venței aberațiilor cromozomiale nestabile (dicentrici și inele centrice). Prin compararea frecvenței dicentricilor și a inelelor centrice cu curba-standard „doză – efect", primită în condiții „in vitro", se poate determina doza de expunere. Această metodă este recomandată de OMS și AIEA pentru aplicarea practică. Însă utilizarea dicentricilor și altor aberații cromozomiale nestabile pentru biodozimetrie nu este posibilă în toate cazurile, deoarece numărul de celule care conțin așa aberații după expunere se micșorează în timp (UNSCEAR, 2000).

Dozimetria RPE a fost utilizată cu succes după accidentul nuclear de la Cernobâl. Esența metodei constă în determinarea cantitativă a afectărilor în smalțul dinților – unicul țesut cu o cantitate mai mare de minerale și în care nu au loc procese metabolice. Smalțul dinților constituie un dozimetru natural individual destul de precis, care există la om din momentul formării dinților. În baza analizei nivelului semnalului RPE se determină cantitatea de radicali liberi în smalțul necariat. Dozimetria RPE are un prag esențial de sensibilitate (circa 50 mGy) și cea mai mare exactitate pentru metodele retrospective (30-50%). Aceasta este unica metodă obiectivitatea căreia poate fi ușor controlată, iar erorile pot fi calculate exact. Exactitatea înaltă a metodei de dozimetrie prin RME a fost confirmată prin diferite intercalibrări internaționale. Au fost obținute dependențele liniare ale valorilor semnalului RPE în funcție de doza absorbită în diapazonul 0,1- 20 Gy, cu precizie de 20% .

Cu toate acestea, există limitări în utilizarea dozimetriei RPE: insuficiența eșantioanelor (pentru dozimetrie pot fi utilizați dinții înlăturați numai la indicațiile stomatologului, fapt care se întâmplă rar. În afară de aceasta, foarte des dinții înlăturați sunt cariați și conțin o cantitate mică de smalț, iar rădăcinile dinților nu-l conțin deloc); prezența factorilor care, în lipsa unei evidențe adecvate, pot influența rezultatele dozimetriei RPE – expunerea medicală pe parcursul vieții, expunerea dinților anteriori la raze UF. În general, numai 25% din dinții înlăturați ai participanților la diminuarea consecințelor accidentului nuclear de la Cernobâl pot fi utilizați în dozimetrie.

În baza investigațiilor RPE a probelor biologice (țesutul osos, dinți, păr, unghii și țesut epitelial), după iradiere pot fi determinate dozele în diapazonul 0,3 – câțiva Gy. Intensitatea semnalelor RPE este mai mare pentru fotonii cu energie mai mică și mai mică pentru neutroni. Cu ajutorul acestei metode pot fi stabilite atât dozele letale, cât și cele subletale.

# ON THE LINEAR-NON-THRESHOLD MODEL OF THE INTERNATIONAL COMMISSION ON RADIOLOGICAL PROTECTION. SOME THOUGHTS

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***Резюме***

*Работа включает несколько соображений, касающихся модели LNT, установленной Международной Комисией по радиационной защите (ICRP), попытка их логического анализа (в пределах возможного), найденные в литературе по специальности аргументы за и против этой модели.*

*Представляем защитное свойство клеток организма человека и официальное оправдание модели ICRP. В работе объясняется почему элементы с очень большим числом облученных животных не могут подтвердить или отклонить LNT.*

## Introduction

The action of ionizing radiation, IR, on living cells and tissues is called "exposure". It is measured by „doses", expressed in Sieverts (Sv).

Every living cell has a sort of "brain", the DNA (DeoxyriboNucleic Acid) which controls the metabolism of the cell. The cell contains also several "organelles": mitochondria, Golgi apparatus, lysosomes, ribosomes, etc. They are the equivalent of human organs. If the DNA or an organelle is destroyed by IR, the cell dies. A special case is when DNA is only partially damaged and an abnormal metabolism is produced; then there is a danger for cancer in some years!

The exposure produces two types of macroscopic effects:

- deterministic effects
- stochastic effects

IR also produces „free radicals", a sort of poisson; there are natural and artificial subtances which eliminate them.

Life appeared on Earth in a natural radioactive background due to cosmic radiation and natural radionuclides such as $^{238}U$, $^{232}Th$, $^{226}Ra$, $^{222}Rn$, $^{40}K$.

