Radiotherapy, Radio-Diagnostics and Radiation Protection – How do They Differ?

Michael P. R. Waligórski

Institute of Nuclear Physics, Polish Academy of Sciences (IFJ PAN), Kraków, Poland

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PRESENT-DAY IMAGING FOR DIAGNOSTICS AND RADIOTHERAPY

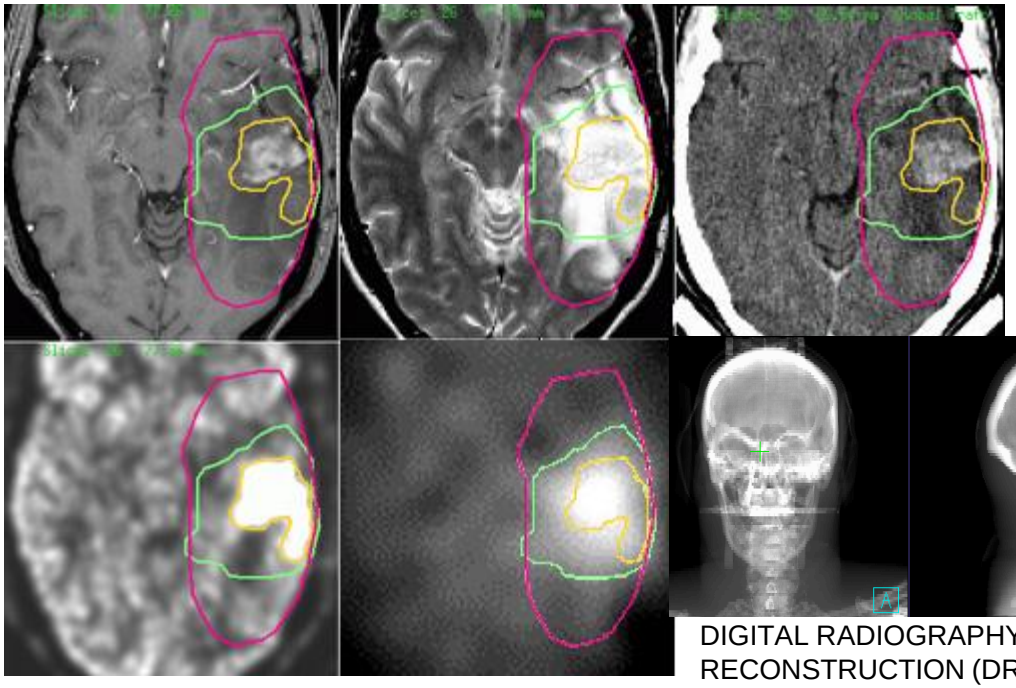
CT

PET

Various diagnostic imaging techniques (CT, MRI, PET) are currently applied in **diagnostics** and to determine the volume to be treated by **radiotherapy**

MRI

MRI (T1) MRI (T2) CT



DIGITAL RADIOGRAPHY RECONSTRUCTION (DRR)

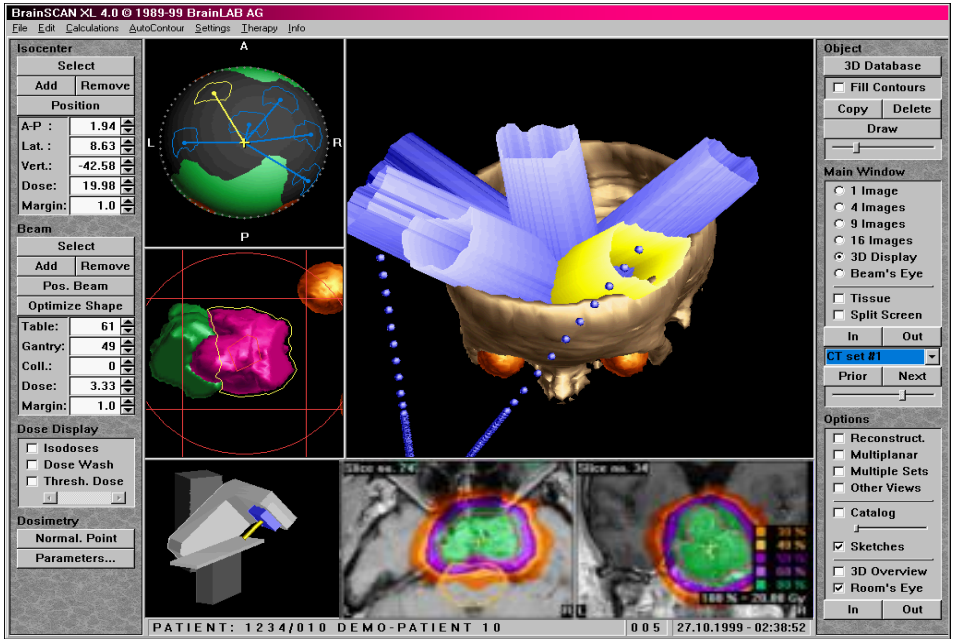
18F-FDG PET 123I-IMT SPECT

(recurrent astrocytoma in the left temporal lobe)

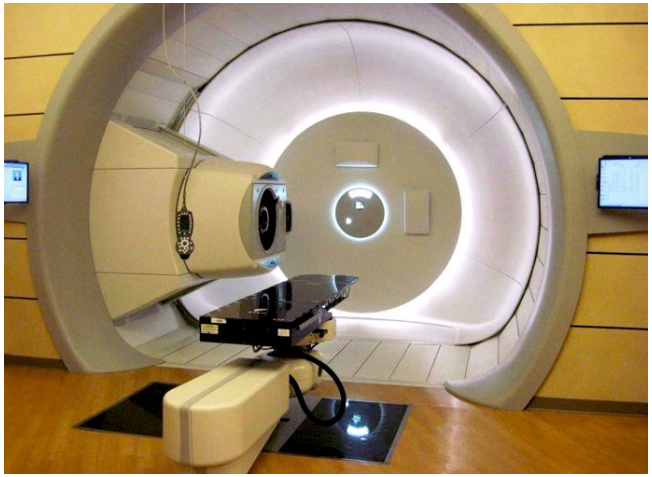
PRESENT DAY RADIOTHERAPY (MV photons, protons, C-ions)

MV X-ray medical accelerator and therapy planning system

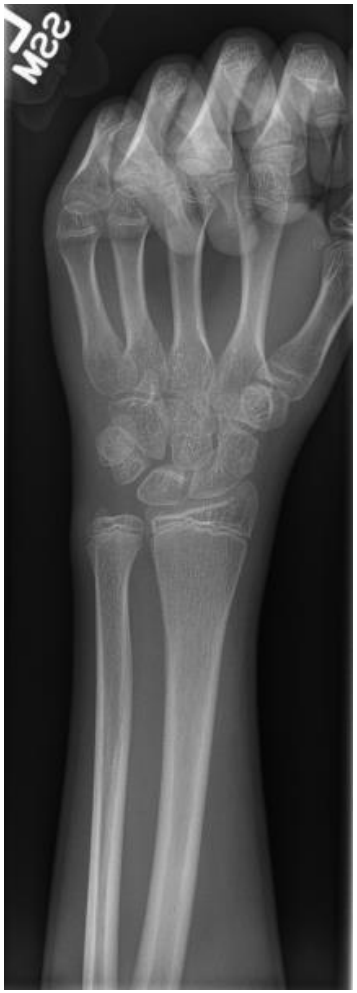
General Aim: Curative
Dose Limits: None („AHARA principle", EBM medical practice)
Mass: 0.1-1 kg (tumour volume)
Dose: ~60 Gy (in 30 x 2 Gy daily fractions)
Dose rate: 1 – 10 Gy/min @ irradiation



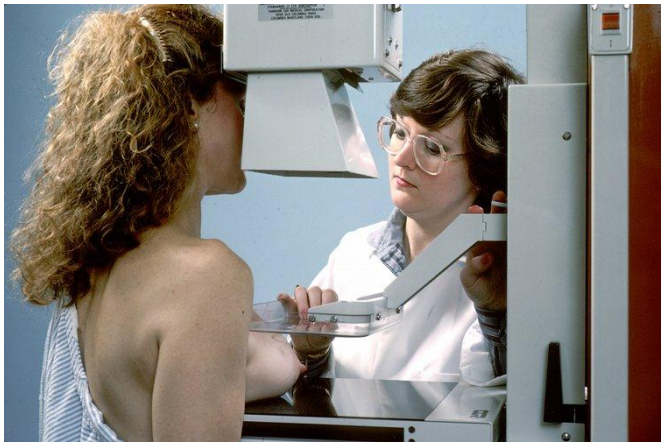
Proton radiotherapy using scanning beam and gantry – now available in Kraków (COOK+ IFJ PAN–CCB)

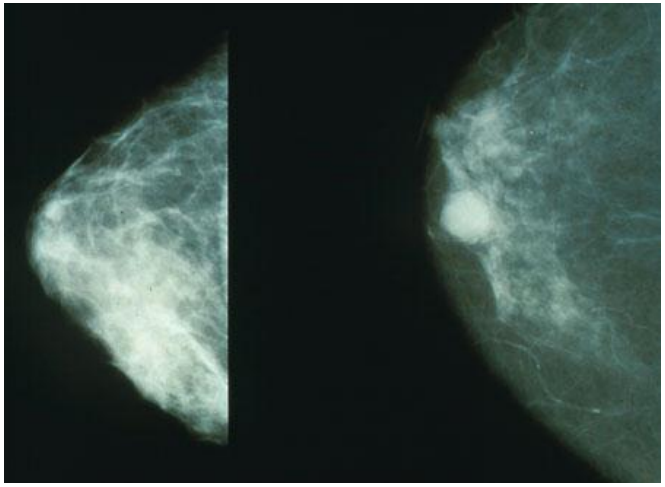


PRESENT-DAY RADIODIAGNOSTICS (Photons 30 keV – 100 keV)

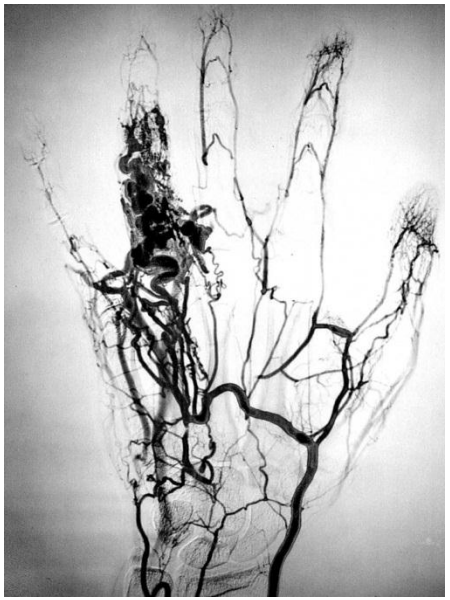


Planar
Radiography





Mammography



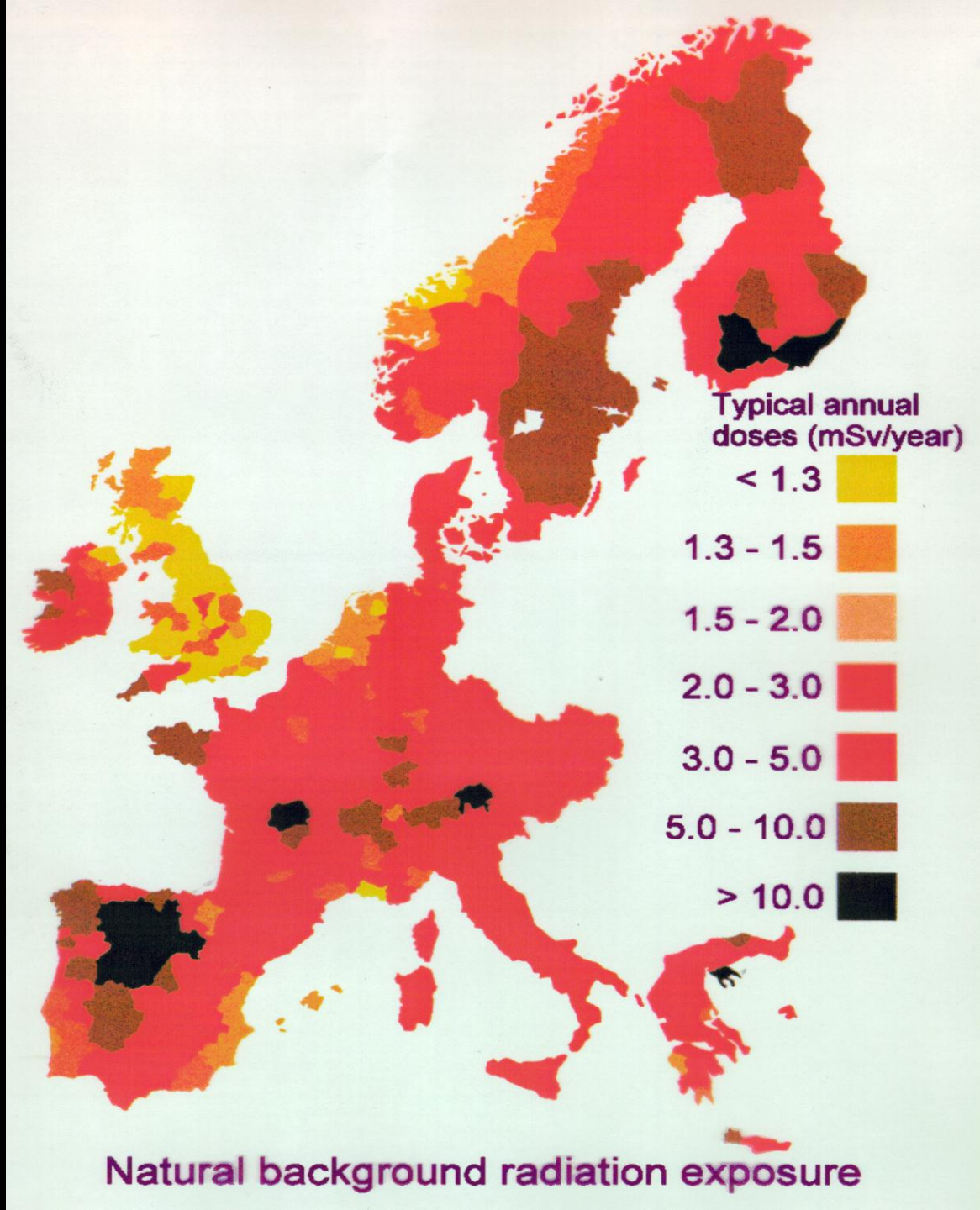
Angiography
(with iodine-based contrast)

