for confirming and quantifying radiation exposure of individuals. However, UNSCEAR, emphasizes, there is not necessarily a correlation between these indicators and health effects actually occurring.

Attributability of Health Effects to Radiation Levels Typical of the Global Average Background

‘In general, increases in the incidence of health effects in populations cannot be attributed reliably to chronic exposure to radiation at levels that are typical of the global average background levels of radiation. This is because of the uncertainties associated with the assessment of risks at low doses, the current absence of radiation-specific biomarkers for health effects and the insufficient statistical power of epidemiological studies.’

From the epistemological limitations of epidemiology described before it becomes clear that it is unfeasible to attribute effects at low radiation doses. The provability of collective effects of radiation doses at levels comparable to the typical global average background levels of radiation, namely around several millisieverts per year, would require epidemiological studies involving cohorts of hundreds of thousands of people, which would render such studies unfeasible.

Mistaken Estimations of Radiation-induced Health Effects

‘Therefore, the Scientific Committee does not recommend multiplying very low doses by large numbers of individuals to estimate numbers of radiation-induced health effects within a population exposed to incremental doses at levels equivalent to or lower than natural background levels.’

This is an extremely important conclusion because these theoretical calculations have done a lot of harm already. Unfortunately, radiation effects have been theoretically calculated retrospectively by using risk estimates. For this purpose the nominal risk coefficients recommended by the International Commission on Radiological Protection (ICRP) for radiation protection purposes, which in turn are based on UNSCEAR estimates, have been specifically used. It is illustrative to recall the origin of these coefficients and to see why they can not be used for calculating human deaths.

The relative risk of stochastic effects had been estimated by UNSCEAR, on the basis of available radio-epidemiological studies in humans exposed to relatively high radiation doses (UNSCEAR, 2009). The extrapolated excess lifetime mortality (averaged over both sexes) is:

- for all solid cancers combined 3.6–7.7% per Sv for an acute dose of 0.1 Sv and 4.3–7.2% per Sv for an acute dose of 1 Sv
- for leukaemia 0.3–0.5% per Sv for an acute dose of 0.1 Sv and 0.6–1.0% per Sv for an acute dose of 1 Sv.

Taking into account the UNSCEAR risk estimates and its own findings the ICRP recommended ‘detriment-adjusted nominal risk coefficients’ for the only purpose of radiological protection (ICRP, 2007). These coefficients are numerals expressed in % per unit dose, which – multiplied by dose

– aim at quantifying the plausibility or ‘degree of believe’ of latent effects as a late outcome of radiation exposure. They are nominal, in the sense that they do not necessarily correspond to a real value, since they relate to theoretical calculations using hypothetical (no real) people who are averaged over age and sex. Since the different possible effects may cause distinct detriment to people, the coefficients are multidimensional, quantifying the plausible expectation of harm, and including *interalia* the weighted plausibility of fatal and non-fatal harm, and life-lost should the harm actually occur. Following a number of paradigmatic sequential steps ICRP calculated and recommended detriment-adjusted nominal risk coefficients, as follows (ICRP, 2007):

- for malignancies:
 - 5.5% Sv⁻¹ for a whole population
 - 4.1% Sv⁻¹ for an adult population.
- for hereditary effects:
 - 0.2% Sv⁻¹ for a whole population
 - 0.1% Sv⁻¹ for an adult population,

which results in a combined value of

- 5.7% Sv⁻¹ for a whole population
- 4.2% Sv⁻¹ for an adult population.

International radiation safety standards have taken the UNSCEAR estimates and ICRP recommendations into account, with a rounded overall nominal risk coefficient of ~5% Sv⁻¹. This approach forms the basis of the international requirements for protecting people against radiation in planned exposure situations (IAEA, 1996; IAEA, 2011).

It shall be noted that this approach was only aimed at planning radiation protection and never intended for assessing deaths. However, these nominal coefficients have been used in mathematical calculations and the result then assumed to be equal to proven effects and attributed to the exposure situation.

The calculations that were done are extremely simplistic: the collective dose of the exposure situation is theoretically assessed and then multiplied by the nominal risk coefficient. The unit of the result of this operation is persons (person-dose × % dose⁻¹) and it is automatically equated to the number of dead people.

