A POST-LNT SYSTEM OF RADIATION PROTECTION
– DESIGN CONSIDERATIONS

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Layout

• A Selective Review of ICRP Radiation Protection System

• Some Ideas for a Post-LNT System of Radiation Protection

• Issues to Resolve
Radiation Protection is a system of principles to regulate the safe use of ionizing radiation and of its sources.

Basic Principles of Radiation Protection

- **Practice** – activity that entails, or could entail, exposure to radiation sources (i.e. exposure or potential exposure).

- **Intervention** – activity intended to reduce exposure to sources which are not part of a practice, or which are out of control as a result of an accident.
Exposures
(normal & potential)

Sources of Exposure

• **Natural** (mostly $\gamma$–rays, but also $\alpha$–particles from radon)
• **Man Made** (usually well-specified radiation fields)

Human Populations Exposed

• **Professional** (usually by repeatable & well-specified radiation fields)
• **Medical** (mostly by X-rays or $\gamma$–rays)
• **General Public** (mostly by $\gamma$–rays, but also $\alpha$–particles from radon)
System of Radiation Protection in Practices

• Justification of a practice – a practice should be adopted only if it yields sufficient benefit to the individual or society to outweigh the radiation detriment it causes.

• Optimisation of a practice – the magnitudes of exposures and the numbers of individuals exposed should be as low as reasonably achievable (ALARA), economic and social factors being taken into account.

• Dose Limits – values of effective dose or equivalent dose to individuals from a controlled practice that should not be exceeded.

• Responsibility for protection & safety – a legal system involving legal persons (users), licences (permits) qualified experts (radiation protection officers, MD’s medical physicists, etc.), national infrastructure and regulatory authorities.
Basic Safety Standards – IAEA & EU

Radiation Protection and Safety of Radiation Sources: International Basic Safety Standards

Jointly sponsored by EC, FAO, IAEA, ILO, OECD/NEA, PAHO, UNEP, WHO

General Safety Requirements Part 3
No. GSR Part 3

IAEA Safety Standards
for protecting people and the environment
Medical Exposures - Legal Basis

US:


Code of Federal Regulations Title 10, Chapter I, Parts 19, 20, 30, and 35.

European Union:

The ICRP system of evaluating radiation risk

Likelihood of health effects

Certainty (100%)

Stochastic

Deterministic

epidemiological clinical

Dose (mGy, mSv)

Risk factors

Cancer $\Rightarrow 0.005\%$ per mSv

Hereditary $\Rightarrow 0.0005\%$ per mSv

These risk factors apply only to the stochastic region

The deterministic region concerns high doses, such as applied in radiotherapy, where risk factors do not apply
The ICRP system of evaluating radiation risk

**BASIC PRINCIPLES:**

- Effects defined as stochastic or deterministic,
- Radiation protection concerns stochastic effects only
- Linear extrapolation to low doses (LNT),
- Defines the Sievert as a measure of „biological dose” relevant to human risk,
- Dose limits are established, based on accepted risk, LNT and risk factors.

**ADVANTAGES:**

- The system is quantitative and well defined mathematically,
- Effective doses are linearly additive,
- Risk factors and dose limits are well defined for legal purposes.

**DISADVANTAGES:**

- Not supported by present science
- Severely overprotective, collective dose is confusing,
- Enforces ALARA (as Low as Reasonably Achievable) principle, resulting in unnecessary costs and concern,
- Generates prohibitive costs and social radiophobia.

**Dose Equivalent in tissue:** in Sieverts (Sv)
\[ H_T = \sum R w_R D_{T,R} \]

**Effective Dose:** in Sieverts (Sv)
\[ E = \sum T w_T H_T \]
\[ E = \sum T w_T \sum R w_R D_{T,R} \]

- \( w_R \) – radiation weighting factor
- \( w_T \) – tissue weighting factor
- \( D_{T,R} \) – absorbed dose in tissue (in Gy)

**Collective dose, Dose Committment**

**Risk factors**

- Cancer \( \Rightarrow 0.005\% \) per mSv
ICRP-recommended BSS Dose Limits (EU)