General Aim: Diagnostics
Dose Limits: None (ALARA principle, guidance levels)
Mass: 1-100 kg (tissue-body)
Dose: 1-200 mGy
Dose rate: 10^{-5} - 10^{-3} Gy/sec

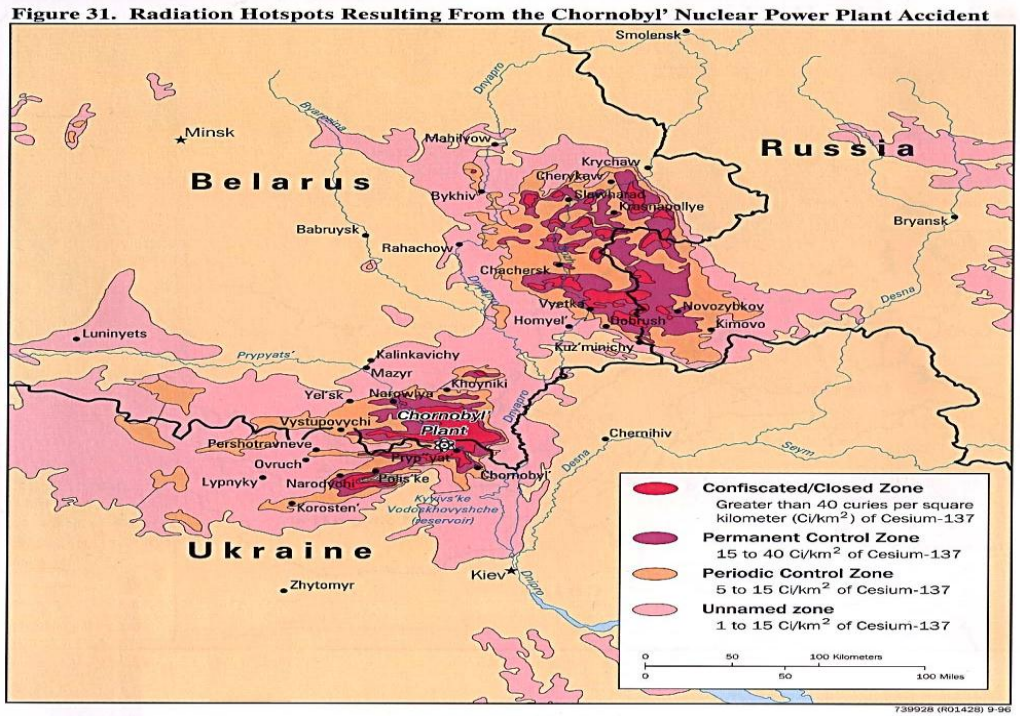
ADULT APPROXIMATE EFFECTIVE DOSES RESULTING FROM GUIDANCE LEVELS IN RADIOLOGY

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

NATURAL BACKGROUND RADIATION EXPOSURE IN EUROPE

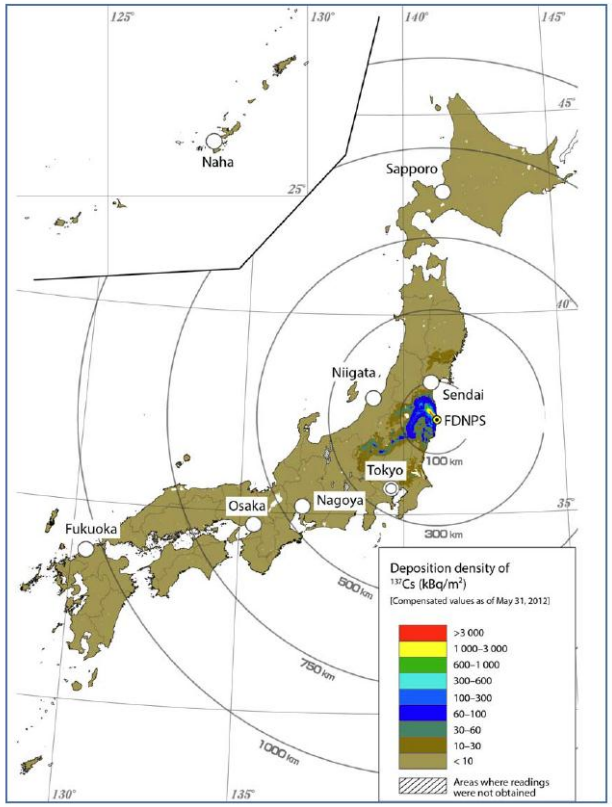


The Chernobyl and Fukushima Accidents

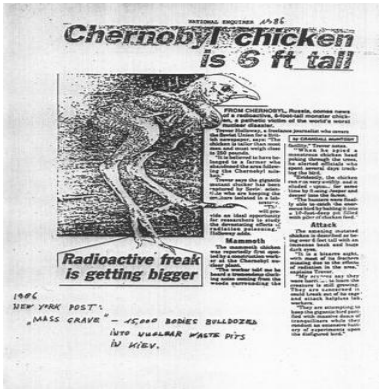


1 Ci = 3.7 x 10^7 kBq
1Ci/km² = 37 kBq/m²
40 Ci/km² = 1480 kBq/m²

Figure B-VIII. Measurement results of the airborne monitoring surveys conducted by MEXT (deposition density of ^{137}Cs) [N18]



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

How do Individual Medical and Accident Exposures compare?

Procedure	Adult Approximate Effective Dose
Computed Tomography (CT)-Abdomen and Pelvis	10 mSv
Computed Tomography (CT)-Abdomen and Pelvis, repeated with and without contrast material	20 mSv
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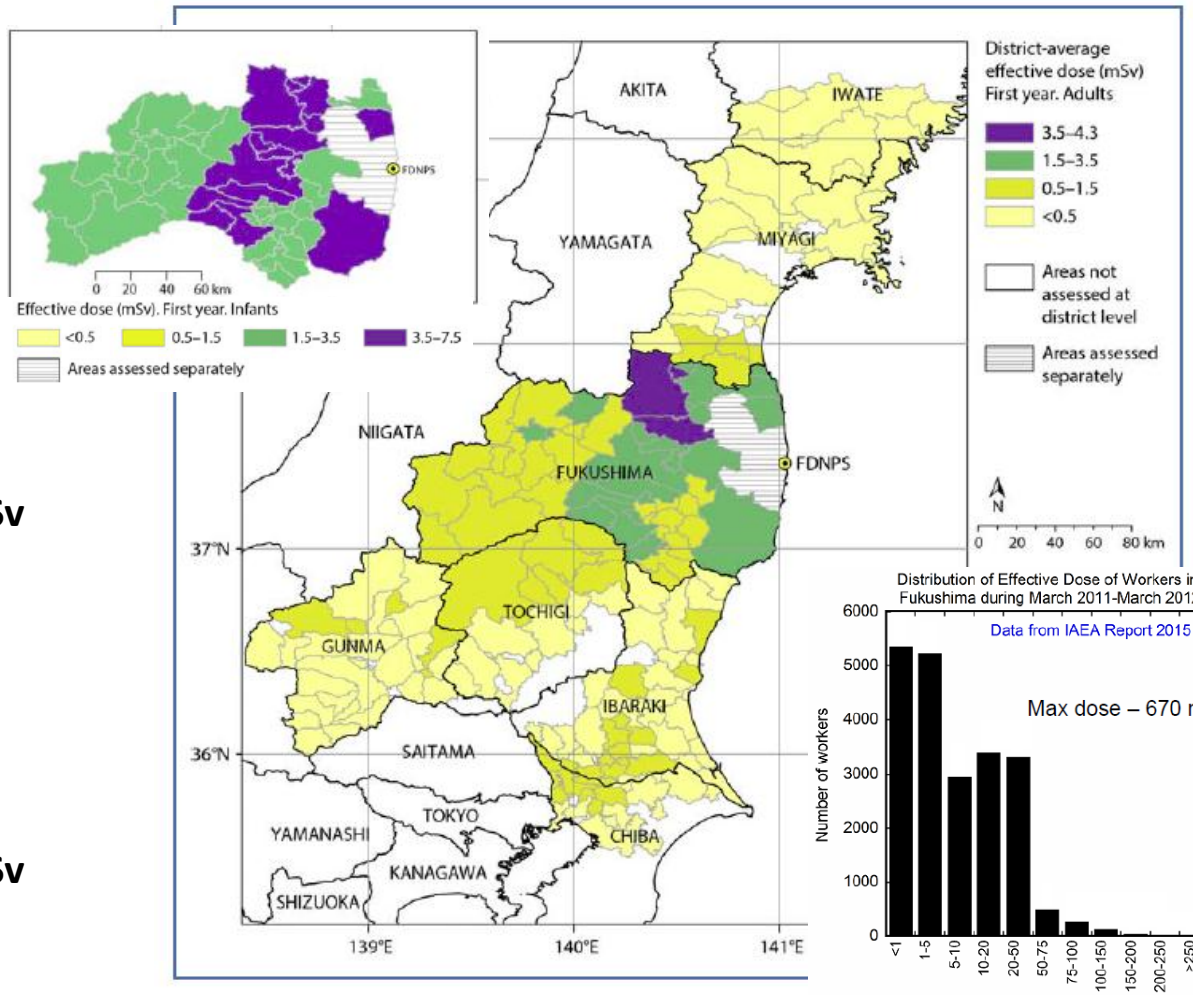
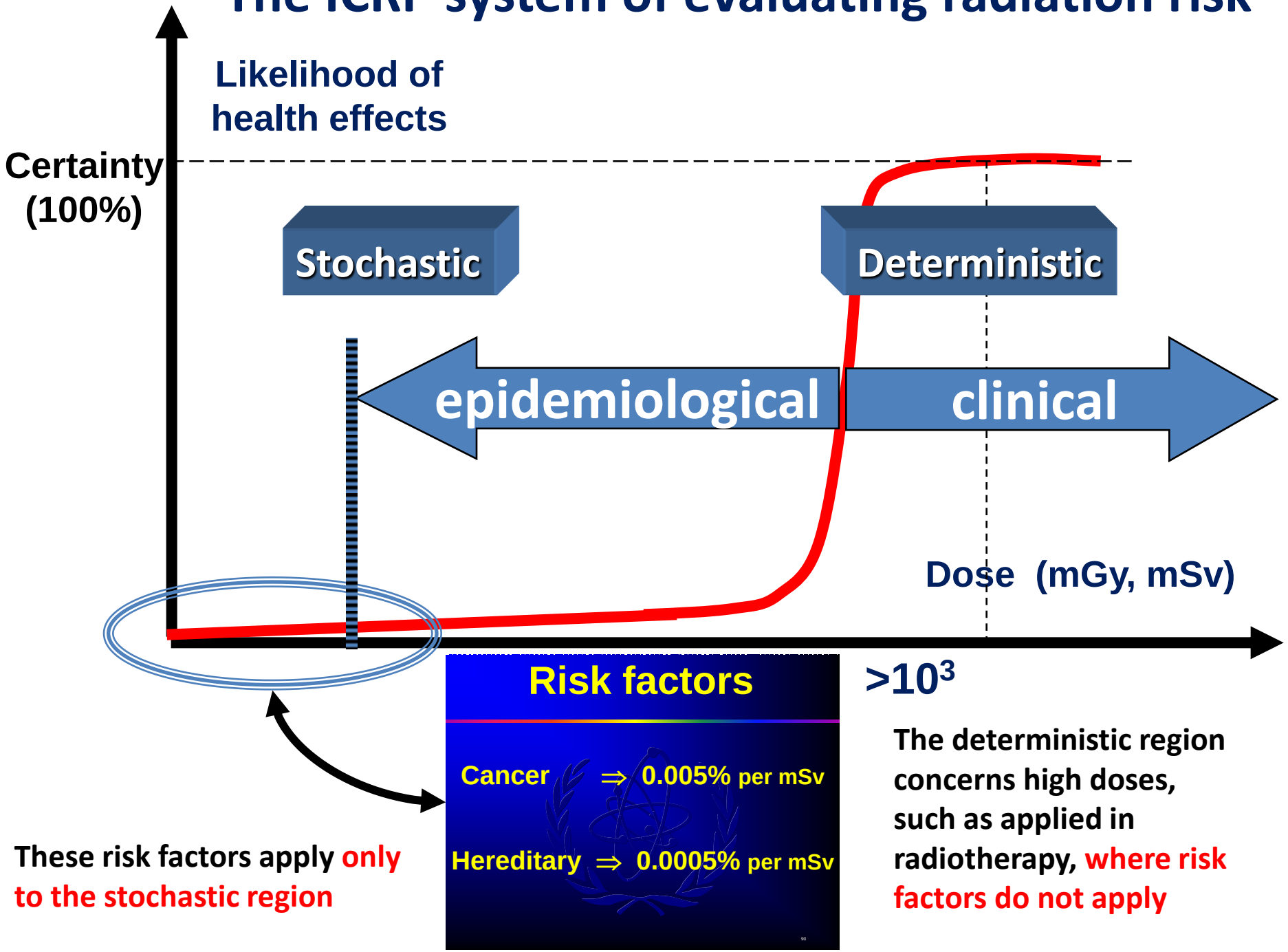
Figure VI. Estimated district-average effective doses in the first year following the accident to adults living in districts of Fukushima Prefecture and some districts of Group 3 prefectures that were not evacuated

The effective doses include contributions from all relevant pathways and radionuclides



The ICRP system of evaluating radiation risk



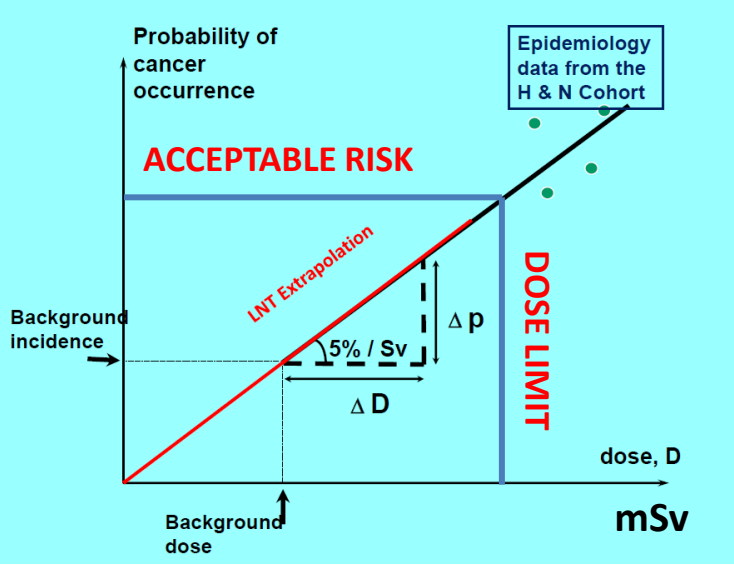
These risk factors apply **only to the stochastic region**

The deterministic region concerns high doses, such as applied in radiotherapy, **where risk factors do not apply**

The base for the ICRP system of evaluating radiation risk

The current most important evaluation of population groups exposed to radiation is the study of approximately 86 500 survivors of the atomic bombings of Hiroshima and Nagasaki at the end of the Second World War in 1945 (H&N, or **LSS-Life Span Study** data, **mainly at doses around 1-5 Gy**). Further, reliable data on the subject comes from experience with irradiated patients, and with workers after accidental exposure (e.g. Chernobyl nuclear power plant accident), and from animal and cell experiments in laboratories. The main endpoints studied were the risks of developing cancer (fatal or non-fatal) or of hereditary effects in humans exposed to relatively high doses of ionizing radiation. **The LNT (Linear No Threshold) linear extrapolation of this epidemiological H&N data to low doses has been recommended as the base of the ICRP system of radiation protection.**

The ICRP system of evaluating radiation risk



Dose Equivalent in tissue: in Sieverts (Sv)

$$H_T = \Sigma_R\, w_R\, D_{T,R}$$

Effective Dose: in Sieverts (Sv)

$$E = \Sigma_T\, w_T\, H_T$$

$$E = \Sigma_T\, w_T\, \Sigma_R\, w_R\, D_{T,R}$$

w_R – radiation weighting factor

w_T – tissue weighting factor

$D_{T,R}$ – absorbed dose in tissue (in Gy)

Collective dose, Dose Committment

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.

