THE RADIATION EXPOSURE REGULATION FOR XXI CENTURY

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PRESENT STATUS

ICRP and IAEA innovations [1-3] initiated harsh retorts from the French Academy of Science and the French Medical Academy [4, 5], the American Health Physics Society [6], Europe parliamentarians [7], many specialists [8-25 et al.], including the ICRP Chief himself [26-27]. One calls upon to decree a moratorium on new recommendations [5].

The use of the linear no-threshold (LNT) dose function for the restriction of a detriment from stochastic effects of irradiation is in the focus of discussion. As far back as in the ICRP Publication 1 [28] a demand to the dose accumulated up to the life end was the base of the exposure regulation. It must not exceed a threshold of deleterious deterministic (in today's terminology) effects (D) of exposure, and a probability of the deleterious stochastic effects (S) of that dose would be at an acceptable low level.

For the simplicity it was supposed that this probability is submitted to LNT dependence. To provide D control the limits of the yearly dose in a critical organ were used, different for four organ groups. The limits were obtained as the life-span dose limit divided by a number of years of exposure. In their subsequent work ICRP was guided on D threshold data in conditions of prolonged exposure [29]. As for S, ICRP noted in the Publication 26: "...the more guardedly linearity assumption the more important to realize that it can result in overestimation of radiation risk and in acceptance of more deleterious alternative decision than related with the use of radiation" [30].

By contrast with this warning the LNT dose response is turned practically in a dogma. Any low dose is considered as inauspicious for health. All individual doses more than zero are included in a collective dose [1]. Therefore the consequences of the Chernobyl accident were estimated as 1.6 mln victims all over the world [31]. To search a compromise between real possibilities and unavoidable detriment from irradiation was needed. To optimize an exposure by the collective dose using its economy on the money cost of one man-Sv was offered.

Ignoring the ICRU the ICRP itself inserted the new equidosimetric [9] quality: the effective dose and its derivatives [1, 9, 30] based on LNT conception. As a result a disappointing necessity on parallel personal control of two quantities appeared: a maximum individual dose equivalent in an organ critical on D and an effective dose characterizing S [9].

To avoid duplication [32] the dose limits in the Publication 60 ICRP were reduced by several times. As a result it made it possible to control D only in a skin what does not complicate the existing practice of exposure control. (Eye's lens, hand and foot exposure are regulated too but it is no necessity at all. Extremities' exposure is regulated by its skin exposure; a simultaneous observance of limits for personal dose equivalents Hp(0,07) upon the face skin and Hp(10) upon a forehead would prevent lens overexposure.

Dose limits were reduced without earnest scientific substantiating. It was administrative one [23]. But in Annex C of the ICRP Publication 60 it was taken into account that accumulated dose of prolonged exposure increases gradually with age. Therefore a death risk from cancer increases in years. On the other hand, non-radiation causes of death are in the competition and cancer do not appear during latent period that may last 25 years or more. This because the exposure in senile age has lesser chance to provoke radiation cancer before a natural end of a life.

The yearly dose limits determined in the ICRP Publication 26 appeared to provide an accepted life-span death risk owing to radiation cancer. There were absolutely unfounded to change them [4, 18]. But the Commission ignored own conclusions on unconvincing grounds. Its decision to make the yearly dose limit for population for five times less can not be justified by the fact that the category B of exposed human was excluded and a new limit in the Publication 60 was determined for population as a whole. In both cases the exposure is regulated on the base of the critical group irradiation and this group would remain the same.

These innovations provoked extraordinary consequences. ICRP was prepared its Publication 60 at the period of detriment from the Chernobyl accident overcoming. The former limit 5 mSv·y⁻¹ for category B was looked too overstated. A radiation would seem to be much more dangerous than the government and experts assured. As a result, the Russian Duma has issued in 1991 the legislative acts according that a population exposure in impact areas of the accident should be diminish to 1 mSv·y⁻¹. The number of districts that would be considered as contaminated ones rose from 4 to 17 and the number of participated residents rose from 150-200 thousand to 2.6 million persons [33]. Area rehabilitation became suddenly more expensive and dragged out. Risen radiophobia wave caused much more harm to population than enhanced irradiation. The radiophobia brought substantial detriment to other European countries where the doses were negligible [34]. In contradiction with the position proclaiming by ICRP the negative attitude of society to the use of atomic energy and radiation...
sources grew sharply.

**CRITICISM OF THE LNT CONCEPTION**

ICRP considerably overstates low-dose risk relying at exposure reglamentation on LNT conception [4-25, 35-38]. It is proved by the fact that cell initiation to malignancy is a stochastic process. It depends on genome damage as a result of an accidental hit of one charged particle in a cell target or passing through near it. Such event in one cell is necessary but insufficient to get a person to suffer of radiation cancer. This initiation is a delicate process: the cell must remain alive and capable to unrestricted proliferation.