**Public:** $E = 1 \text{ mSv/year}$ (against average natural background 2.5 mSv/y)
- Lens of eye: $H = 15 \text{ mSv/y}$
- Skin: $H = 50 \text{ mSv/y}$
(Health comforters: constrained to < 5 mSv/y)

**Occupational:** $E = 20 \text{ mSv/y}$
- (Averaged over 5 yr., < 50 mSv in any one year)
- Lens of eye: $H = 150 \text{ mSv/y}$
- Skin (hands & feet): $H = 500 \text{ mSv/y}$

There are no dose limits for medical exposures

In radiotherapy, radiodiagnostic & nuclear medicine procedures

**Guidance levels** determine most appropriate exposure required to obtain meaningful diagnostic image – these are recommendations, not limits
The ICRP-recommended yearly dose limits over the years 1924-1990 decreased from 700 mSv (1924) to 1 mSv (1990).

\[ 1 \text{ rem} = 10 \text{ mSv} \]
\[ 70 \text{ rem} = 700 \text{ mSv} \]
\[ 100 \text{ mrem} = 1 \text{ mSv} \]

Risk and its Social Perception

\[
\text{Risk (Expected Loss/unit time)} = \text{Probability (Loss events/unit time)} \times \text{Severity (Loss/Loss event)}
\]

**Example 1:** Over the year 2015 in Poland (38.5 mln) there were 32 967 car accidents in which 2 938 people died and 39 777 persons were injured. Per 100 car accidents, 8.9 people died and 120.7 were injured. **For an inhabitant of Poland, the yearly risk of death due to a car accident was then about** $1 \times 10^{-4}$ **and of injury due to a car accident was about** $1 \times 10^{-3}$.

**Example 2:** If the population of Poland were exposed to a dose of 10 mSv of gamma-rays, the number of hypothetical „deaths” due to cancer would be $38.5 \times 10^6 \text{ persons} \times 10^{-2} \text{ Sv} \times 5 \times 10^{-2} \text{ Sv}^{-1} = 19 250 \text{ persons. (the ICRP-103 risk factor is } 5 \times 10^{-2} \text{ Sv}^{-1})
\]

**For comparison:** over the year 2015, about 100 000 Polish inhabitants (over 55 000 males and over 45 000 females) died of cancer. **For an inhabitant of Poland, the yearly risk of dying of cancer is therefore about** $2.5 \times 10^{-3}$ **for both sexes.**

A total of 146,520 residents were evacuated from the Fukushima as a result of the government’s evacuation orders. **The number of deaths attributed to this relocation was about 1600. While these deaths were not directly due to radiation, they are real.** Due to the tsunami itself, some 16 000 people perished.

**Some 330 000 people were evacuated from the Chernobyl area.** The number of deaths caused by this immense social disruption and distress is unknown.
What’s wrong with the ICRP system?

- The observed dose-responses (effects) are not linear and likely to have a threshold or hormesis-like behaviour at low doses & dose-rates;
- RBE is known to be non-linearly dependent on endpoint, dose, LET(?), and other factors(?), so the linear calculation of the Sievert is unrealistic; e.g. the health effect of $\alpha$ – particles (Rn), in terms of Sv, is highly over-rated;
- The present ICRP dose limits are unrealistically low, against natural doses and dose-rates and against observed health effects of ionizing radiation;
- There is new biological and molecular evidence to demonstrate differences between mechanisms relevant to low dose & low dose-rate effects against higher dose & dose-rates – the border between is uncertain, but downward linear extrapolation is unrealistic;
- The LNT-based justification and ALARA principles unrealistically limit medical and industrial applications of radiation and enhance their cost;
- Consistent use of terms: radiation „risk” or „hazard”, further enhances radiophobia. Since low-dose & dose-rate radiation is likely curative, „effect” is better!

Why then do we still maintain this system?