ICRP-recommended BSS Dose Limits (EU)

Public: E = 1 mSv/year

Lens of eye: H = 15 mSv/y

Skin: H = 50 mSv/y

(Health comforters: constrained to < 5 mSv/y)

Occupational: E = 20 mSv/y

(Averaged over 5 yr., < 50 mSv in any one year)

Lens of eye: H = 150 mSv/y

Skin (hands & feet): H = 500 mSv/y

There are no dose limits for medical exposures!

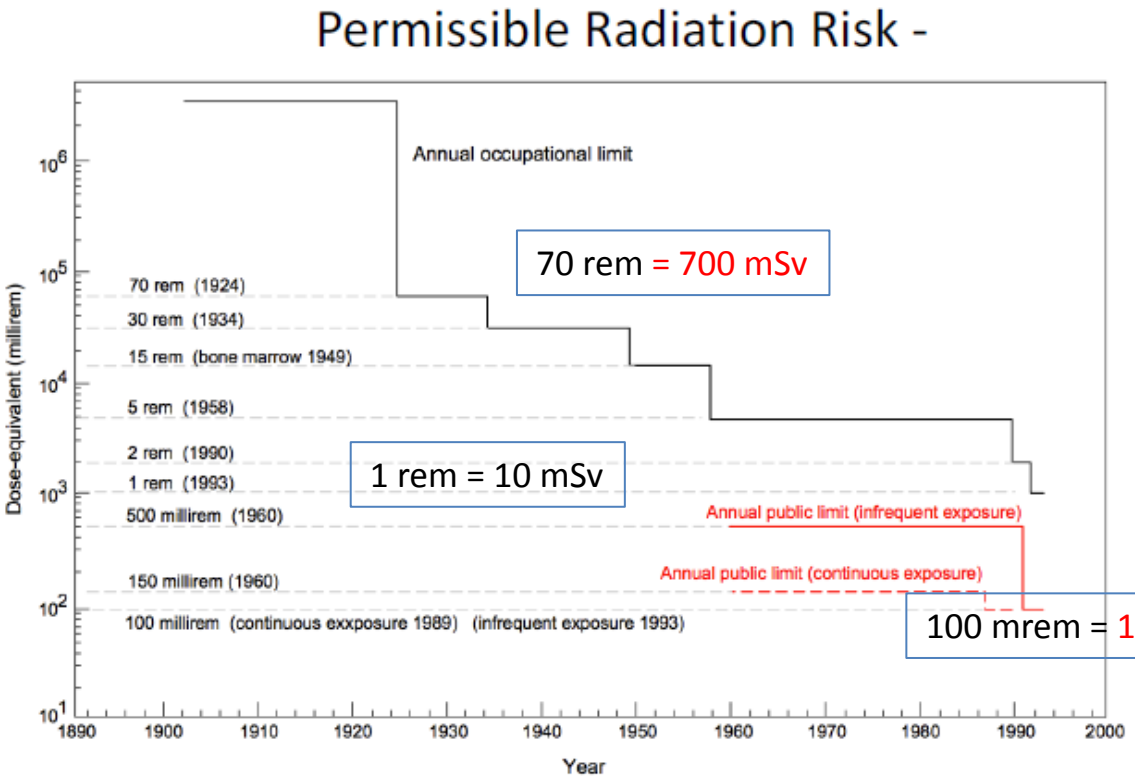
In radiotherapy, radiodiagnostic & nuclear medicine procedures

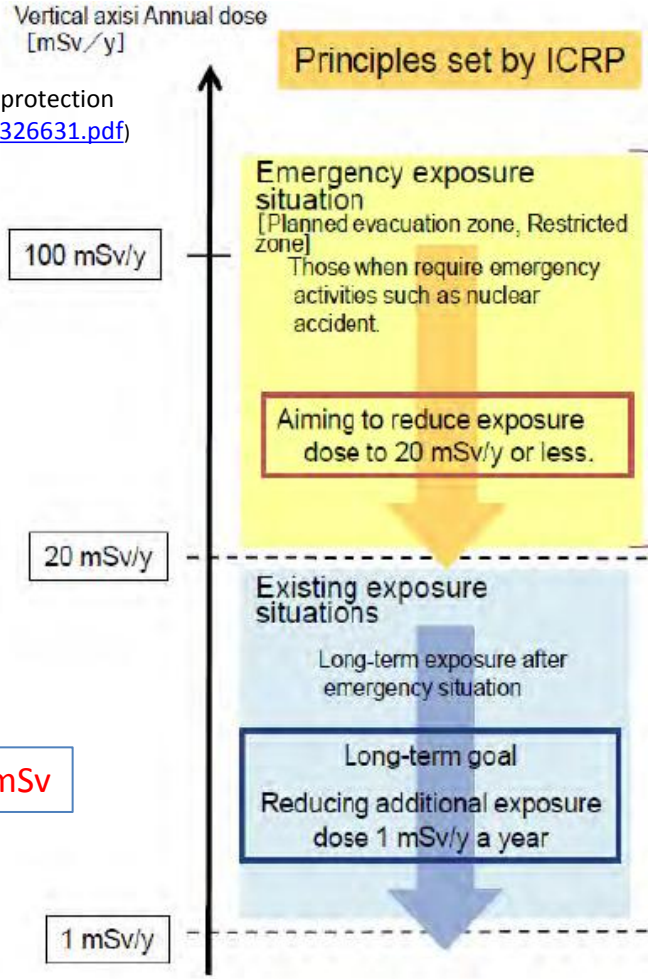
Guidance levels determine most appropriate exposure required to obtain meaningful diagnostic image – these are recommendations, not limits

The ICRP-recommended yearly dose limits over the years 1924 -1990 decreased from 700 mSv (1924) do 1 mSv (1990)

International Commission on Radiological Protection

(Inkret WC, Meinhold CB, Taschner JC. Radiation and risk—a hard look at the data. A brief history of radiation protection standards. Los Alamos Science. 1995; 23:116-123. Available at: ttps://fas.org/sgp/othergov/doe/lanl/pubs/00326631.pdf)





Risk and its Social Perception

Risk (Expected Loss/unit time) = **Probability** (Loss events/unit time) x **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 x10^{-4} and of injury due to a car accident was about 1 x 10^{-3}** . The Polish risk of death per 100 car accidents: 9.2 is the highest in the UE (Spain and Belgium: 1.8, UK: 1.2)

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 x 10^{6} persons x 10^{-2} Sv x 5x10^{-2} Sv^{-1} = 19 250 persons. (the ICRP-103 risk factor is 5x10^{-2} 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 x 10^{-3} for both sexes.**

Some 330 000 people were evacuated from the Chernobyl area. The number of deaths caused by this immense social disruption and distress is unknown.
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.

HEALTH PHYSICS SOCIETY

100 mSv – HPS Position Statement (1996-2016)

RADIATION RISK IN PERSPECTIVE

POSITION STATEMENT OF THE HEALTH PHYSICS SOCIETY*

Adopted: January 1996
Revised: July 2010
Further revised: May 2016

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[1] 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.

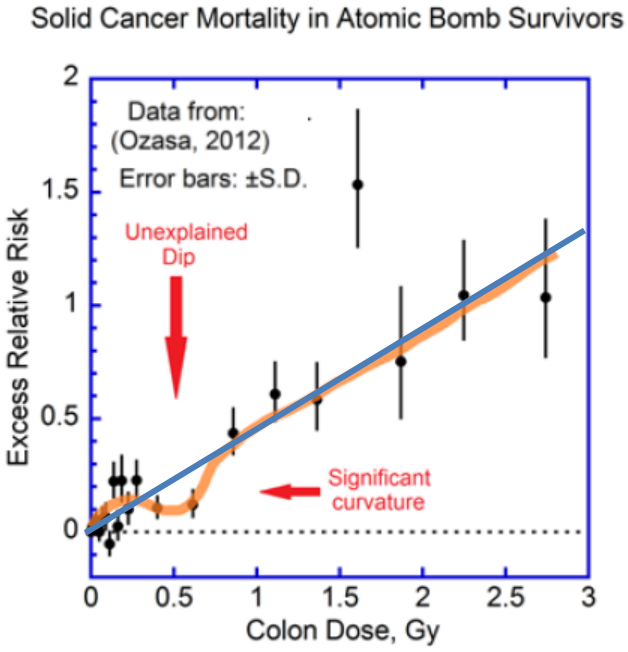
Substantial and convincing scientific data show evidence of health effects following high-dose exposures (many multiples of natural background). However, below levels of about 100 mSv above background from all sources combined, the observed radiation effects in people are not statistically different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

[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.

The Updated Atomic Bomb Survivor Data (2012) are Inconsistent with the LNT Model



Relevant Physical Quantities & Units

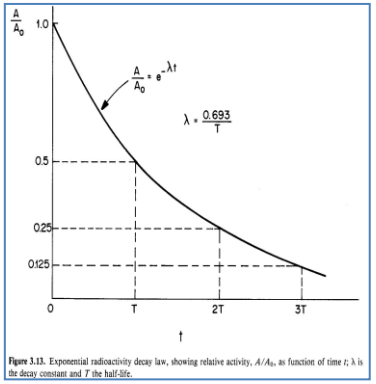


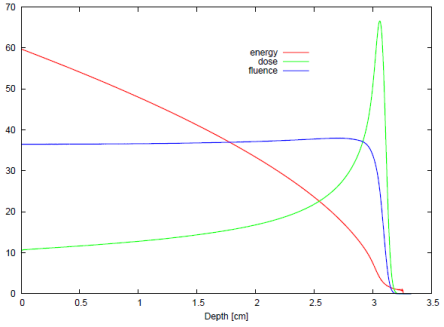
Figure 3.13. Exponential radioactivity decay law, showing relative activity, A/A_0, as function of time t; λ is the decay constant and T the half-life.

- **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 x10^{15} eV per g mass, or

  in eV/ng: 6.24 x 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: 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**

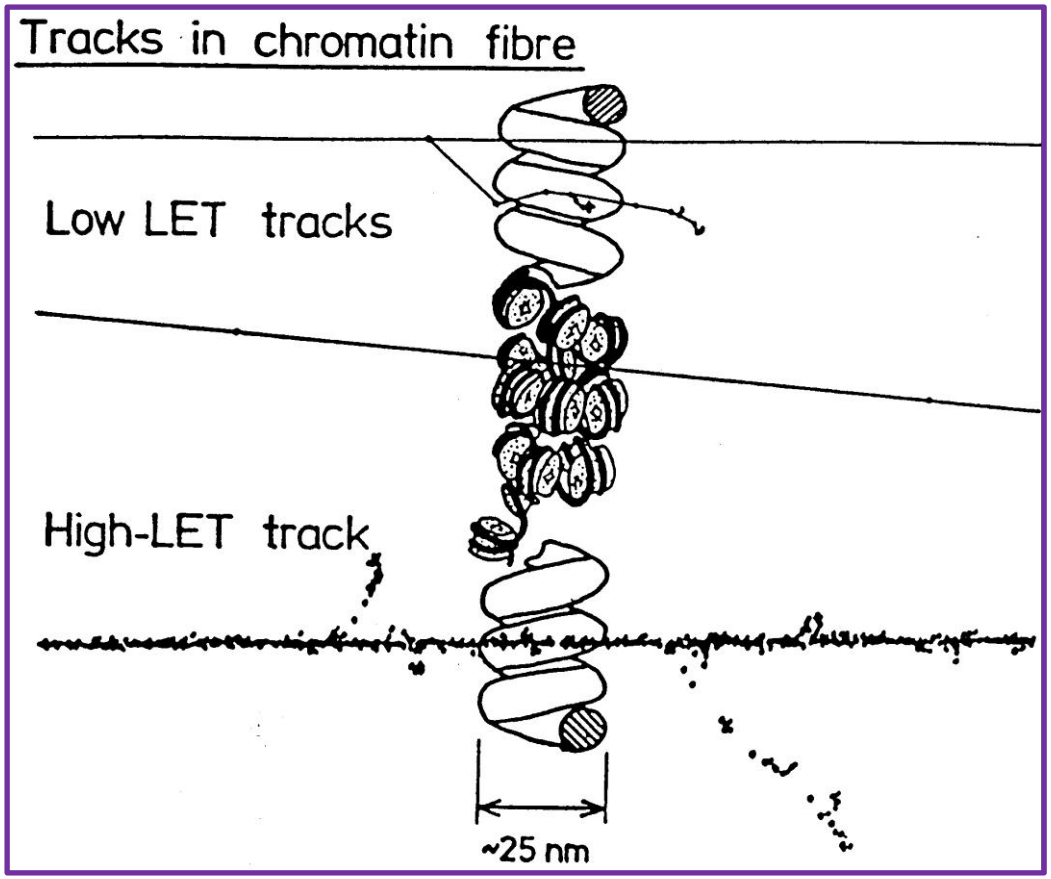
  Radiotherapy: 1 Gy/min, to a target mass of 0.1- 1 kg