During a great time-interval human body has developed important means of defense by cell processes and by the immunity system.

There are theories which estimate that the evolution of species could also be explained by the genetic mutations induced by natural exposure.

Human organisms are adapted to a natural mean exposure of about 3 mSv, see *Table 1*.

**Table 1**

*Annual Effective Doses in Romania*

| No. | Sources of Exposure | Effective Dose (mSv) | % |
|---|---|---|---|
| 1. | Cosmic radiation | 0.4 | 14.3% |
| 2. | Terrestrial gamma radiation | 0.3 | 10.7% |
| 3. | Radionuclides in the body ($^{222}$ Rn excepted) | 0.3 | 10.7% |
| 4. | $^{222}$ Rn and descendents | 1.4 | 50.0% |
| 5. | Artificial background (medical, applications) | 0.4 | 14.3% |

Total: 2.8

From *Table 1*, three observations result:

- $^{40}$K activity of 5000 Bq present in human body produces only 0.2 mSv;
- secondary cosmic radiation produces only 0.4 mSv; this value increases significantly with altitude (mountains, aircrews);
- the contribution of $^{222}$Rn may be estimated in an equivalent dose for lung of about 30 mSv (using the tissue weighting factor); this explains the important $^{222}$Rn and its descendents contribution to lung cancers (≈50%).

## Deterministic effects

If a big number of cells are destroyed in a tissue, the deterministic effect appear, characterized by local equivalent "threshold-doses". Above these thresholds, damages increase linear with dose. This is a "cause-effect" process. These effects appear shortly after exposure.

The usual deterministic effects, together with their "thresholds doses" in Sv are: cataract (7), erytheme (10), sterility (5), marrow aplazia (1), induced liver fibrosis (7), acute diarrhea (8), gonads (5) etc.

The corresponding effective doses are under 5 Sv, so that they do not induce death (5 Sv is the lethal global dose). It can be noticed that the delicate gonads are very resistant. It is interesting to note that human organisms trained to 3 mSv are very resistant to several Sieverts. Most of effects may be medically cured. One must mention that the cell is trained to eliminate free radicals, because they appear in a normal way by normal metabolism.

## Stochastic effects

The effect is represented by the malignant disease and heritable effects which appear with a small probability in a population which suffered exposure. Only a few people present the effect. The effect appears after years. Some classic diseases could also appear (cardiovascular, lung or gastric diseases, etc.), but they are much less important.

A special effect of exposure is the damage of DNA, this real brain of the cell. This may produce genetic modifications and the result is a modified cell. So the stochastic effects appear. From a group of exposed peoples, the effects appear only at some of them. We may presume that the stochastic character is due to a variety of immunity systems.

If the damage is not important, DNA is repaired by some special "repairing enzymes", found in the cell. In the same time the DNA commands the production of more enzymes and the ceasing of cell division. If the repairing is not complete, DNA commits " suicide", stopping the metabolism and the cell dies. A cell may die even if DNA is not damaged in case other parts of the cell are strongly damaged. Abnormal cells may be destroyed by the famous "T-killer" white cells.

If none of the above processes succeed, a cancer may appear. At least theoretically, as no threshold dose was observed, cancer may be produced at very low doses. The stochastic effects generally appear late after exposure (years).

Some comments must be done.

How the enzymes are aware of the DNA damage, how they know to repair it and how DNA decides a suicide, this is the „inteligence" off the cell ( ask cell biologists!)

It is interesting to note also that DNA behaves as „a person", wich asks for more medicines and refrains himself from procreating, in order not to produce abnormal children!

The fact that the cell division may be not stopped and the suicide does not succed (cancer risk) is due perharps to the „illnes" of the DNA and the cell does not obey its commands. The recognition of an abnormal cell by the T-killer lymphocites is explained by the modified dejections of the cell, wich are transmitted through the membrane.

*Table 2* presents same risk of cancer for a group of exposed persons.

*Table 2*, is virtual: No 1000 peoples were irradiated thougher!

But the data in the table 2 are based on many human cases (Hiroshima end others) and a huge number of irradiated animals.

One sees that the delicate gonads are resistant, but they produce malformations ( hereditary effects). The probability of stochastic effects is 5.7 % per Sievert. It may be extrapolated to 0.006 % per mSv.

*Table 2* is sinister from one point of view, but encouraging from another: 950 persons from 1000, irradiated with 20% of the lethal dose, do not develop cancer, not even the irradiation disease (acute irradiation syndrom).