- Because LNT assures the legal implementation of this system of radiation protection;
- Because, currently, we have no better legally implementable ideas!
The LNT Dependence of Risk on Effective Dose (acceptable level of risk and dose limit)

(adapted from draft to UNSCEAR 2012 Report, Annex A)

Figure 1. Some possible dose–response curves describing the excess risk of stochastic health effects at low doses of radiation. The choice of the dose limit depends on the choice of the acceptable level of risk and on the shape of the risk vs. dose dependence.
Approaches to development of radiogenic cancer

Conventional

Systems

(IR – ionizing radiation)

(UNSCEAR 2014)
Hierarchical Levels of Biological Systems
Increasing organization brings increasing complexity

- Organism
- Tissues
- Cells
- Molecules-DNA
- Atoms

Three signaling loops rely on electrons and molecules moving in and between cells:
- ~10^9 cells / g tissue
- ~10^11 molecules / cell
- ~2 × 10^4 atoms / molecule

Life needs ~30 elements, >99% are C, H, O, N, S, P

Physiologic Defenses against Perturbations

- Ionizing Radiation
- Metabolic Responses

Hierarchy Levels of Biological Systems

- Organism
- Tissues
- Cells
- Molecules-DNA
- Atoms

Defenses against Damage + Propagation

- Death
- Cancer
- Pathology

Disease

- Immune Response
- Inflammation
- Apoptosis
- Cell Senescence
- DNA Repair
- Scavenging of Toxins

Ludwig E. Feinendegen, Int. J. Low Radiation, Vol. 8, No. 2 (2011)
Schematic development of events leading to biological radiation effects.

- Energy deposition
- Excitation/ionization
- Initial particle tracks
- Radical formation
- Diffusion, chemical reactions
- Initial DNA damage
- DNA breaks / base damage
- Repair processes
- Damage fixation
- Cell killing
- Mutations/transformations/aberrations
- Proliferation of "damaged" cells
- Promotion/completion
- Teratogenesis
- Cancer
- Hereditary defects

**TIME (sec)**:
- 10^-15
- 10^-12
- 10^-9
- 10^-6
- 10^-3
- 10^-1
- 10^0
- 10^1
- 10^2
- 10^3
- 10^4
- 10^5
- 10^6
- 10^7
- 10^8
- 10^9
- 10^10
- 10^11
- 10^12
- 10^13
- 10^14
- 10^15

**Physical Interactions**
- Initial particle tracks
- Energy deposition
- Radical formation

**Physico-Chemical Interactions**
- Diffusion, chemical reactions
- Initial DNA damage
- DNA breaks / base damage

**Biological Response**
- Repair processes
- Damage fixation
- Cell killing
- Mutations/transformations/aberrations
- Proliferation of "damaged" cells

**Medical Effects**
- Teratogenesis
- Cancer
- Hereditary defects
By one nanosecond ($10^{-9}$ seconds) after the passage of a 5 MeV alpha particle in water, reaction and diffusion of reactive oxygen species has begun. New products are being formed and reactive radicals are being consumed. This track structure is lost through diffusion and reactions after about 1 microsecond ($10^{-6}$ seconds).
Does this really happen in cells? Ion tracks can be seen in nuclear emulsion and, as Double Strand Breaks (DSB), in cell nuclei.

**Figure 1.2.** A comparison of particle tracks in nuclear emulsions and human cells. The right panel shows tracks of different ions, from protons to iron, in nuclear emulsions, clearly showing the increasing ionization density (LET=$\Delta E/\Delta x$) along the track by increasing the charge $Z$. The left panel shows three nuclei of human fibroblasts exposed to $\gamma$-rays, Si-, or Fe-ions, and immunostained for detection of $\gamma$-H2AX$^{14}$. Each green focus corresponds to a DNA DSB. While in the cell exposed to sparsely ionizing $\gamma$-rays the H2AX foci are uniformly distributed in the nucleus, the cells exposed to HZE particles present DNA damage along tracks (one Si- and three Fe-particles, respectively), and the spacing between DNA DSB is reduced at very high-LET (Cucinotta and Durante, 2006).
ROS are chemically reactive chemical species containing oxygen. Examples include peroxides, superoxide, hydroxyl radical, singlet oxygen, and alpha-oxygen. In a biological context, ROS are formed as a natural byproduct of the normal metabolism of oxygen and have important roles in cell signaling and homeostasis. The ROS induced by ionizing radiation are biochemically similar to those that are constantly and abundantly produced in different cellular compartments, mainly mitochondria, during normal oxidative metabolism. Due to oxygen metabolism, mitochondria alone let leak out some $10^9$ ROS into the cytosol per cell per day (Pollycove and Feinendegen 2003, Hum Exp Toxicol 22:290–306). During times of environmental stress (e.g., UV or heat exposure), ROS levels can increase dramatically. This may result in significant damage to cell structures. Cumulatively, this is known as oxidative stress. The production of ROS is strongly influenced by stress factor responses. One needs to consider the effects of both endogenous and radiogenic ROS alongside with direct effects, especially on DNA. The latter effects generally are more toxic but less frequent than the first.
The ratio of metabolic (oxygen) DNA damage rate to radiation DNA damage rate from low LET background of 10 mGy/year, is about \(10^7\)