  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**

Double Strand Breaks (DSB) in cell nuclei



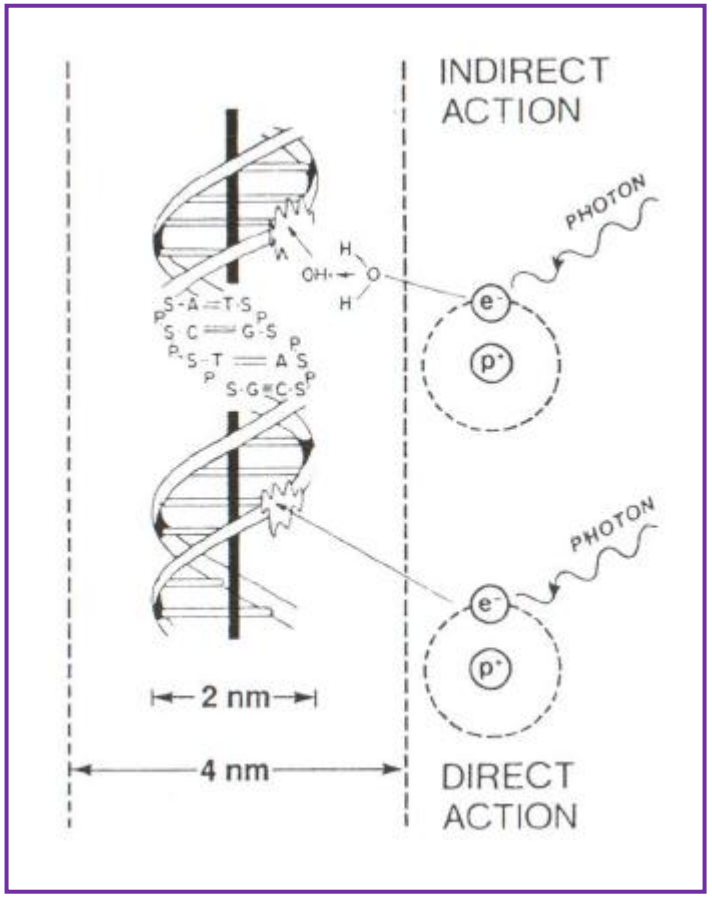
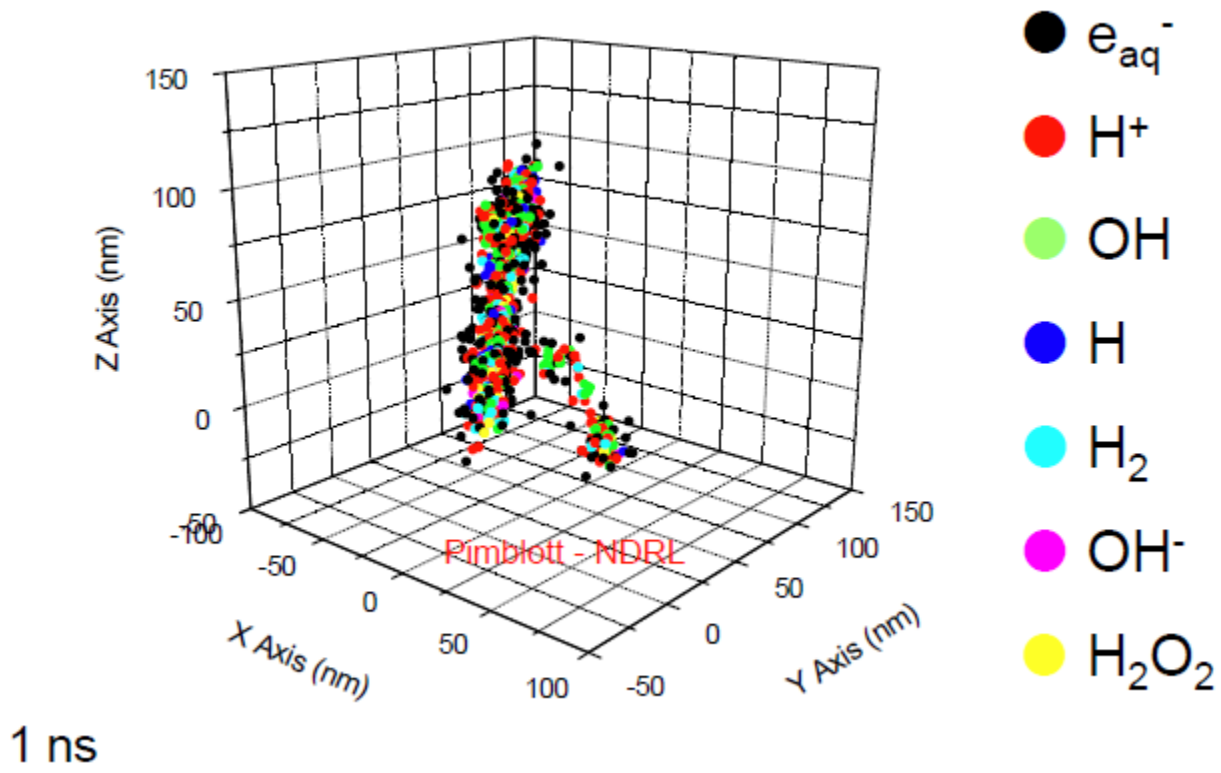


Diagram of high and Iow LET tracks passing through a section of chromatin (a mixture of DNA and protein)

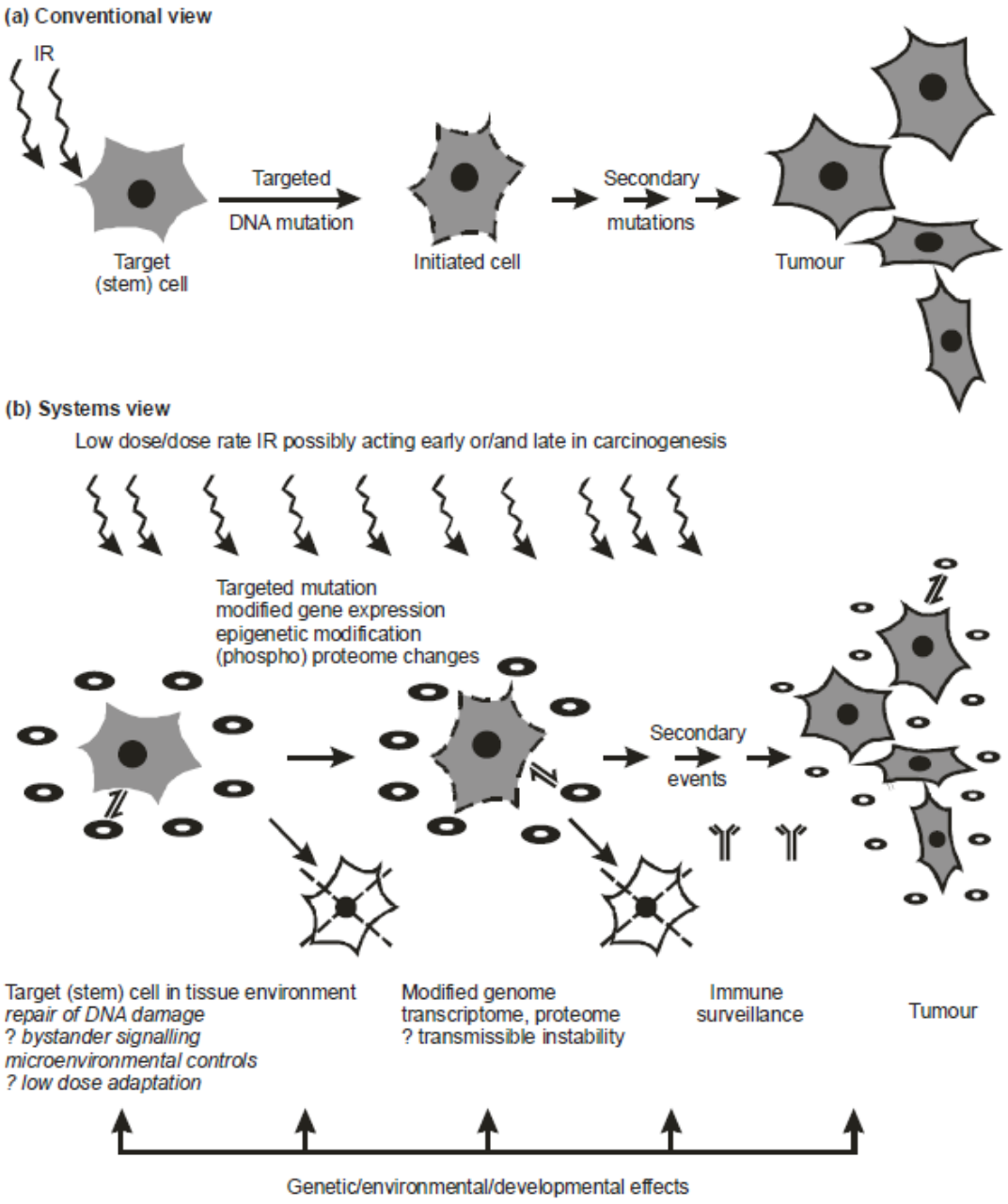
1990 RECOMMENDATIONS OF THE ICRP

INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure



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).



Approaches to development of radiogenic cancer

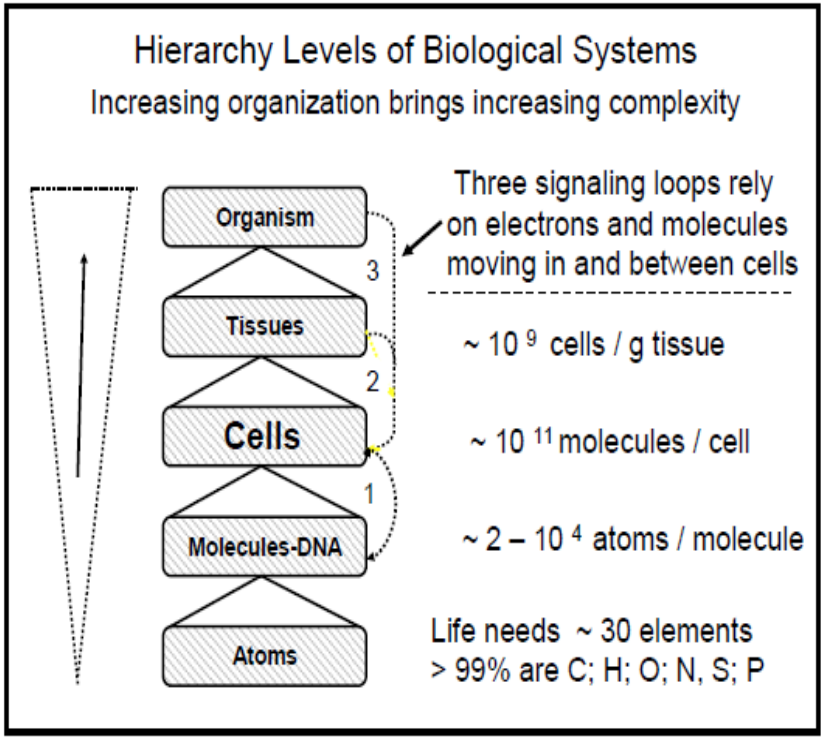
Conventional

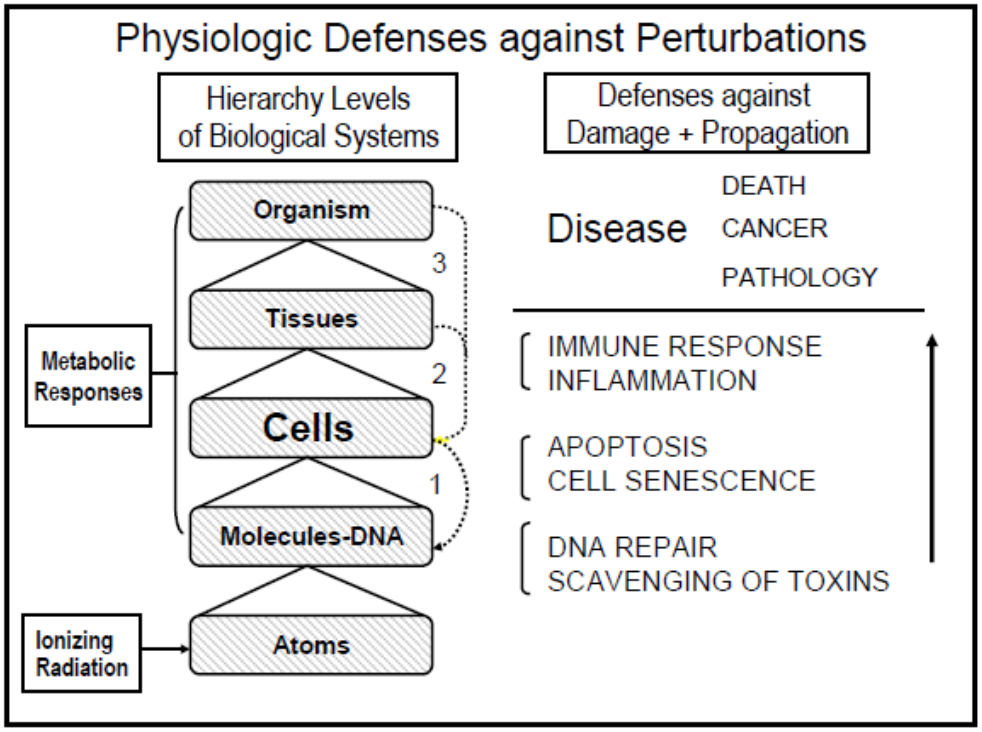
Systems

(IR – ionising radiation)

(UNSCEAR 2014)

BIOLOGICAL HIERARCHY & PROTECTION MECHANISMS

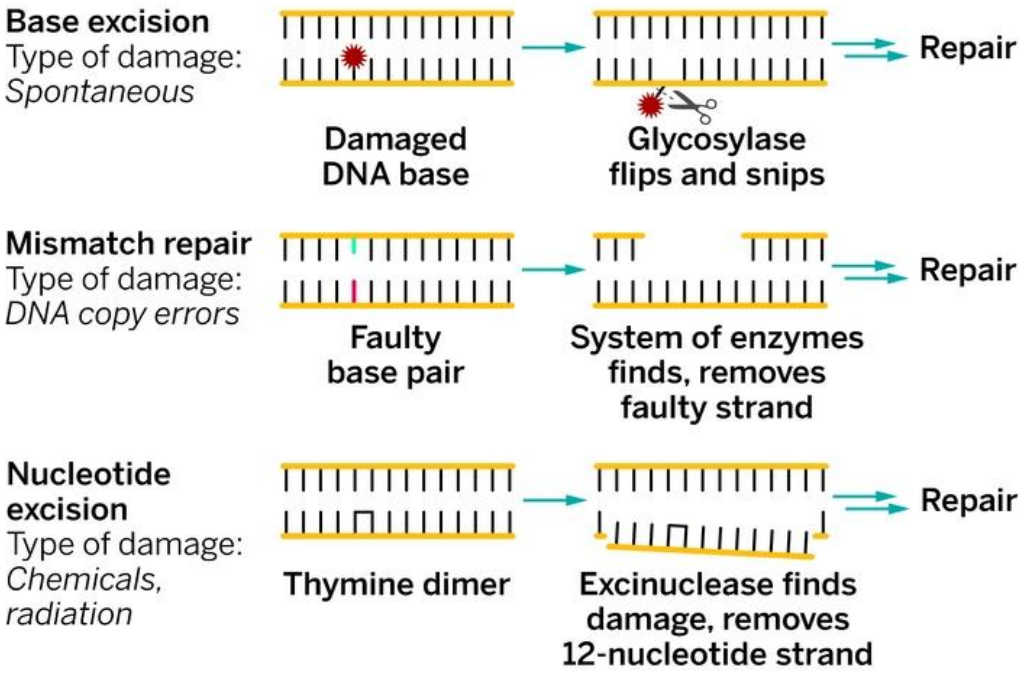




Ludwig E. Feinendegen, Int. J. Low Radiation, Vol. 8, No. 2 (2011)

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.