**Table 2**

*Stochastic Effects. Number of cancers for 1000 peoples irradiated with 1 Sv*

| No. | Organ | Lethal Cancer | Non Lethal Cancer | Hereditary Effects |
|---|---|---|---|---|
| | Breast | 3.5 | 7.0 | - |
| | Marrow | 3.0 | 1.0 | - |
| | Lung | 9.0 | 1.0 | - |
| | Thyroid | - | 3.0 | - |
| | Gonads | 1.5 | 0.5 | 0.2 |
| | Liver | 3.0 | - | - |
| | Colon | 3.0 | 2.5 | - |
| | Stomach | 7.0 | 1.0 | - |
| | Ovary | 6.0 | 4.0 | - |
| | Urinary bladder | 1.5 | 1.5 | - |

Once again, the organism has a great power of defense. The generally adopted radiation protection principle ensures a radiation risk equal to risks produced by other human activities.

## The LNT model of ICRP

The LNT model of ICRP presented the following consideration [6]:

Although there are recognised exceptions (wich ones?), for the purposes of radiological protection the Commission judges that the weight of evidence on fundamental cellular processes coupled with dose-response data supports the view that, in the low dose range, below about 100 mSv, it is scientifically plausible to assume that the incidence of cancer or heritable effects will rise in direct proportion to an increase in the equivalent dose in the relevant organs and tissues.

Therefore, the practical system of radiological protection recommended by the Commission will continue to be based upon the assumption that at doses below about 100 mSv a given increment in dose will produce a directly proportionate increment in the probability of incurring cancer or heritable effects attributable to radiation.

This dose-response model is generally known as 'linear-non-threshold' or LNT.

This view accords with that given by UNSCEAR (2000). Other estimations have been provided by various national organisations, some in line with the UNSCEAR view (e.g., NCRP, 2001, NAS/NRC, 2006) while a report from the French Academies (2005) [5] argues in support of a practical threshold for radiation cancer risk (50-100 mSv). However, from an analysis conducted by the Commission (Publication 99, ICRP, 2005d), the Commission considers that the adoption of the LNT model combined with a judged value of a dose and dose rate effectiveness factor (DDREF) provides a prudent basis for the practical purposes of radiological protection, i.e., the management of risks from low-dose radiation exposure.

The DDREF was introduced by UNSCEAR for extrapolating the cancer risk at high doses and dose rates (5%/Sv) to the risk at low doses and low dose rates.

The factor divides by 2 the risk at low doses; its value is still subject of discussion for different situations. [3] The logic of this factor is that at low doses and dose rates the repair mechanisms have a reduced number of damages to repair and time enough to do their jobs.

From LNT and DDREF the extrapolated risk of cancer at 1 mSv is $3 \times 10^{-5}$. It looks negligible but it represents 30 cancers at a million people. It is not easy to accept the LNT model.

What to think about the idea that 6 bilions of people on earth, suffering natural exposure every year, live all their life with a cancer riskfrom IR. And what to think about the necessary medical investigations wich add some more exposure.

It is about 1 mSv, but iCRP says " it is dangerous"!

ICRP dedicated its publication 105 to this last subject.

There are ten (10) recognized factors inducing cancer: excessive smoking, alcohol in excess, pollutants, IR radon, UV radiation, obesity, genetic transmission, cell division, HPV( Human Papiloma Virus).

Due to this mixed pattern, one can not determine the risk of cancer for low doses of 1-3 mSv superimposed on the statistical risk produced by all the other factors in the same time interval under study.

To demonstrate the LNT model, two huge lots of animals must be compared, one irradiated with 1-3 mSv and the other non-irradiated. It will last years and it would be practically impossible to ensure both lots an "identical" life and an "identical" number of cancers produced by other factors than the initial exposure.

For medium and high doses the variation of risk with dose is linear, then grows quadratic and then drops, due to the fact that at high doses the DNA is rather killed instead of damaged [2].

However, there are two strong arguments to sustain LNT:

- the great number of ionizations produced by 1 mSv in a cell;
- the opinion of cell – biologists who declare "the cluster – damages in DNA can not be repaired with fidelity [4]. The track of a single ionizing particle in a DNA produces clusters".

More then that every repairing mechanism has a statistical character.

The established dose limits are correlated with the LNT model. It is 1 mSv for population (why not 0.1 mSv?). The professional dose limit of 20 mSv is established by comparing the risk for cancer produce by IR with all other risks accepted by the society for different professional activities.