"The biologic effect of radiation is not determined by the number of mutations it creates, but by its effect on the biosystem that controls the relentless enormous burden of oxidative DNA damage. At low doses, radiation stimulates this biosystem with consequent significant decrease of metabolic mutations. Low-dose stimulation of the immune system may not only prevent cancer by increasing removal of premalignant or malignant cells with persistent DNA damage, but used in human radioimmunotherapy may also completely remove malignant tumors with metastases."

REPAIR OF OXIDATIVELY DAMAGED DNA

In the past several years it has become abundantly clear that DNA oxidation is a major consequence of life in an oxygen-rich environment. Concomitantly, survival in the presence of oxygen, with the constant threat of deleterious DNA mutations and deletions, has largely been made possible through the evolution of a vast array of DNA repair enzymes.

The 2015 Nobel Prize in Chemistry has been awarded jointly to Tomas Lindahl of the Francis Crick Institute and Clare Hall Laboratory in England, Paul Modrich of Duke University School of Medicine, and Aziz Sancar of the University of North Carolina School of Medicine for their mechanistic studies of DNA repair. They clarified biochemical mechanisms in three of the major kinds of DNA repair: Lindahl, base excision repair; Modrich, mismatch repair; and Sancar, nucleotide excision repair.
Physical Quantities & Units Relevant for Radiation Protection

- **Activity**: 1 Bq = 1 decay/sec (1 Ci = 3.7 x 10^{10} Bq)
- **Decay half-time**, $T_{1/2}$: $A(t) = A_0 \exp(-\lambda t)$; $\lambda = 0.693/T_{1/2}$
- **Absorbed Dose**: 1 Gy = 1J/1kg
  - in eV/g: $6.24 \times 10^{15}$ eV per g mass, or
  - in eV/ng: $6.24 \times 10^6$ eV per ng mass.
  (the „micromass” of 1 ng is generally taken to correspond to the average mass of a mammalian cell *in vivo*)
- **Linear Energy Transfer**: $\text{LET}$ (keV/µm in water)
  - approximate values: fast electrons: 0.2 keV/µm,
    protons: 1-100 keV/µm, C-ions: 10-900 keV/µm
- **Average Dose Rate**: Dose/time
  - Radiotherapy: 1 Gy/min, to a target mass of 0.1-1 kg
  - Radiodiagnostics: 10 mGy/0.1 sec, to organ mass of 5-50 kg
  - Background: 5 mGy/year, to a body mass of 75 kg

The ratio of dose rates: RT/Background is about $10^8$
The ratio of metabolic DNA damage rate to 10 mGy/year of background photon radiation is $\sim 10^7$.

The ratio of dose rates in radiotherapy (1 Gy/min) and of low-LET background radiation (10 mGy/year) is:

$$\frac{(1 \text{ Gy/min})}{(10 \text{ mGy/y})} = \frac{(1000 \text{ mGy/min})}{(10 \text{ mGy/525600 min})} \sim 5 \times 10^7$$

so damage from radiation ROS may not be efficiently repaired at the high dose rates applied in radiotherapy and radiobiology.