Arguments of LNT defenders are limited by a fate analysis of the one transformed cell giving rise to a cancer [23]. But it is a multistage process. One threshold stage is enough to get the whole function to be a threshold one; and a sum of linear dose functions is enough to have it non-linear or even quasithreshold [38]. Besides this it is extremely rare event. In our organism there is permanently ~10^{14} spontaneously transformed cells [39]. But only one of them per 4-6 mln will reach the progression study. In order to start this process a fluctuation of transformed cells number on a tissue part may randomly exceed a defined level at which one of many such cells may randomly overcome organism protective forces. That is the second stochastic stage of carcinogenesis, that can cause its non-linear dose dependence. Dose dependencies of hormesis [19] and of latent period duration [40-43] may by associated with this stage.

Indeed if the detriment from radiation cancer is assumed equal to 5 \cdot 10^{-2} Sv [1] so a spontaneous cancer rate would be comparable with radiation one after exposure in life-span dose about 4 Sv [18]. It is obvious that they will interact at low (and at no so low) doses and may disturb LNT and stipulate radiation hormesis [18, 44-46]. At least for some cancer forms the latent period from cell initiation to clinical manifestation of cancer is increasing with a dose decreasing [18] and consequently with a number of transformed cells. Therefore at low doses the illness may not have time to become apparent till natural death especially at exposure in elderly age. This is one of the phenomena that may explain threshold dose dependence.

To substantiate LNT its adherents are also exploiting the empiric data that the same dependence of the death rate over solid cancer sum and cancers of most organs among Japanese AB survivors would have the same form. It is known that it is the main source of knowledge on S in human. But this conclusion's justice depends on the reliability of an estimation the radiation doses from atomic bomb explosion and the doses possess the unique characteristics. It was not only unusual high dose rate but also the combined \(\gamma\)-neutron radiation at that the neutron contribution and RBE increases as a distance from the hypocenter and hence as a dose [47, 48]. But only one value 10 was used for RBE in DS 86 dosimetry system [49]. It may diminish the risk coefficients in low dose interval and increase them for high doses. Neutron contribution was been corrected repeatedly and now they again cast doubt [26, 47, 48]. Therefore there is no confidence that Japanese data are really obeying to LNT and that they can be spread on S of other radiation types. Above all the radiation effects by low dose rate differ in essence [24].

Scientists that came to believe in LNT often average arithmetically their empirical risk data in a wide dose range [24, 50-54]. High risk values in high dose interval may mask completely hormesis or plateau area in low dose interval (less ~ 0.3 Sv in life-span). But regardless of wide spread conviction [26] there are possible the factual information that refuses the applicability of LNT in low dose range. So Cardis and joint authors do not found additional solid cancers in the International program [50, 51]. It envelops more than 2 mln person-years of observation the personnel of atomic enterprises that was working from the onset of nuclear industry. It is evident from other accumulated data that the dose dependence of S has the threshold or hormesis area from ~ 0.3 to tens of sieverts (see table). If guided by the ICRP estimates that these cancers in total can be shown to be responsible for more than a half of whole death risk from radiation malignant illnesses [24]. Such results are accumulating slowly because of statist ic difficulties but there are some reasons to suppose that this conformity is universal.

Adepts of LNT often justify themselves as that this position would simplify the regulation of exposure [27] allowing to sum up the radiation action on separate organs and from several sources. Nevertheless it does not compensate its shortcomings as stated above. It is incompetent to rest upon LNT. But giving it up destroys the whole contemporary system of the exposure reglamentation [20-23]. Do not fear an exposure in low doses. So there is no need to optimize it especially because a detriment - benefit balance depends not only and more often not so much from radiation. One should not use the collective dose and ALARA principle [27]. One should not use the effective dose in its present shape. During a half of a century no human hereditary effects were revealed [56-59]. So it is no reason to make them into account.