REACTIVE OXYGEN SPECIES (ROS)

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.

Feinendegen, Pollycove & Neumann, in *Therapeutic Nuclear Medicine, Medical Radiology. Radiation Oncology* R. P. Baum (ed.) DOI: 10.1007/174_2012_686, Springer-Verlag Berlin Heidelberg 2012

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

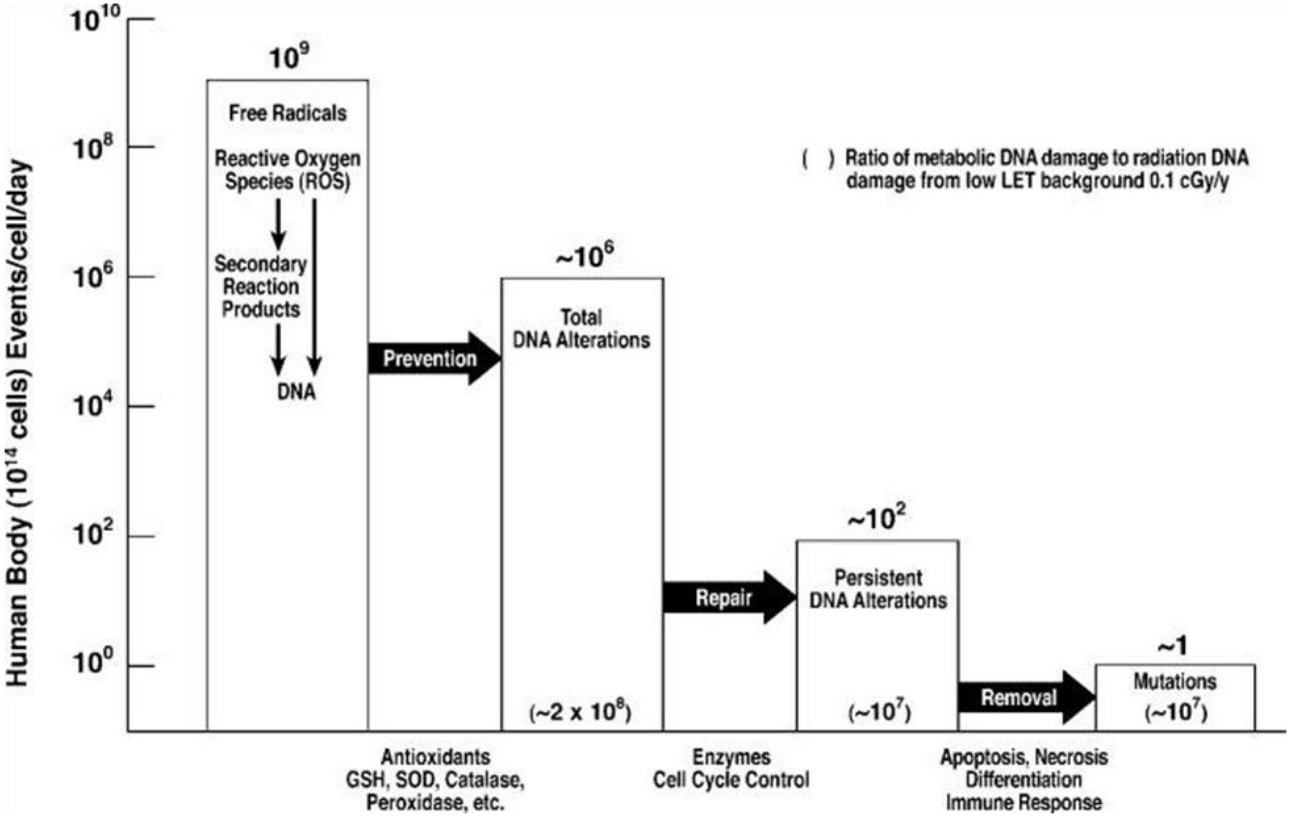


Figure 1. The antimutagenic DNA damage-control biosystem.
Estimates based on data in literature (Pollycove M., Feinendegen L.E.).

(Figure & text courtesy of Prof. Ludwig E. Feinendegen)

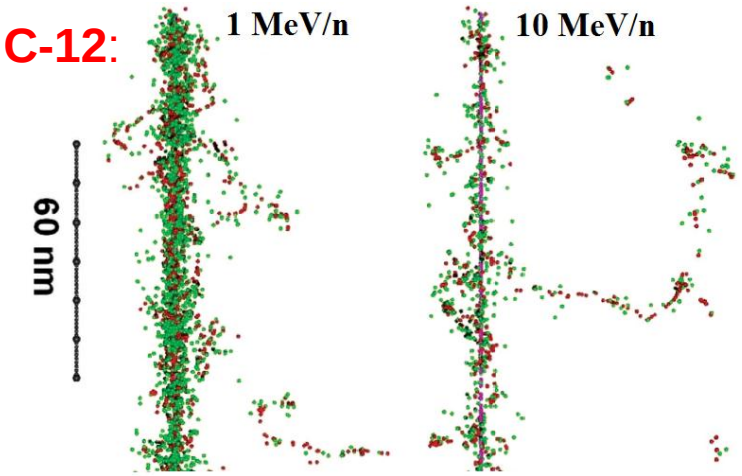
„The biologic effect of radiation is not determined by the number of mutations it creates, but by its effect on the biosystem that controls the relentless enormous burden of oxidative DNA damage. At low doses, radiation stimulates this biosystem with consequent significant decrease of metabolic mutations. 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. „
(*Pollycove & Feinendegen, C.R. Acad.Sci.Paris Science de la vie/Life Sciences 199,322 p. 197-201*)

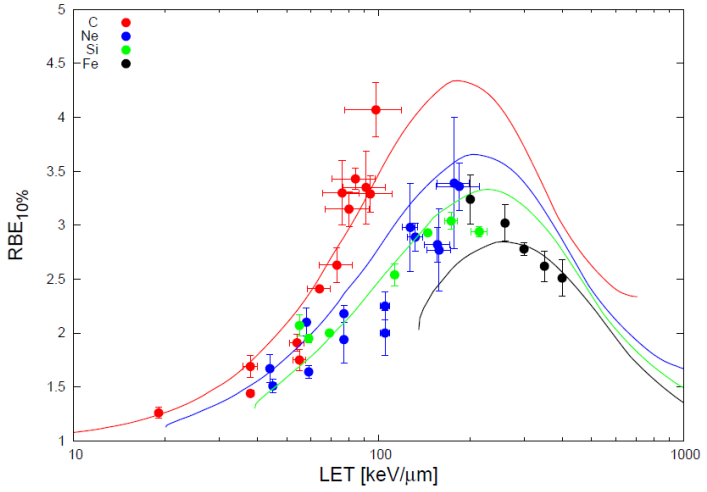
The ratio of metabolic DNA damage rate to 10 mGy/year of background photon radiation is ~ **10^7**

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

(1 Gy/min)/ (10 mGy/y) = (1000 mGy/min)/(10 mGy/525600 min) ~ **5×10^7**

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





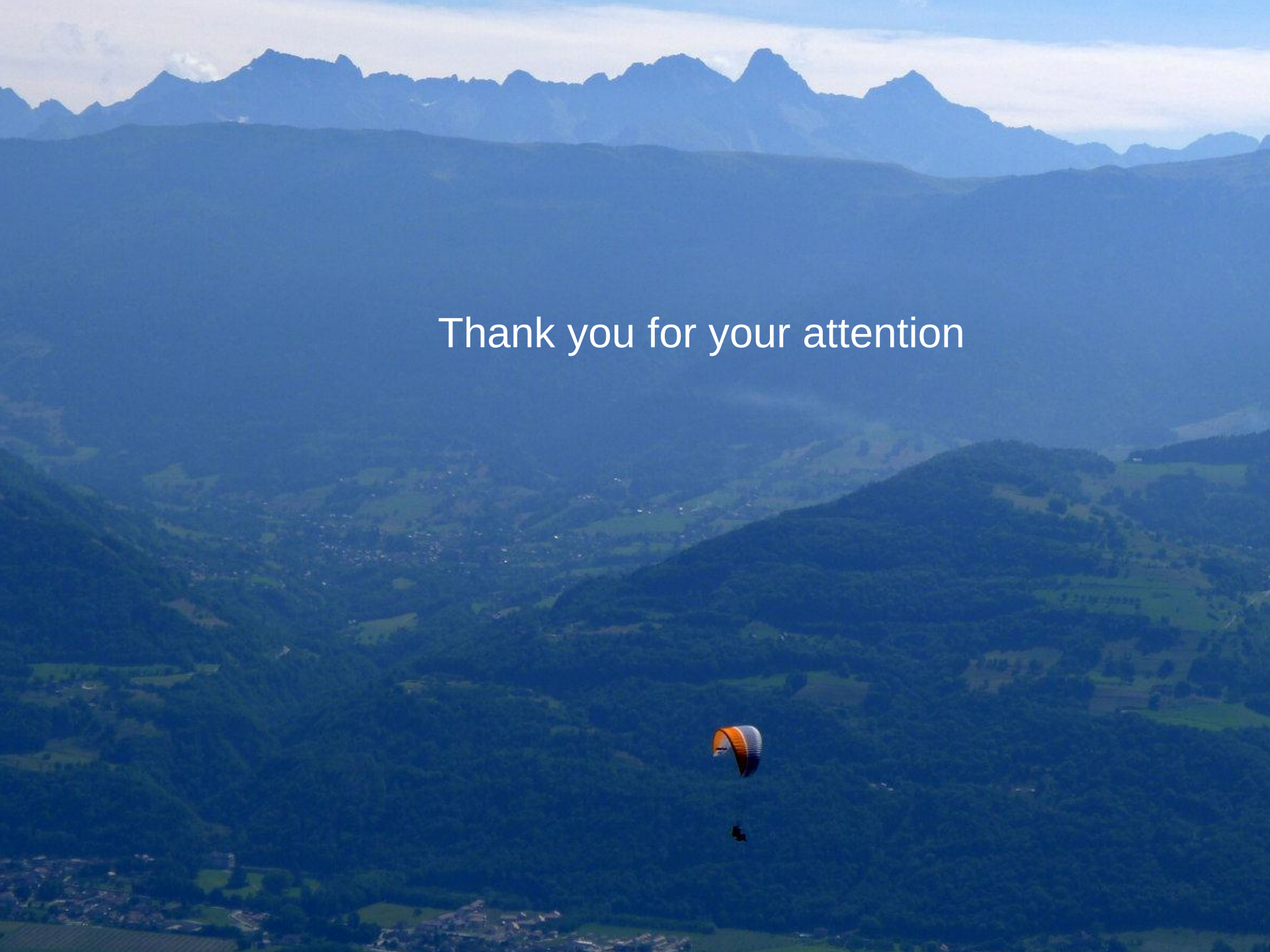
Linear Energy Transfer (LET) is an important consideration in calculation of damage rates from ionizing radiation and in track structure modelling of RBE-LET dependences

Conclusions

- Current studies indicate high-rate production of Reactive Oxygen Species (ROS) from breathing oxygen. Mitochondria alone produce some 10^9 ROS into the cytosol per cell per day, affecting DNA at that rate. The ROS induced by radiation are biochemically similar, but will dominate only at sufficiently high doses and dose rates to 1 ng „microvolumes" representing mammalian cells.
- Extremely efficient repair mechanisms of this ROS-induced DNA damage exist at the molecular and cellular levels. Oxidative stress also elicits responses at higher biological levels, in most cases preventing cancer development. One needs to consider the effects of both endogenous and radiogenic ROS alongside with direct effects, especially on DNA.
- At low doses and dose rates, cancer induction mechanisms appear not to be linear and may be different from those at higher dose/dose rates. Thresholds or hormetic responses may occur at low dose/dose rates. Thus linear no-threshold extrapolation (LNT) from higher dose/dose rates is scientifically unjustified.
- The dose/dose rate at the „microvolume" level is highly dependent on the track structure of ionizing radiation. RBE-LET dependences appear to follow from initial effects of physical interactions rather than from long-term biological effects. Most in vitro cellular studies relevant to radiotherapy are performed at high dose rates.

Conclusions (2)

- The LNT (Linear No Threshold) paradigm is no longer tenable as a *scientific* model of radiation hazard in man. At the molecular level, dose response is non-linear and different at low or high doses and dose rates. At the 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.
- The LNT-driven view that even the smallest doses contribute to radiation hazard, the ALARA principle, calculations of collective and committed dose and gradual introduction by ICRP of increasingly lower dose limits, have led to public „radiophobia”, to lack of acceptance and unreasonably high costs of nuclear power and nuclear technology, and to unnecessary loss of life due to relocations and social trauma in nuclear accidents, such as those of Chernobyl or Fukushima.