Experimental RBE-LET dependences can be reproduced by track structure modelling. Cell parameters, ion species (Z), its energy and fluence enter such calculations. Linear Energy Transfer (LET) is an important but not sufficient parameter in evaluating the effect of ionizing radiation in radiobiology and radiotherapy.
Relevant observations

• Radiotherapy and experimental radiobiology are performed at doses and dose-rates high enough to overwhelm the rate at which repair of metabolic (oxygen) DNA damage occurs;

• Track structure modelling is able to quantitatively represent the response of cells *in vitro* (and, possibly of whole simple organisms) after low-LET and high-LET radiations, at radiotherapy and radiobiology doses & dose rates;

• In terms of radiation protection, such modelling is representative of the initial (physical) stage of radiation effects, at the „high” end of dose and dose-rate – where natural repair of metabolic (oxygen) DNA damage is no longer effective, thus could represent a „worst case” scenario;

• In track structure calculations relevant for radiotherapy and radiobiology, the low-LET response (survival) of cells *in vitro* is represented by non-linear dependences, better represented by m-target than by linear-quadratic expressions– thus by power-law rather than linear extrapolation to low dose (with zero initial slope), precluding addititivy of dose & effect after any doses of X-rays or γ-rays;

• In quantitative track structure modelling some four parameters are required to characterize the biological detector (cell line). The radiation field is described by dose (for low-LET fields, such as X-rays or γ-rays) and by ion species (Z), and its energy and fluence distributions (energy-fluence spectrum) rather than by ion dose and LET alone.
Due to large statistical uncertainties, epidemiological studies have not provided consistent estimates of radiation risk for whole-body equivalent doses less than 100 mSv. Underlying dose-response relationships at molecular levels appear mainly nonlinear. The low incidence of biological effects from exposure to radiation compared to the natural background incidence of the same effects limits the applicability of radiation risk coefficients at organ equivalent doses less than 100 mSv (NCRP 2012).

The Health Physics Society advises against estimating health risks to people from exposures to ionizing radiation that are near or less than natural background levels because statistical uncertainties at these low levels are great.

The average annual equivalent dose from natural background radiation in the United States is about 3 mSv. A person might accumulate an equivalent dose from natural background radiation of about 50 mSv in the first 17 years of life and about 250 mSv during an average 80-year lifetime.

Substantial and convincing scientific data show evidence of health effects following high-dose exposures (many multiples of natural background). However, below levels of about 100 mSv above background from all sources combined, the observed radiation effects in people are not statistically different from zero.

Scientists evaluate and estimate radiation risk using several assumptions that, taken together, may lead to a range of hypothetical health risk estimates for any given exposure scenario.

For radiation protection purposes and for setting radiation exposure limits, current standards and practices are based on the questionable premise that any radiation dose, no matter how small, could result in detrimental health effects such as cancer or heritable genetic damage. Implicit in this linear no-threshold (LNT) hypothesis is the core assumption that detrimental effects occur proportionately with radiation dose received (NAS/NRC 2006). However, because of statistical uncertainties in biological response at or near background levels, the LNT hypothesis cannot provide reliable projections of future cancer incidence from low-level radiation exposures (NCRP 2001).

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1 Dose is a term used to express or quantify the amount of radiation a person or object has received. Equivalent dose to an organ or tissue is a quantity derived from the absorbed dose. Equivalent dose is used in radiation protection to relate absorbed dose to the probability of a stochastic radiation effect (cancer induction and hereditary changes) in that organ or tissue. The equivalent dose represents the sum of all of the contributions from radiations of different types multiplied by their respective radiation qualities.
My proposed LRT (Linear Regulatory Threshold) System modifying the LNT Paradigm

- NBR – natural background rate
  (if NBR=2.5 mGy/y, then 100 mGy= 40 NBR)
- The HPS „de minimis dose” of individual dose (100 mGy=40 NBR) should not be normally exceeded
- The national regulator establishes the value of Regulatory Threshold (RT < 40 NBR)
- For doses below RT risk = 0
- For doses „much above” RT the LNT risk factors apply