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Table. Threshold doses for cancer initiation at low/high dose rate [22]

<table>
<thead>
<tr>
<th>Organ, tissue</th>
<th>Dose, Sv</th>
<th>References</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red bone marrow</td>
<td>0.3 / 0.3</td>
<td>[20,24,50,51] / [49]</td>
<td>Personnel of nuclear enterprises / AB survivors</td>
</tr>
<tr>
<td>Lung respiratory section</td>
<td>16 / 0</td>
<td>[24,65,66] / [49]</td>
<td>²³⁹Pu incorporation, middle dose rate / AB survivors</td>
</tr>
<tr>
<td>Thyroid</td>
<td>&gt;0.3 / 0</td>
<td>[54,67,68] / [1,69,70]</td>
<td>Diagnostic with ¹³¹I / Treatment with ¹³¹I, AB survivors</td>
</tr>
<tr>
<td>Liver</td>
<td>30 / 0</td>
<td>[35,70] / [1,49,69]</td>
<td>Diagnostic with torotrast / treatment with torotrast, AB survivors</td>
</tr>
<tr>
<td>Endost</td>
<td>&gt;10 / 0</td>
<td>(35,70,71) / (1,49,69)</td>
<td>²²⁶Ra, dial painters / Treatment with ²²⁶Ra, AB survivors</td>
</tr>
<tr>
<td>Epidermis</td>
<td>11-16 / 0.8</td>
<td>[72-74] / [1,69]</td>
<td>Experiments with rodents / AB survivors</td>
</tr>
</tbody>
</table>

OTHER DEFECTS OF THE ICRP RECOMMENDATIONS

Accepted risk level of S for population chosen by the ICRP is unwarrantable low in comparison with other risks of contemporary life [17]. It can see on the example of radon exposure. According to [60] protective measures for population recommended by the ICRP would give in Canada the same effect as a decrease of a smoker number by 0.05% the former being far lower, cheap, easy, and not so onerous. Ionizing radiation is very weak carcinogen no strong [26].

The death probability from all causes in developed countries is equal to $1.4 \times 10^{-2} \text{y}^{-1}$ with age variation from $1 \times 10^{-4}$ to $2 \times 10^{-1}$. But a dose limit for population is determined on the annual risk level $1 \times 10^{-5}$ [26]. This adds less than 1/1000 to natural death risk or about 4/1000 of the risk to dye from spontaneous cancer. The suggestion to consider a risk as insignificant only if it is lower $1 \times 10^{-6} \text{y}^{-1}$ [1] looks exceptionally arbitrarily. Such risk level has been attributed to a danger of catastrophic accident with many victims.

Assessing the ICRP position it is impossible to forget that an yearly dose limit is under question to which an exposure of a negligible part of contingent can approach in some years. It is taken into account in the Publication 26 but ignored in the Publication 60. Evidently now the ICRP does not understand the stochastic nature of individual dose distribution. It is extremely unlikely that the same persons can come upon this part of contingent the most years of their life. That's why the life-span dose of referred persons will be substantially lower than the corresponding limit and its mean value for all the contingent will be lower in an order of value. By the way, the life-span dose limitation is an important referent point in conditions of increased prolonged exposure [3, 27].

The equidosimetric quantity system of the ICRP is littered. The equivalent dose in an organ duplicates the mean dose equivalent in it. The radiation weighting factor duplicates crudely the radiation quality factor. Offered by ICRU ambient and directional dose equivalents represents a lame attempt to unite the incompatible tasks [9]: to define a radiation field and an irradiation of a man in the field. The calibration upon the quantities is very difficult [75].

Extensive criticism of the ICRP Publication 60 was given in the article [18]. Lately Commission is supposedly realizing an excessive complication of its recommendations and is ready to refuse of linking them with social factors [27]. It recognizes that the threshold ascertainment of affected dose would be important in justice praxis at court examination of illegal exposures [27] and (let's add it from ourselves) of environment radioactive contamination.

PROPOSALS

I proposes to base the future system of exposure regulation on two basic principles. The first one: radiation doses which are less the threshold one are at least no dangerous. The second one: one should avoid to exceed the individual life-span dose limit. If it is observed for the most exposed group member so the whole group is protected [27]. It is quite reasonable to consider as insignificant the death risk from irradiation for the critical group of population less than $1 \times 10^{-3}$ y⁻¹ for different age or $1 \times 10^{-4}$ y⁻¹ in average. The de minimis dose equivalent is reasonable to choose by the least dose threshold (for leukemia, see table) at the level 300/70=4 mSv·y⁻¹ over the background exposure.