These computations of dead bodies have flourished after the Chernobyl accident. Serious international organizations, such as the IAEA, WHO and the

UNDP have fallen in that mathematical trap. On 5 September 2005, they issued a press release on the finalization of the so-called Chernobyl Forum. Under the title *Chernobyl: The True Scale of the Accident – 20 Years Later a UN Report Provides Definitive Answers and Ways to Repair Lives*, the release reports that ‘A total of up to four thousand people could eventually die of radiation exposure from the Chernobyl nuclear power plant (NPP) accident nearly 20 years ago, an international team of more than 100 scientists has concluded’ (In fact, the scientist attending the Forum had not concluded that!). The connotation for the public was apparent: 4000 individuals have been confirmed to be killed in Chernobyl. After few months, on 25 March 2006, the Guardian of London published the following headline: ‘UN ignores 500,000 Chernobyl deaths: IAEA says will be less than 4000’, reporting on the same theoretical calculations with different assumptions. Even respected academies fall into the trap: the New York Academy of Sciences in its annals published the book ‘*Chernobyl: Consequences of the Catastrophe for People and the Environment*’, which concludes that some 985,000 people died of cancer caused by the Chernobyl accident (Yablokov et al., 2009).

Following the Fukushima accident in Japan a similar pattern of disparate and unfounded calculations have occurred and continue to be reported.

The dilemma confronted by specialists was how to respond unambiguously to the following questions: if the 5% per Sv is real, then: – Why is attributing real fatalities to low-dose radiation exposure wrong? Conversely, if the risk 5% per Sv is not real, then: – Why is radiation protection needed at all when doses are low? Specialists have been dealing with this conundrum somehow ambiguously basically stating that such theoretical calculation cannot be done. But many people asked themselves, why not? Why cannot the product of collective dose and nominal risk coefficients be equated to the number of attributable deaths to an exposure situation?

The new UNSCEAR report brings an ultimate answer to this dilemma. The thesis implicitly demonstrated by the report is that,

- RISKS of health effects can be prospectively inferred to low-dose planned radiation exposure situations for purposes of radiation protection, but
- ACTUAL HEALTH EFFECTS cannot

generally be attributed to low-dose radiation exposure situations just on the basis of those risks and, therefore, actual fatalities cannot be assigned on that basis.

Inference of Radiation Risk

‘The Scientific Committee notes that public health bodies need to allocate resources appropriately, and that this may involve making projections of numbers of health effects for comparative purposes. This method, though based upon reasonable but untestable assumptions, could be useful for such purposes provided that it were applied consistently, the uncertainties in the assessments were taken fully into account, and it were not inferred that the projected health effects were other than notional.’

This is a final and important conclusion: the fact that health effects cannot be attributed to low radiation doses does not mean that radiation risk cannot and should not be prospectively inferred for low-dose planned exposure situations for radiation protection purposes.

ICRP had deeply analyzed the issue of radiation risk at low levels (ICRP, 2006), and concluded that while observing that the existence of a low-dose threshold of risk does not seem to be unlikely for radiation-related cancers of certain tissues, the evidence does not favour the existence of a universal threshold. On the basis of studies of uncertainty, low-dose extrapolation and the threshold hypothesis, and by using techniques of quantitative uncertainty, ICRP analyzed the consequences of allowing for the uncertain possibility of a risk threshold.

By using the approach of *reductio ad absurdum*, the ICRP concluded that the uncertain possibility of a threshold does not drastically reduce either central estimates or upper probability limits for low dose risk compared with those obtained using a non-threshold model, unless that the possibility of a threshold be very high. This important conclusion was reached by analysing the implications of a possible, but uncertain, low-dose threshold, which are summarised by the dependence of the mean value and the upper 95% probability limit on the presumed threshold probability value. The following figure illustrates the mean and upper 95% probability limit for Excess Relative Risk