If individual dosimeters show yearly doses below Regulatory Threshold (RT), risk=0 is recorded. Suitable values of RT for radiation workers, general public and accidents are introduced in each country by the national regulator, given in local NBR units (but also in Sv?). Except in emergency situations, the RT should not much exceed the HPS „dose limit” of 100 mSv=40 NBR in our example. Note that collective and cumulative dose = 0 below RT.
Conclusions (1/2)

• Due to its complexity, social impact and legal implications, any changes in the system of radiation protection should be made gradually. As the first step, the Linear Regulatory Threshold (LRT) system could maintain the present linearity and the Sievert as a "biological measure of risk" – above threshold. While an internationally accepted threshold should not exceed 100 natural background units (say, 250 mGy/year), the national regulator would define the national regulatory threshold in its national background units (NBU). Use of NBU, rather than Gy (or Sv) would make the public aware of the ubiquity of natural background radiation.

• Current studies indicate high-rate production of Reactive Oxygen Species (ROS) from natural breathing of oxygen by man. The ROS induced by radiation are biochemically similar, but will dominate only at sufficiently high doses & dose rates. The dose & dose rates at which radiation-produced ROS begin to affect those repair mechanisms would provide scientific support for the choice of the Regulatory Threshold.

• Introduction of the LRT would eliminate the ALARA principle and calculations of collective and committed dose below threshold, opening the possibility of studies of beneficial effects of low-dose & low-dose rates in medicine, reducing the costs of nuclear technology and nuclear power in particular, reducing public "radiophobia", and unnecessary loss of life due to relocations and social trauma in nuclear accidents, such as those of Chernobyl or Fukushima, which are unlikely but inevitable in the future.
Conclusions (2/2)

• The dose/dose rate at the „microvolume” level is highly dependent on the track structure of ionizing radiation. RBE-LET dependences observed in radiotherapy or radiobiology most likely follow from the initial effects of physical interactions. Most cellular *in vitro* studies relevant to radiotherapy are performed at doses & dose rates orders of magnitude higher than those of natural background.

• Natural radiation relevant to radiation protection at dose & dose-rate levels below regulatory threshold, of concern to the general public, consist mainly of low-LET fields (X- or γ-ray radiation).

• Track structure calculations could serve as „worst case” scenarios, to be used in the future to guide radiation protection dosimetry of high-LET fields.

• Track structure modelling of non-linear radiation detectors may lead to development of physical detectors able to simulate the response of cells in terms of radiobiological effectiveness (RBE) in high-LET fields (supralinear TLDs, bubble detectors, nuclear track detectors, nuclear emulsions). The signal of such detectors could replace the present „dose equivalent” calculations.

• At the molecular level, dose response is non-linear and different at low or high doses and dose rates. At higher systemic levels in man, immune responses also appear to contribute to elimination of carcinogenic changes in affected cells. Further research in the low-dose area may lead to another general model of radiation-induced cancer on which to base the new system of radiation protection..
Thank you for your attention
Can c-hit detectors have RBE > 1? Yes, if c > 1!

Relative thermoluminescent efficiency of LiF:Mg,Ti (MTS-N) and LiF:Mg,Cu,P (MCP-N) detectors after their irradiation by protons of energies ranging between 10 MeV and 55 MeV. (Bilski, 2013)

TLD detectors which show supralinearity in their gamma-ray response may present with RE > 1. LiF:Mg,Ti (MTS-N) is supralinear and LiF:Mg,Cu,P (MCP-N) is not.

Waligórski & Katz, Nuclear Instruments and Methods 172 (1980)
Natural background radiation exposure in Europe
The Chernobyl and Fukushima Accidents

Chernobyl nuclear reactor - 26 April 1986

WHO, 5 SEPTEMBER 2005 | GENEVA - An international team of more than 100 scientists has concluded that a total of up to 4000 people could eventually die of radiation exposure from the Chernobyl nuclear power plant (NPP) accident over 30 years ago.

Fukushima-Daiichi nuclear power plant - 11 March 2011

1 Ci = 3.7 x 10^7 kBq
1 Ci/km^2 = 37 kBq/m^2
40 Ci/km^2 = 1480 kBq/m^2
How do Individual Medical and Accident Exposures compare?