Thank you for your attention

The LNT Dependence of Risk on Effective Dose (acceptable level of risk and dose limit)

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

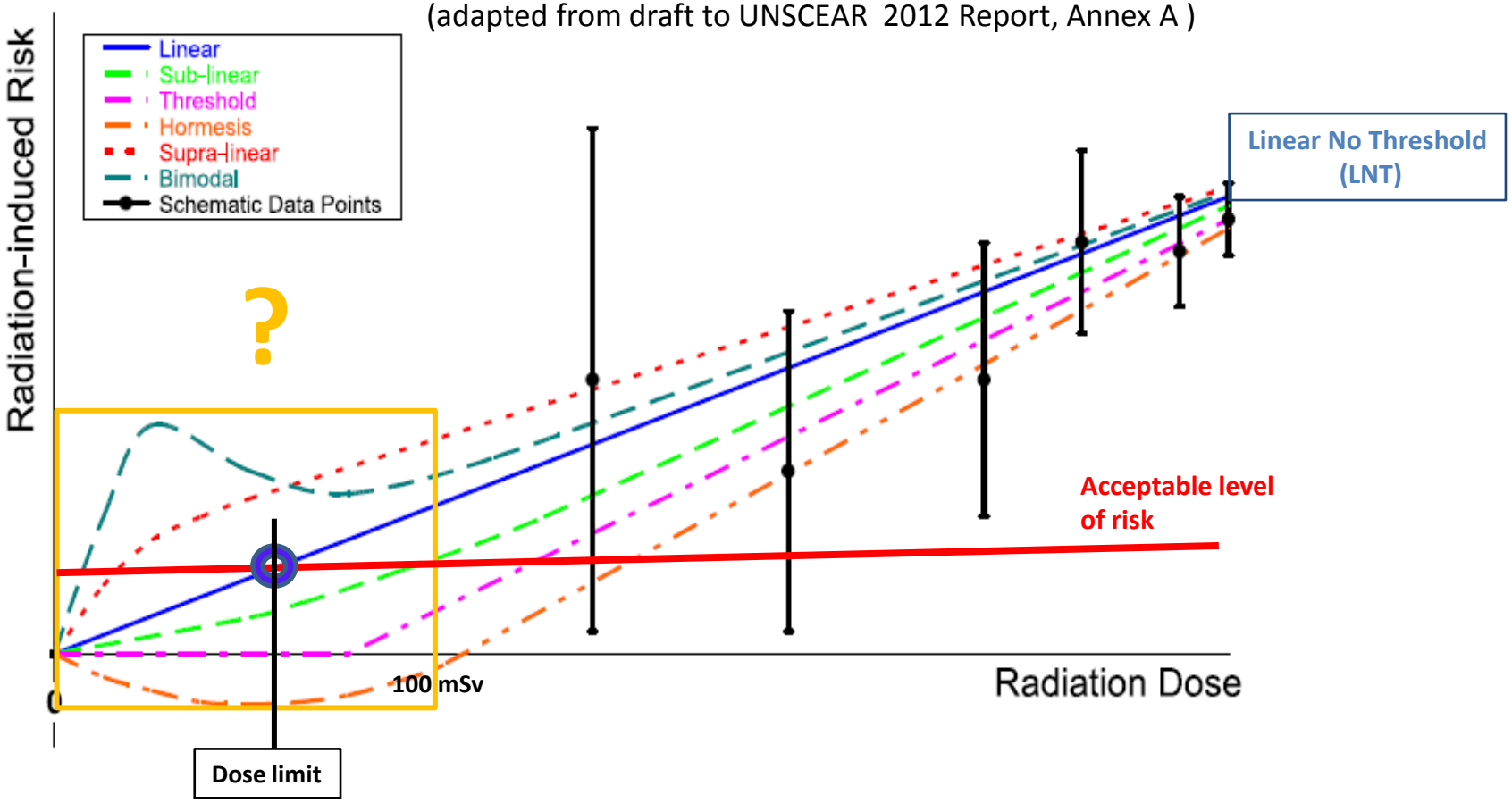
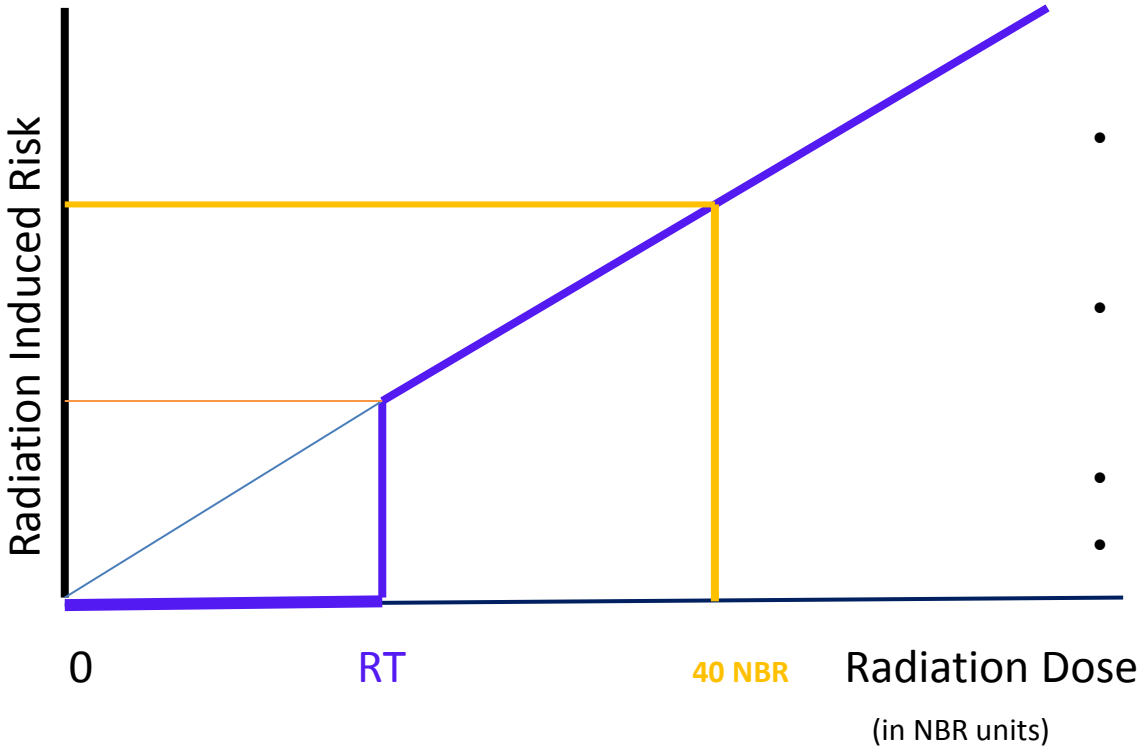


Figure 1. Some possible dose–response curves describing the excess risk of stochastic health effects at low doses of radiation.

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**.

My proposed LRT (Linear Regulatory Threshold) System modifying the LNT Paradigm



- **NBR – natural background rate**
  (if NBR=2.5 mSv/y, then
  100 mSv= 40 NBR
- **The HPS „dose limit" of individual dose (100 mSv=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 above RT the LNT risk factors apply**

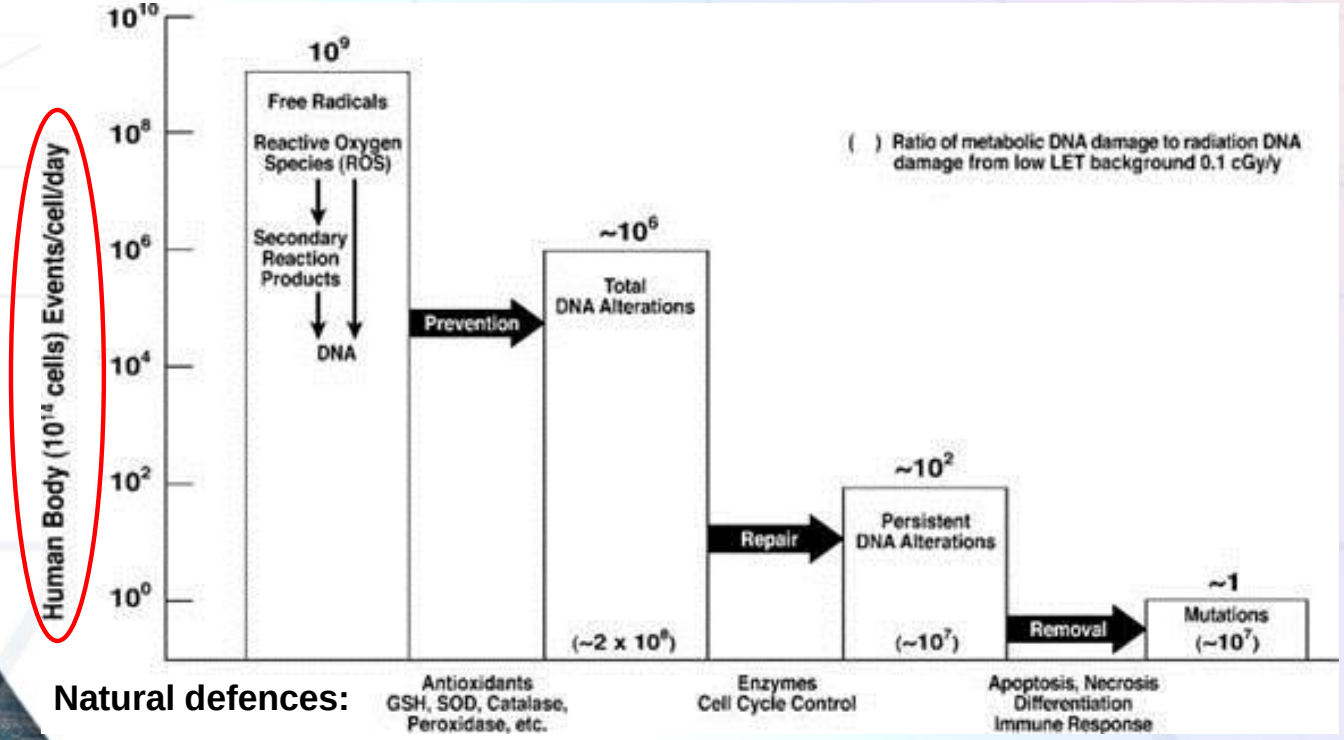
If individual dosimeters show yearly doses below RT, risk=0 is recorded. Suitable values of RT for radiation workers, general public and accidents are introduced in each country by the national regulator, in Sv, but also given in local NBR units. Except in emergency situations, the RT should not exceed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

The ratio of metabolic DNA damage rate to radiation DNA damage from low LET background of 10 mGy/year, is about 10^7

http://het.sagepub.com/content/22/6/290

Feinendegen-Pollycove model

DNA damage rate only from oxygen (no radiation)



Organisms react oxygen and glucose for energy; however, oxygen also damages their biomolecules

(Slide courtesy of Dr. Jerry Cuttler, SARI)

INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure

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,- 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.**

INTERACTION OF IONIZING RADIATION WITH MATTER

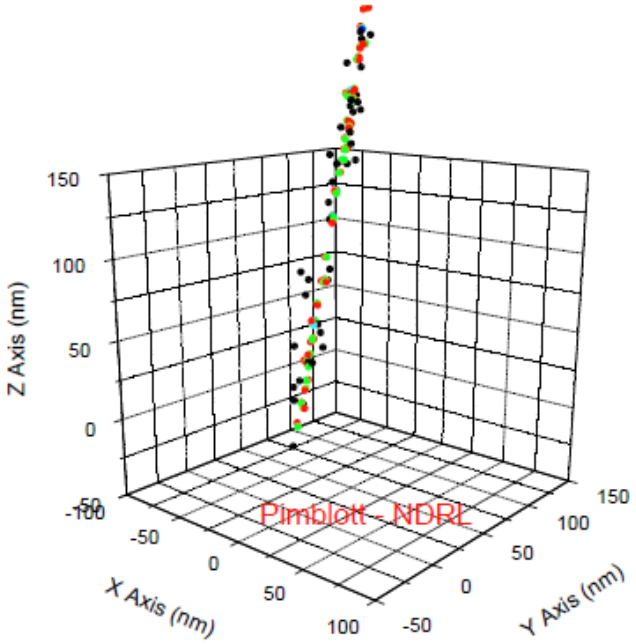
Track Structure

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

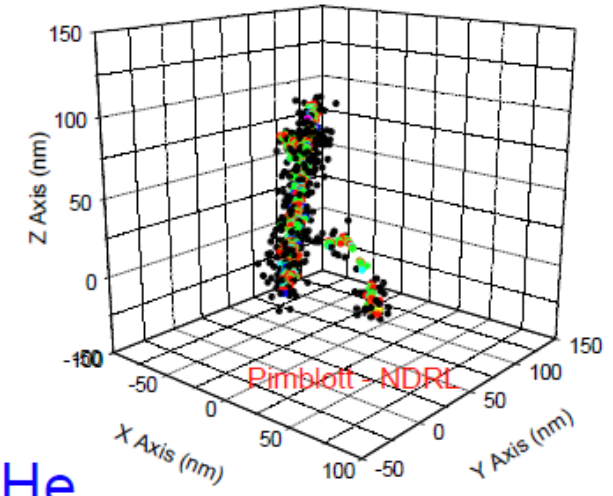
INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure

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



10 MeV ^{1}H

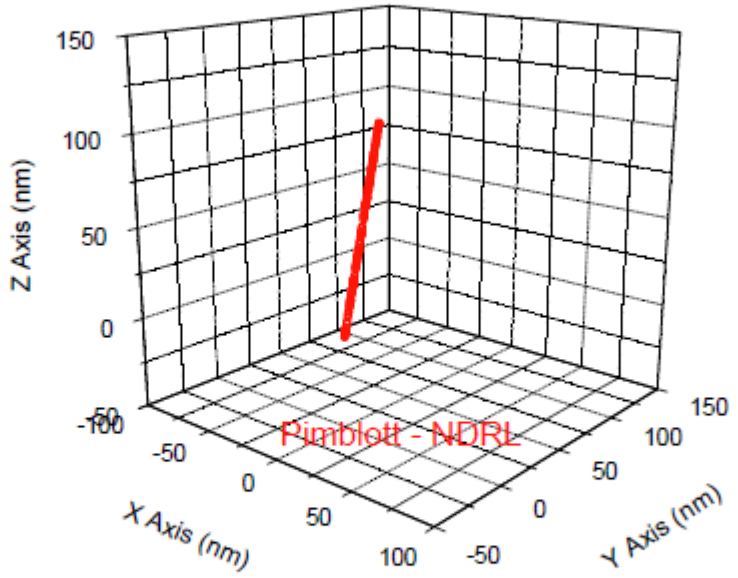


5 MeV ^{4}He

INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure

The following figures show the evolution of the initial 10 keV section of a 5 MeV helium ion track in water.