It would be most reasonable in this situation to come back to the simultaneous reglamentation of S and D in separate organ groups [23]. But for them all the LNT conception must be replaced by linear-threshold one.
Along with the dose equivalent \( H_T \) it is needed to introduce the delta-dose \( \Delta_T = H_T - H_{T1} \) where \( H_{T1} \) is threshold dose in T’s organ and \( H_T > H_{T1} \). For personnel the limit of the life-span delta-dose in a critical organ equals \( \Delta_T = 2.5 k_i \) Sv and for population \( 0.35 k_i \) Sv. Here \( k_i \) is a coefficient of i-th organ group equal to 1 by a body uniform exposure and for instance to 6, 20 and 50 for each organs of the 2nd, 3rd, and 4th groups accordingly. Deterministic effects are excluded by stochastic effect restrictions in all organs except ovaries and eye lenses to which \( H_{T1} = 10 \) Sv and \( \Delta_T = 0 \). This is all the more justified for population. There will be no stochastic consequences until accumulated dose equivalent in any organ would be less 0.3 Sv (see table). If \( 0.3 < H_T < 1 \) Sv so only leukemia and possibly thyroid cancer can appear. If at last \( H_T > 1 \) Sv in all significant tissues so radiation cancer would in principle appear in them. Subsequently as the data would accumulate for other cancer types it will be possible to increase the threshold value from 1 up to 4 Sv, for instance.

Thanks to dose thresholds first years of exposure entails no risk of S. But for the current control it is convenient to use the limit of the yearly dose equivalent. It equals \( (H_{T1} + 2.5k_i)/50 \) Sv for personnel and \( (H_{T1} + 0.35k_i)/70 \) Sv for population. Not great exceeding of the annual limit level are criminal since appropriate doses entail with no noticeable health detriment. But it is undesirable to break the life-span dose limits.

If as now to try to take into account the irradiation of all important organs simultaneously so the degree of stochastic effects would express the new additive quantity: the weighted dose \( \Delta W = \Sigma W_i \Delta T \) Sv. Here \( W_i = r_i/\Sigma r_i \) is the issue weighting factor for sensitive part of the T's tissue; \( r_i \) is a nominal risk coefficient of the death over cancer of T's tissue for \( H_{T1} > H_{T1} \). Therefore unlike of accepted today values \( W_i \)'s will depend on the dose distribution in the whole body. The \( \Delta W \) value calculation is available with the use of tables or an easy computer program. Just as effective dose the \( \Delta W \) is not a metrological quality but an algorithm for processing of instrumental data. It may be used for internal exposure regulation. For external irradiation it is easier to lean upon values of \( H_T(10) \) and \( H_T(0.07) \), the individual dose equivalents [3].

A probability that the same resident group would be critical for several radiation sources and that its annual dose would be regularly close to the dose limit is extremely low. Therefore the dose limit may be applied independently for the exposure of each of these sources [27]. There is no need to fix a dose quota for them. Technogen and technogenic altered exposures must be summed. Accident, medical exposures and irradiation by natural sources of water supply must be regulated independently. There is no need to take into account other background exposures. People accommodate to the background level differing by two order of value.

The exposure by incorporated nuclides shall be restricted by life-span committed delta- or weighted dose. Along with the establishment of the dose coefficients for the single intake of radioactive substances it is needed to restore dose coefficients for external exposure related with solitary volume activity of inert gases in the air of premises of different dimensions and outside them [77].

One can seriously underestimate an internal exposure by intake if to lost a steady control for example in an accident as it was with Chernobyl firemen. That's why along with the intake it is necessary to regulate the activity content of Y (and sometimes W) class substances in an organism/critical organ. In contrast to the intake the content of many radionuclides may be determined by instrument means, not only by calculation, and with the accuracy that is more by the order of value than for the intake [77]. For the class D there is no sense to regulate a content.

Though content defines a dose rate but not a committed dose its average annual value determines a real level of annual internal exposure of person. For substances of W class it differs a little from the committed dose. For substances of the class Y it would be probably reasonable to establish a limit of an average annual content depending on the age or on the duration of a work. The measurements with periodicity about the effective value of half-excretion will allow the calculation of the committed dose as well [78].

In the system of the equidosimetric quantities that are used now for a description of a radiation field it is useful to substitute the ambient and directional dose equivalents for the field dose equivalent \( H_T(10) \) [79, 80] (and \( H_T(0.07) \)), a metrologically strict quantity introduced in our country [81]. It is a dose equivalent in the center of a 10 (or 0.07) mm radius sphere of tissue-equivalent material. It is easy to measure it for instance by ionization chambers. For transition to an exposure level of a man in this field one may use an isotropy coefficient [77, 81, 82] which depends not only on field parameters but also on the man position inside it.

CONCLUSION

Next century undoubtedly will accumulate new empirical data upon the radiation cancer in human and improve the quality models of radiation carcinogenesis that may permit to substantiate more reliable the proposed system of radiation regulation.
To a considerably extent the erroneous position of the ICRP is explained by the fact that it is renewed by means of co-optation. It is known that such practice promotes the fixation of conservative tendentious. Subsequently it would be better to elect the ICRP members on the IRPA Congress. New staff will require a courage to established the scientifically more well founded levels of the reglamentation for people irradiation regardless of the false public opinion.

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REFERENCES