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Adult Approximate Effective Dose</th>
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</thead>
<tbody>
<tr>
<td>Computed Tomography (CT)-Abdomen and Pelvis</td>
<td>10 mSv</td>
</tr>
<tr>
<td>Computed Tomography (CT)-Abdomen and Pelvis, repeated with and without contrast material</td>
<td>20 mSv</td>
</tr>
<tr>
<td>Computed Tomography (CT)-Colonography</td>
<td>6 mSv</td>
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<tr>
<td>Intravenous Pyelogram (IVP)</td>
<td>3 mSv</td>
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<tr>
<td>Computed Tomography (CT)-Head</td>
<td>2 mSv</td>
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<tr>
<td>Computed Tomography (CT)-Spine</td>
<td>6 mSv</td>
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<tr>
<td>Computed Tomography (CT)-Chest</td>
<td>7 mSv</td>
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<tr>
<td>Radiography-Chest</td>
<td>0.1 mSv</td>
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<tr>
<td>Intraoral X-ray</td>
<td>0.005 mSv</td>
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<tr>
<td>Coronary Computed Tomography Angiography (CTA)</td>
<td>12 mSv</td>
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<td>Cardiac SPECT (Myocardial Perfusion)</td>
<td>9.3 mSv</td>
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<td>Positron Emission Tomography – Computed Tomography (PET/CT)</td>
<td>25 mSv</td>
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<tr>
<td>Bone Densitometry (DEXA)</td>
<td>0.001 mSv</td>
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<tr>
<td>Mammography</td>
<td>0.4 mSv</td>
</tr>
</tbody>
</table>

Figure VI. Estimated district-average effective doses in the first year following the accident to adults living in districts of Fukushima Prefecture and some districts of Group 3 prefectures that were not evacuated. The effective doses include contributions from all relevant pathways and radionuclides.
Double Strand Breaks (DSB) in cell nuclei

Diagram of high and low LET tracks passing through a section of chromatin (a mixture of DNA and protein)

1990 RECOMMENDATIONS OF THE ICRP
Ionizing radiation produces tracks defined by the geometry of the energy deposition events. An incident ion loses energy by Coulomb interactions with electrons of the medium. These primary interactions lead to many low-energy secondary electrons that have short ranges and further ionize the medium in very localized regions. The rate at which an incident ion loses energy is called the linear energy transfer, LET, and is usually equivalent to the stopping power or energy loss per path length, \(- \frac{dE}{dx}\). LET is often used to describe the energy deposition density in radiation tracks. However, radiation chemical yields are not strictly dependent on LET, but rather on the localized track structure.
Radiation-induced tracks are very dynamic and evolve from their initial geometry because of the reaction and diffusion of reactive species. Any radiation-induced chemistry is dependent on both the track structure and the time that the chemistry occurs in the evolution of the track. The initial formation of the track is governed by the physics of the energy deposition by the incident ion and the transport of that energy by secondary electrons. Energy deposition and medium decomposition usually occurs within a few picoseconds. Remnants of the track structure may last up until a few milliseconds.
INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure

Differences in 10 keV Track Segments at 1 ps \((10^{-12} \text{ s})\)

10 MeV \(^1\text{H}\)

5 MeV \(^4\text{He}\)
The following figures show the evolution of the initial 10 keV section of a 5 MeV helium ion track in water.

The simulation starts at the bottom and continues with primary interactions until the incident helium ion has lost 10 keV. All primary interactions are in a straight line because of little helium ion scattering.
Secondary electrons are produced by the primary interactions. Most of the secondary electrons are low energy and do not travel far from their origin. An occasional secondary electron of high energy, a **delta ray**, will form its own track.
All secondary electrons lose energy by collisions with the medium and they are eventually thermalized and then hydrated. Hydration of the electron in water takes a few hundred femtoseconds or about $10^{-13}$ seconds.
By about 1 picosecond ($10^{-12}$ seconds) the ionized water molecules have decomposed to give a number of reactive radical species which are relevant in biological effects. The geometrical distribution of these species can be seen to strongly resemble the initial track structure.
Very little change in geometry is noticed from 1 to 100 picoseconds. The self diffusion of water occurs on about the 100 picosecond timescale so nothing can really move on shorter timescales. Some reaction occurs between neighbouring species.
INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure