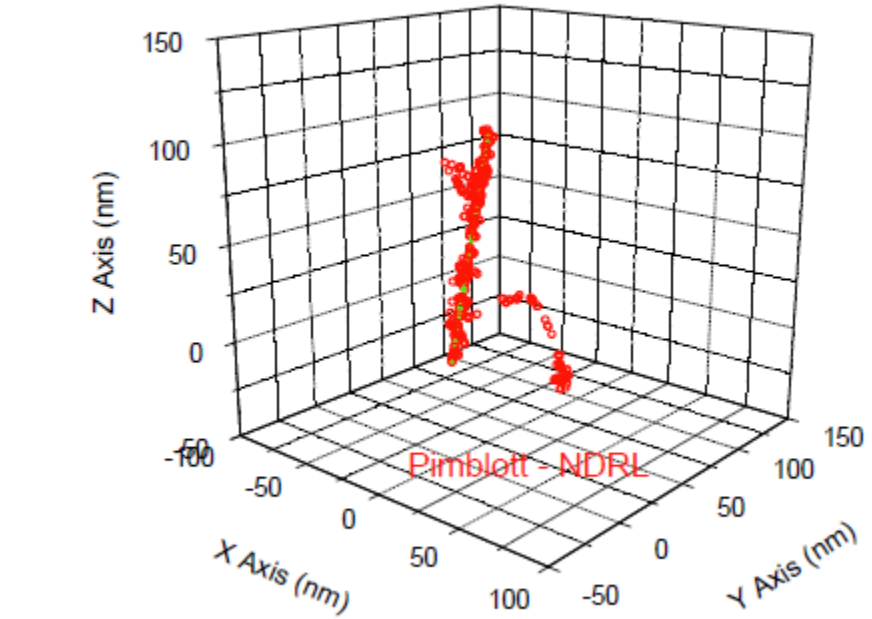


Primary track

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.

INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure

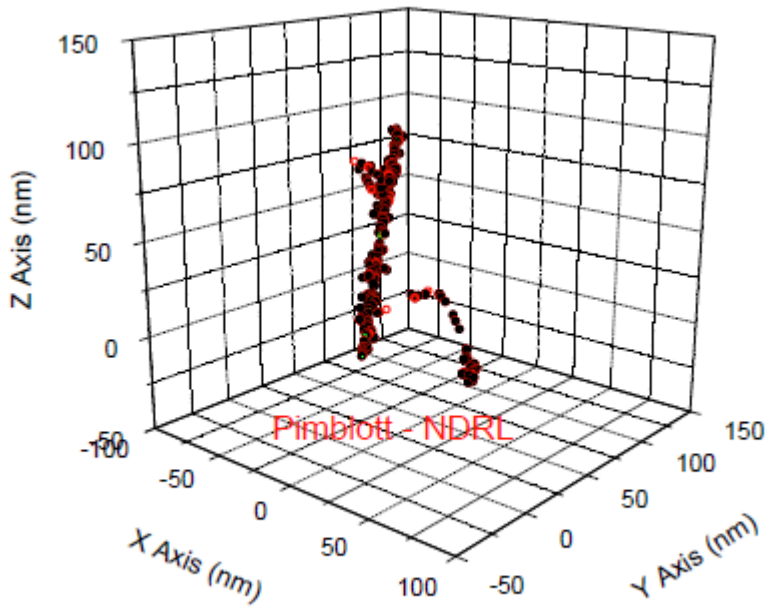


Secondary tracks

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.

INTERACTION OF IONIZING RADIATION WITH MATTER

Track Structure

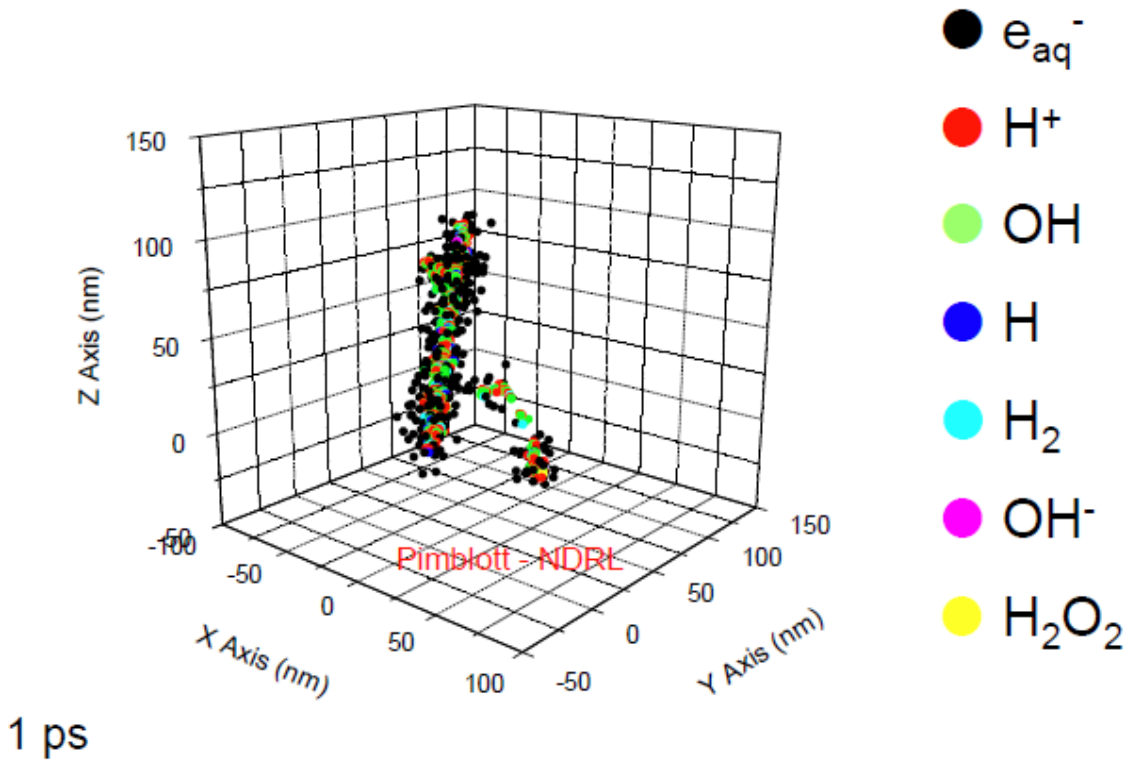


Low energy electrons

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.

INTERACTION OF IONIZING RADIATION WITH MATTER

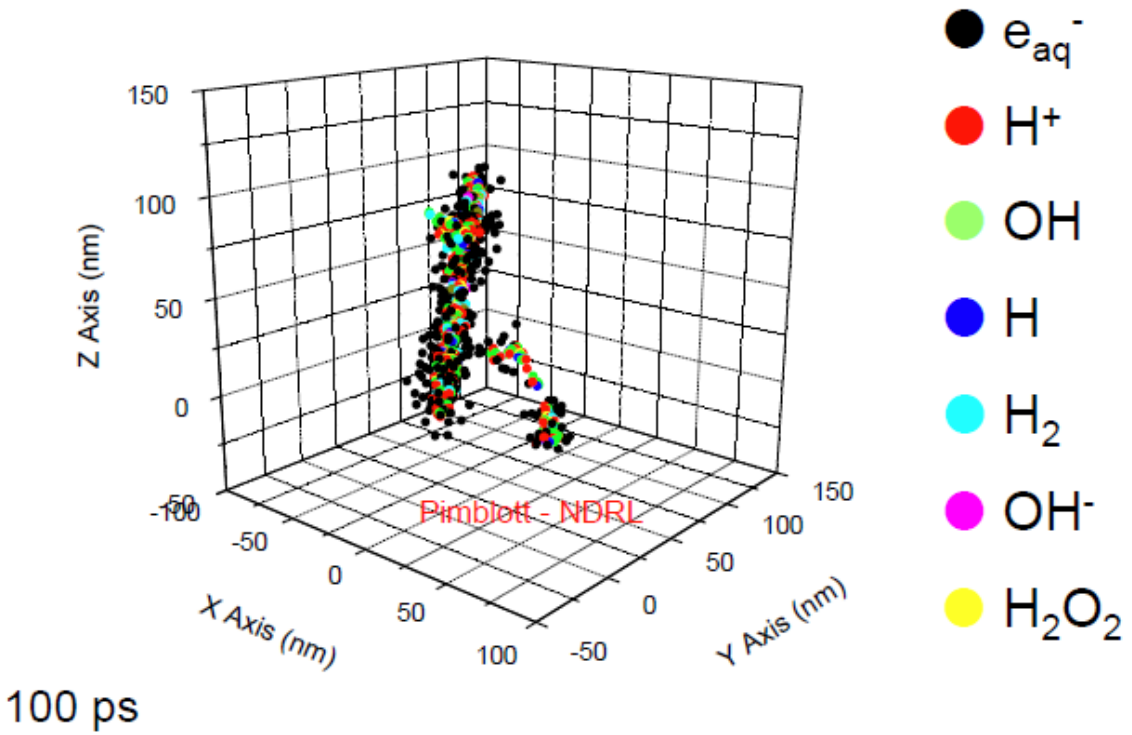
Track Structure



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.

INTERACTION OF IONIZING RADIATION WITH MATTER

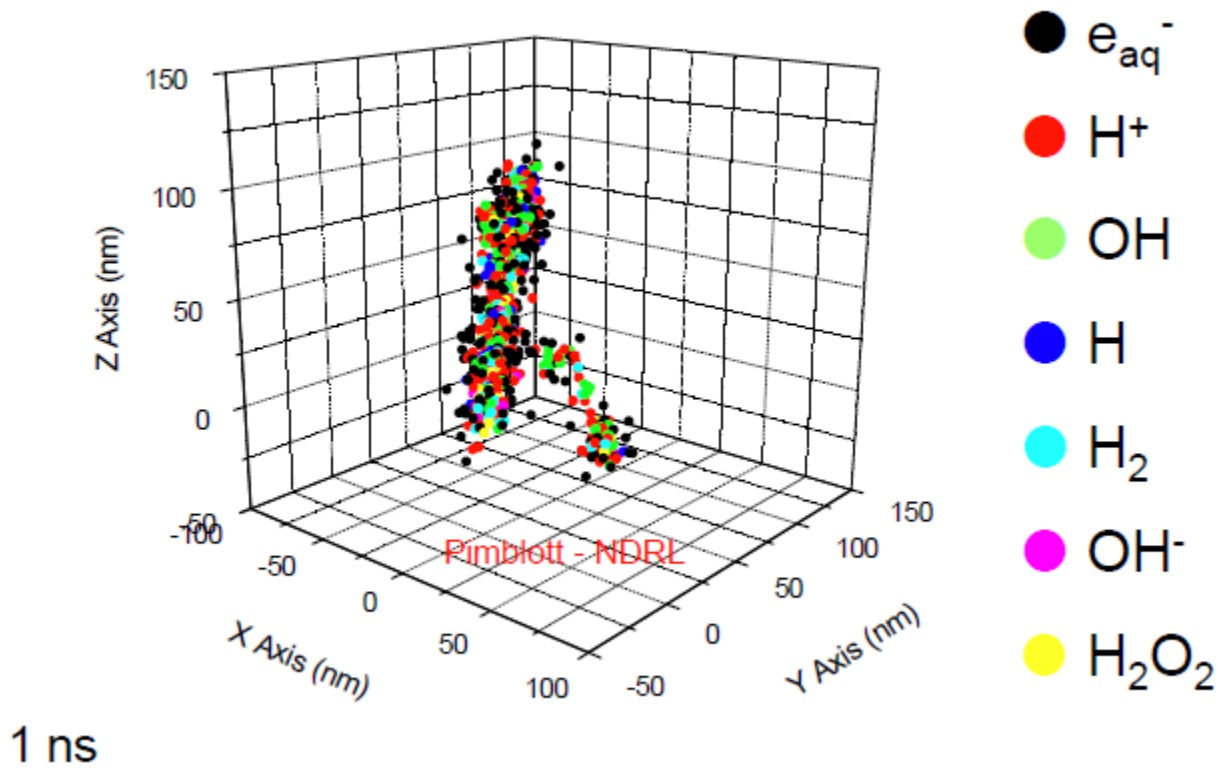
Track Structure



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

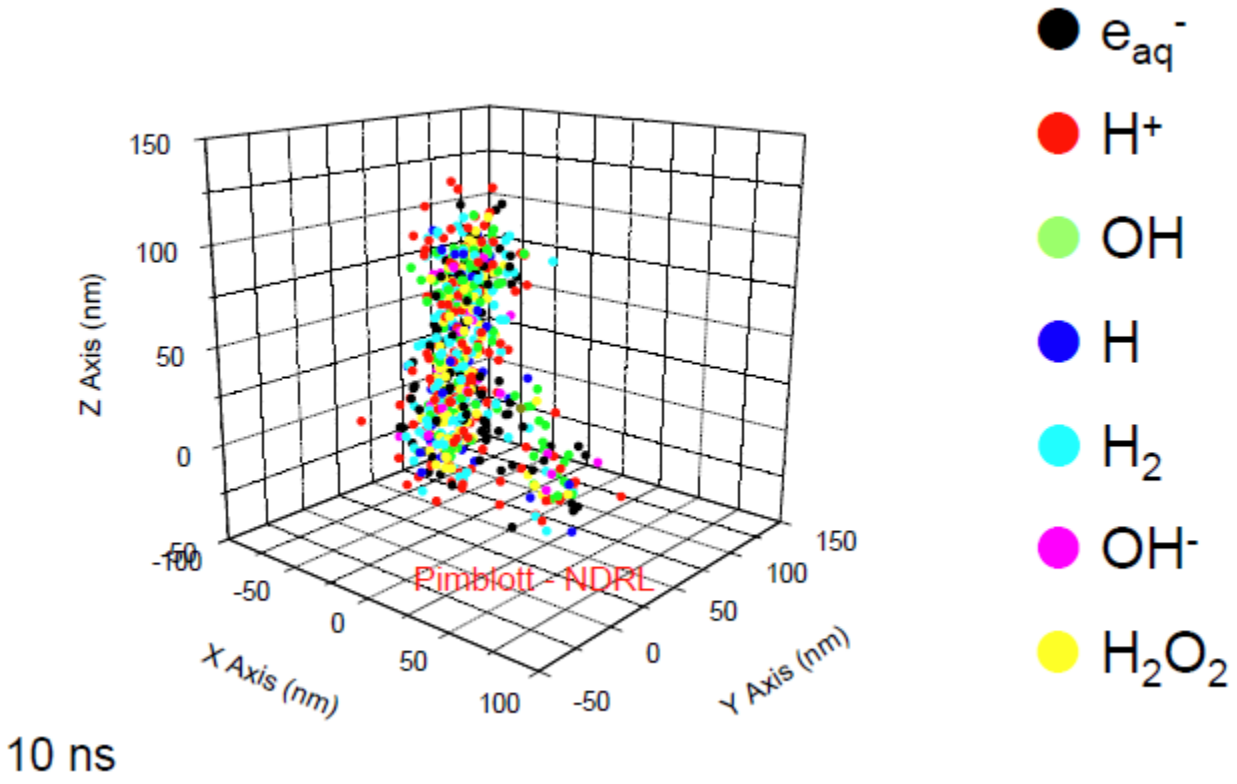
Track Structure



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.