The effort to clarify the problem on diferents risks is enormous, as proved by the contents of ICRP 103.

It is not easy to put life in equations!

Duty Hiroshima the populations has vary great fear for IR, but there is a lot of dangers wich shorter the life of people, much, much more, see [1].

**Conclusions**

To decide between LNT and the threshold is very difficult, in spite of the huge experimental data accumulated at medium and high doses.

Arguments for LNT are:

- the great number of ionizations in cell and DNA
- the cluster damage problem
- the statistical character of repairing mechanism.

Arguments against LNT are:

- the action of the repairing enzymes and the suicide of the cell
- the action of T-killer lymphocytes, not affected by cluster problems
- high threshold for the deterministic effects

One may appreciate that, at present, the LNT model is an opened question.

**References**

1. B.L. Cohen, I. Lee, *Health Physics*, 1979, 36, 707.
2. ICRP, *1990 Recommendations of the International Commission on Radiological Protection,* ICRP Publication 60. Ann. ICRP, 1991, 21 (1-3).
3. UNSCEAR, *Sources and Effects of Ionizing Radiation.* In: UNSCEAR Report to the General Assembly with Scientific Annexes. Vol. II, 2000.
4. ICRP, *Low-dose Extrapolation of Radiation-related Cancer Risk,* ICRP Publication 99. Ann. ICRP, 2005, 35 (4).
5. French Academy, *La relation dose-effet et l'estimation des effets cancérogènes des faibles doses de rayonnements ionisants,* French Academies Report, 2005.
6. ICRP, *The 2007 Recommendations of the International Commission on Radiological Protection,* ICRP Publication 103. Ann. ICRP, 2007, 37 (2-4).

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## MONITORINGUL CONCENTRAȚIILOR DE RADON ($^{222}$RN) PE TERITORIUL REPUBLICII MOLDOVA

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Centrul Național de Sănătate Publică

Estimarea riscului expunerii populației la radiații ionizante întâmpină probleme considerabile de interpretare, generate de existența multiplelor surse de radiații de diversă natură, a diferitelor căi de expunere și a numărului limitat de date disponibile. Rezultate veridice pot fi obținute doar în cazul în care monitorizarea este realizată în mod sistematic timp de câțiva ani, în baza utilizării echipamentelor performante.

Sursele principale de acumulare a $^{222}$Rn în locuințe sunt: exalarea acestuia din sol, emanarea din materialele de construcție ale locuinței, apa potabilă menajeră, precum și gazul din bucătării sau sobe de încălzit.

Emanarea/exalarea $^{222}$Rn depinde de structura și dimensiunea rocilor minerale, de migrarea în sol reglementată de parametrii geofizici și geochimici ai solului, de condițiile hidrometeorologice.

În cercetările noastre anterioare din anii 1991–2006, monitorizarea concentrațiilor de radon în probele de aer prelevate din diverse încăperi de pe teritoriul Republicii Moldova a demonstrat că în majoritatea cazurilor concentrațiile de $^{222}$Rn nu au depășit nivelul maxim admisibil și au constituit 92,0...179,1 Bq/m$^3$. Totodată, cuantificarea concentrațiilor de $^{222}$Rn în probele de aer prelevate din galeriile subterane de păstrare a vinului de la Cricova, din galeriile subterane din mun. Chișinău și s. Mileștii Mici, unele mine din Orhei a înregistrat valori ale concentrațiilor de radon de 200...1800 Bq/m$^3$, care depășesc nivelul maxim admisibil, ceea ce impune necesitatea unei monitorizări în dinamică, cu elaborarea hărților concentrațiilor de $^{222}$Rn. Este de remarcat faptul că minele neuranifere trebuie să formeze obiectul unei preocupări permanente de protecție a muncii. Este necesar a elabora urgent norme specifice de radioprotecție pentru aceste spații și a organiza supravegherea expunerii personalului la radon.

Studierea radioactivității în 331 de probe de sol adiacent diferitelor tipuri de roci, la diferite adâncimi – 0,5-0,8 m, a demonstrat variația concentrațiilor $^{222}$Rn în funcție de tipul solului. Rezultatele denotă valori înalte ale concentrațiilor de radon, care depășesc CMA, conform normativului național – 200 Bq/m$^3$ pentru solurile nisipoase și argiloase, argila