By the nanosecond ($10^{-9}$ seconds) timescale reaction and diffusion of reactive species has begun. New products are being formed and reactive radicals are being consumed.
Reaction and diffusion continue with the passage of time. The competition between these two processes follows the track structure and determines much of the long time chemistry.
Radiation tracks begin to look very diffuse within a few hundred nanoseconds following the passage of the incident radiation. Details of the track structure are gone by this timescale.
The track structure is finally lost at very long times. The species produced in this track will react with added solutes in the bulk medium or with the walls of the container. At very high dose rates the species of this track will react with those of a neighbouring track.
# 1990 RECOMMENDATIONS OF THE ICRP

## $W_R$ values

Table S-1. Radiation weighting factors\(^1\)

<table>
<thead>
<tr>
<th>Type and energy range(^2)</th>
<th>Radiation weighting factor, $w_R$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Photons, all energies</td>
<td>1</td>
</tr>
<tr>
<td>Electrons and muons, all energies(^3)</td>
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<tr>
<td>Neutrons, energy</td>
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<tr>
<td>$&lt; 10$ keV</td>
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<tr>
<td>10 keV to 100 keV</td>
<td>10</td>
</tr>
<tr>
<td>$&gt; 100$ keV to 2 MeV</td>
<td>20</td>
</tr>
<tr>
<td>$&gt; 2$ MeV to 20 MeV</td>
<td>10</td>
</tr>
<tr>
<td>$&gt; 20$ MeV</td>
<td>5</td>
</tr>
</tbody>
</table>

(See also Figure 1)

Protons, other than recoil protons, energy $> 2$ MeV 5

Alpha particles, fission fragments, heavy nuclei 20

---

\(^1\) All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

\(^2\) The choice of values for other radiations is discussed in Annex A.

\(^3\) Excluding Auger electrons emitted from nuclei bound to DNA (see paragraph 26).
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$W_T$ values

Table S-2. Tissue weighting factors\(^1\)

<table>
<thead>
<tr>
<th>Tissue or organ</th>
<th>Tissue weighting factor, $w_T$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gonads</td>
<td>0.20</td>
</tr>
<tr>
<td>Bone marrow (red)</td>
<td>0.12</td>
</tr>
<tr>
<td>Colon</td>
<td>0.12</td>
</tr>
<tr>
<td>Lung</td>
<td>0.12</td>
</tr>
<tr>
<td>Stomach</td>
<td>0.12</td>
</tr>
<tr>
<td>Bladder</td>
<td>0.05</td>
</tr>
<tr>
<td>Breast</td>
<td>0.05</td>
</tr>
<tr>
<td>Liver</td>
<td>0.05</td>
</tr>
<tr>
<td>Oesophagus</td>
<td>0.05</td>
</tr>
<tr>
<td>Thyroid</td>
<td>0.05</td>
</tr>
<tr>
<td>Skin</td>
<td>0.01</td>
</tr>
<tr>
<td>Bone surface</td>
<td>0.01</td>
</tr>
<tr>
<td>Remainder</td>
<td>$0.05^{2.3}$</td>
</tr>
</tbody>
</table>

1 The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex.

2 For purposes of calculation, the remainder is composed of the following additional tissues and organs: adrenals, brain, upper large intestine, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other tissues and organs subsequently become identified as having a significant risk of induced cancer they will then be included either with a specific $w$, or in this additional list constituting the remainder. The latter may also include other tissues or organs selectively irradiated.

3 In those exceptional cases in which a single one of the remainder tissues or organs receives an equivalent dose in excess of the highest dose in any of the twelve organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder as defined above.
Absorbed Dose (Gy)

Effective Dose (Sv)

Conversion Factors (Sv Bq\(^{-1}\))

Conversion Factors (Sv cm\(^2\))

Activity (Bq)

Fluence (cm\(^{-2}\))

Effective dose by ingestion & inhalation

…too complicated?