INTERACTION OF IONIZING RADIATION WITH MATTER

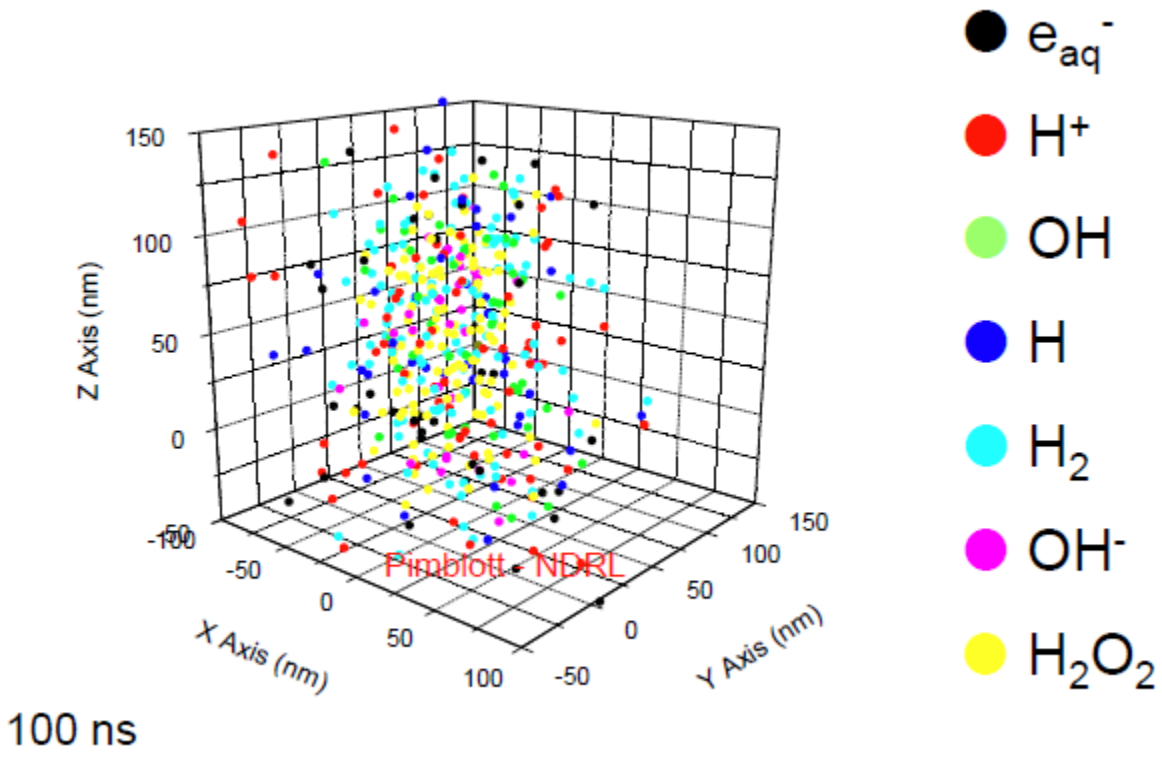
Track Structure



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.

INTERACTION OF IONIZING RADIATION WITH MATTER

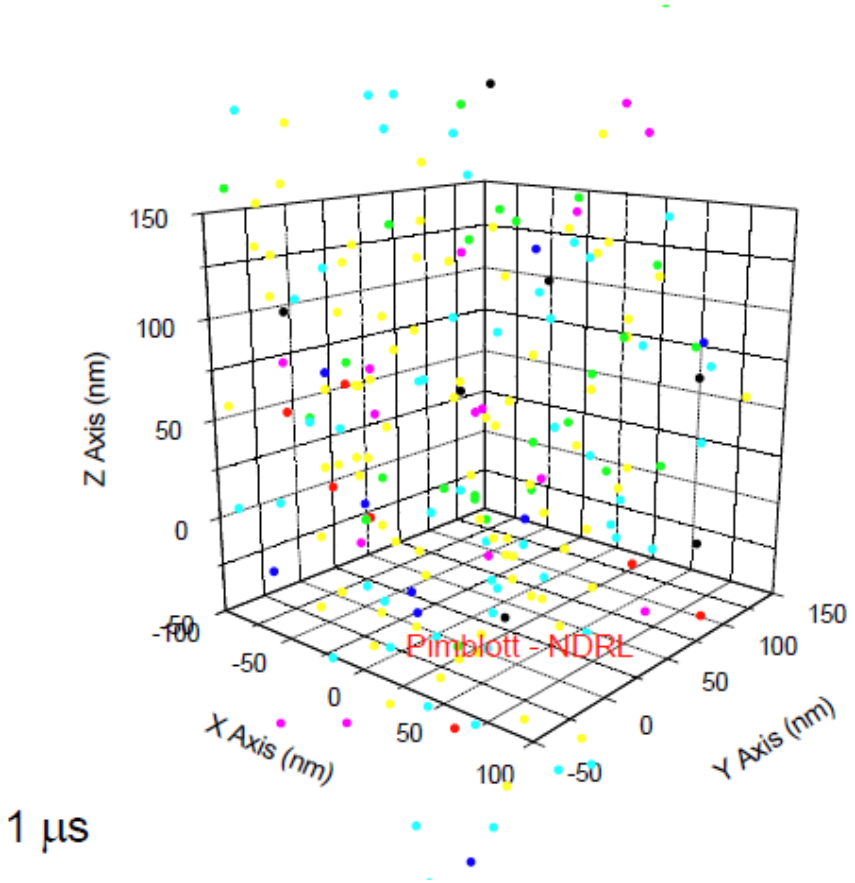
Track Structure



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.

INTERACTION OF IONIZING RADIATION WITH MATTER

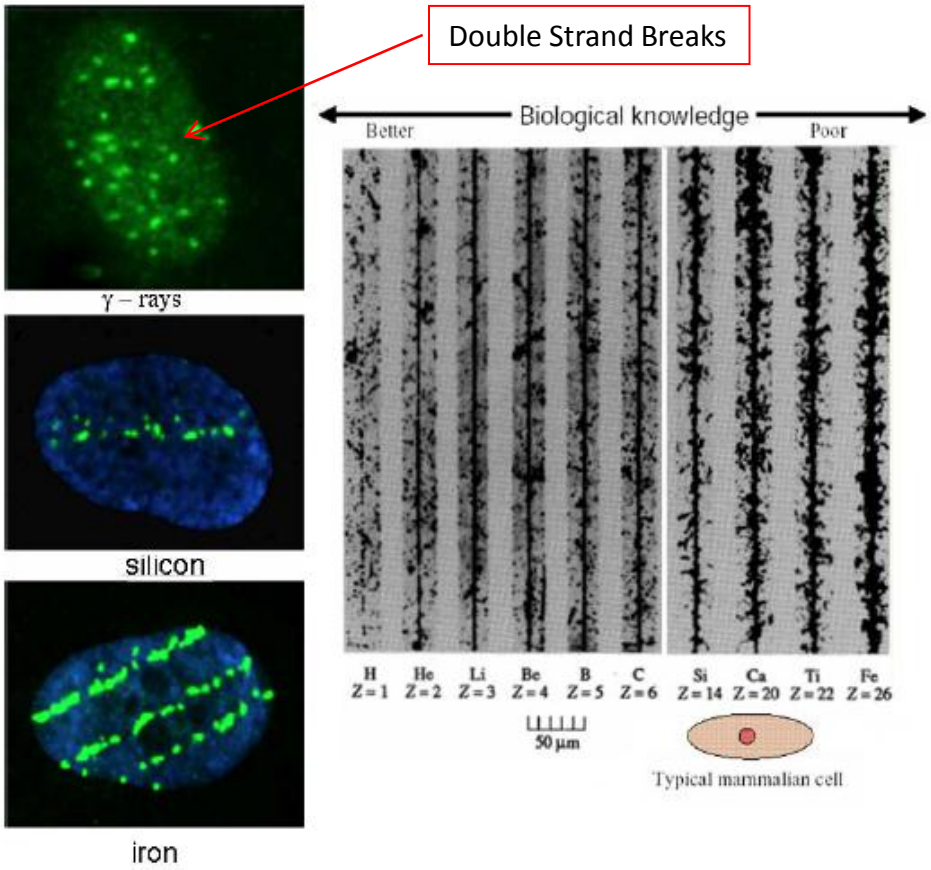
Track Structure

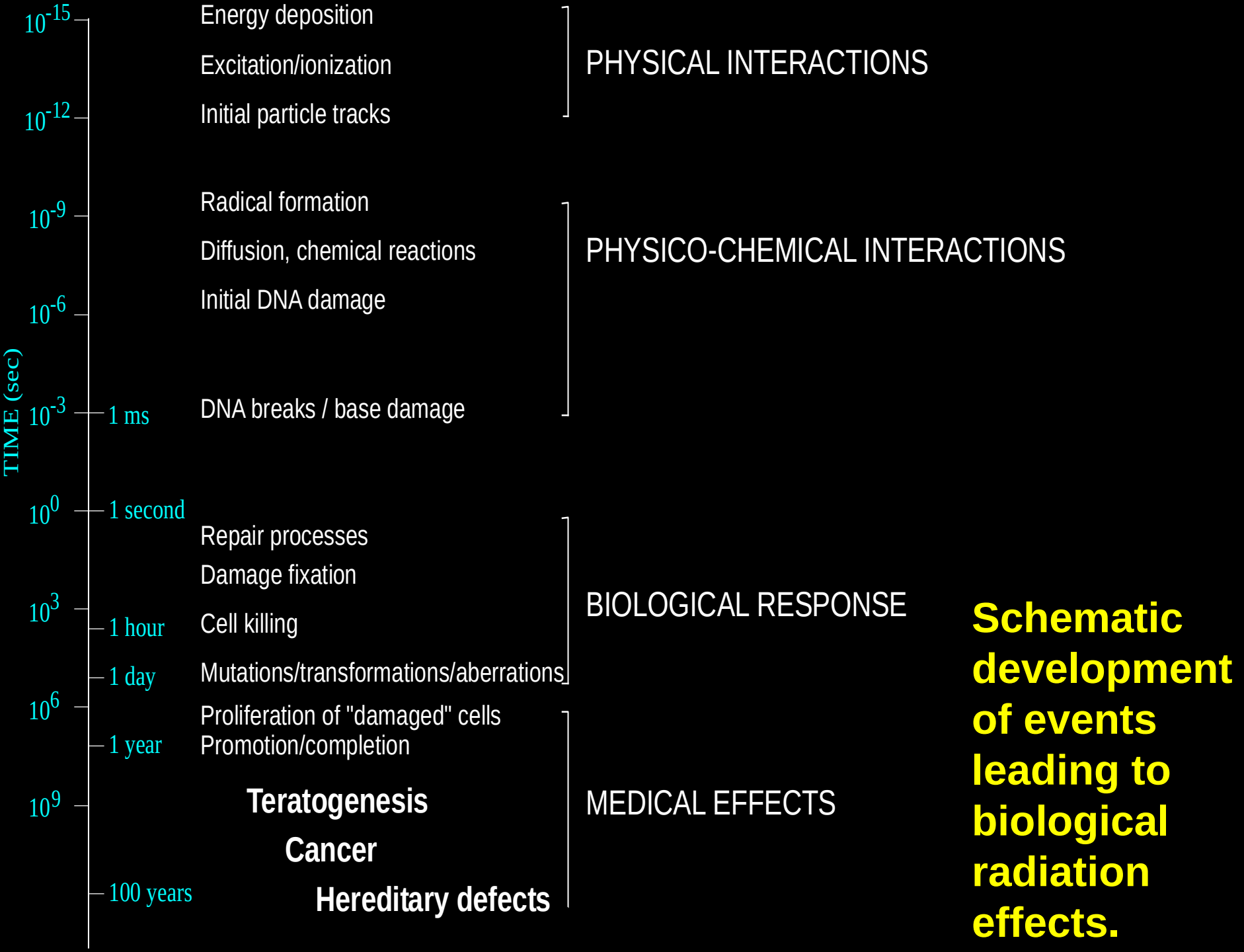


The track structure is finally lost at very long times. The species produced in this track will react with added solutes in the bulk medium or with the walls of the container. **At very high dose rates the species of this track will react with those of a neighbouring track**.

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=ΔE/Δx) along the track by increasing the charge Z. The left panel shows three nuclei of human fibroblasts exposed to γ-rays, Si-, or Fe-ions, and immunostained for detection of γ-H2AX[14]. Each green focus corresponds to a DNA DSB. While in the cell exposed to sparsely ionizing γ-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).





Schematic development of events leading to biological radiation effects.

1990 RECOMMENDATIONS OF THE ICRP

W_R values

Table S-1. Radiation weighting factors[1]

Type and energy range[2]		Radiation weighting factor, w_R
Photons, all energies		1
Electrons and muons, all energies[3]		1
Neutrons, energy	< 10 keV	5
	10 keV to 100 keV	10
	> 100 keV to 2 MeV	20
	> 2 MeV to 20 MeV	10
	> 20 MeV	5
(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).

1990 RECOMMENDATIONS OF THE ICRP

W_T values

Table S-2. Tissue weighting factors[1]

Tissue or organ	Tissue weighting factor, w_T
Gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05[2,3]

[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.

…too complicated?

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

Activity (Bq)

Absorbed Dose(Gy)

W_R

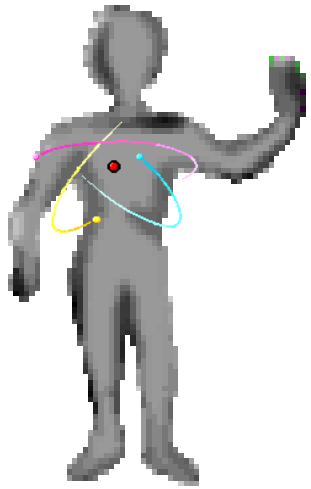
Equivalent Dose (Sv)

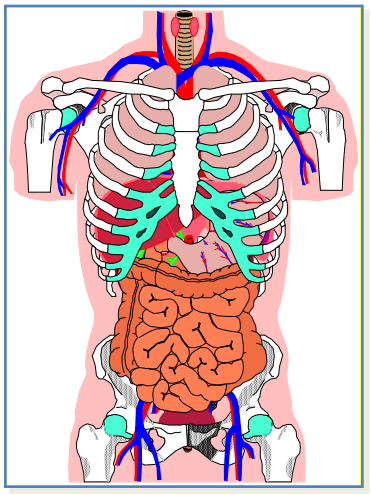
W_T

Effective Dose (Sv)

Fluence (cm^{-2})

Conversion Factors ($Sv\ cm^2$)





Effective dose by ingestion & inhalation