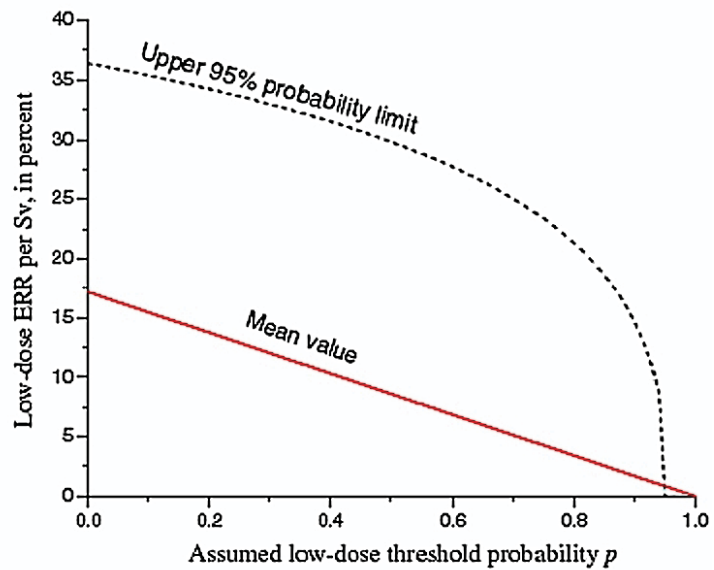


Figure 2

(ERR) per Gy as a function of threshold probability p , given (in the absence of a threshold) a log normal uncertainty distribution with a mean of 0.17 and an upper 95% limit of 0.36.

The mean value of estimated ERR per Gy is proportional to $(1 - P)$ for known threshold probability P and proportional to $(1 - E(p))$ for an uncertain threshold probability p with expected value $E(p)$. The effect on the upper 95% probability limit is less drastic, unless the assumed probability of a threshold is high. As shown in Figure 2, the upper limit decreases with increasing P , but not nearly as steeply as for the mean until P approaches the probability level of the upper limit, for example about 0.85 in the case of a 95% limit. Obviously, the lower 95% limit (the 5th percentile of the distribution) is zero for $P \geq 0.05$. Figure 3 illustrates different ‘degrees of belief (or disbelief)’ on a risk threshold applied to the cumulative distribution of probability, converting it into a cumulative distribution of plausibility. It can be seen that the plausible upper bound for threshold, of 8%/Sv, changes very little as the disbelief increase. For a large disbelief, as high as 80%, the upper bound is 5%/Sv, i.e. equal to the nominal risk coefficient used in international radiation protection standards.

In sum, taking into account the available objective evidence a nominal risk coefficient of around 5% per Sv seems to be an unavoidable prudent consideration for radiation protection purposes.

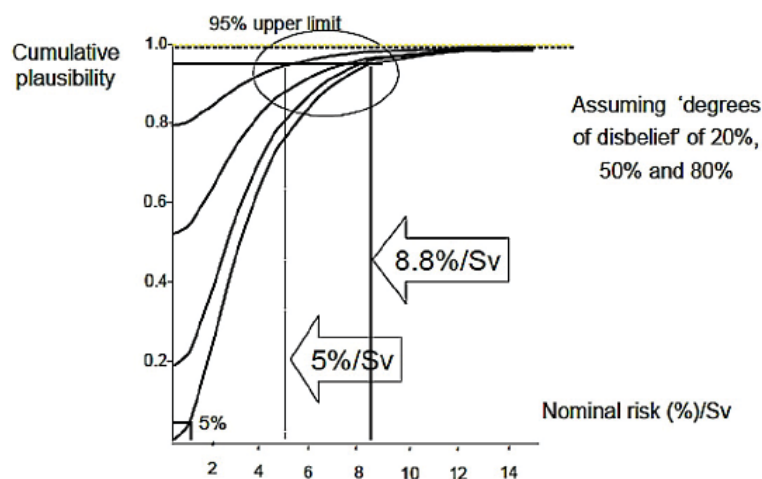


Figure 3

EPILOGUE

The highest international intergovernmental body, the United Nations, has reported the international consensus of the vastly debated issue of health effects derived from exposure situations. Such a consensus just ratifies what already was an agreement in most scientific fora. Namely:

- The attribution of radiation health effects should always be retrospective rather than prospective; conversely, radiation risk can always be prospectively inferred.
- Deterministic health effects are individually attributable to exposure situations involving high doses.
- Stochastic effects of radiation-induced cancers are collectively (not individually) attributable and only in the case that radiation doses are sufficiently high as to permit epidemiological discernment.
- Stochastic effects of radiation-induced cancers are not attributable to radiation exposure situations involving radiation doses similar to the typical levels of global average background.
- Stochastic hereditary effects are not attributable to any radiation exposure situation.
- The presence of biological indicators of radiation exposure is not synonym of radiation effects.
- The calculation of prospective radiation

effects multiplying collective doses by nominal risk factors is wrong.

- Radiation risk from planned situations can and should prospectively be inferred but only for purposes of radiation protection and allocation of resources.

The time is now ripe for regulatory authorities to convert this international consensus into commensurate instruments for regulating radiation exposure; namely: a real **drawing the line for radiation protection**.

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