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Dosimetry Approach of Coronavirus

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ABSTRACT

The human body is exposed to external ionizing radiation when the source of radiation is outside the body and to internal radiation when the source of radiation is inside the body. The energy absorbed by the ionizing radiation per unit mass (J / kg) represents the absorbed dose (Gy) in the target organ, T. Depending on the radiation quality, the relative biological efficacy, in the same target; the dose becomes the equivalent dose, H_T (Sv), in organ T. This is multiplied by the organ weighting factors (w_T), ie the ratio between the risk coefficients for the organs or tissues, R_T, and the total risk coefficient ($R = \sum R_T$), then the sum ($\sum w_T H_T$), represents the effective dose E (Sv). Adverse health effects due to radiation exposure are deterministic at high doses, with threshold, and stochastic type, at low dose and rate, without threshold, below 100 mSv. Dosimetric and epidemiological studies on these effects have shown a linear relationship between dose (Gy) and the probability of incurring of stochastic effects (cancer and hereditary effects). In the case of radon daughter external exposure and in workplaces other than mines, the linear relationship is between the mortality rate due to inhalation of radioactive aerosols and radon exposure (mJ h m^{-3}). The conversion from exposure (WLM) to effective dose (Sv) was based on equal detriment, not on dosimetry. Regarding the mode of action of SARS Cov-2 virus (100-150) nm, given that they can only grow in living cells, (50-100) µm, it follows that the life cycle of a virus has two distinct stages. In the first stage, the virus appears outside the cells in the form of a neutral virus particle as a passive transport vehicle, which provides opportunities for the infection to spread both from cell to cell within the multicellular organism and between individuals. In the second stage, the virus is inside the infected cell (50 µm, 10^{-9} g), where the replication and directing of processes in the host cell to the synthesis of new infectious virus particles takes place. Other building blocks for the virus particle, such as the energy required and the main mechanisms for assembling a virus are provided by the infected cell. Starting from the radon conversion convention, in this paper, it is proposed, as exposure, the photon field fluence rate Φ, (photons/cm^2.s), as being equal to the rate fluence of the field SARS Cov-2, φ (coronavirus-particles/cm^2.s), and their report will be finalized by radiobiological and epidemiological studies to determine the number and energy of the photon that will be equivalent to the Coronavirus particle.

Keywords

Radiation Dosimetry, Effective Dose, Detriment, Coronavirus Particle, Virology, Radon Exposure.

General Remarks

Direct human epidemiological data from the Life Span Study (LSS) for the incidence of cancer over a period of 47 years (October 1959 - December 1997) led to the postulate of a linear relationship between a given dose increment and an increment in the probability of incurring cancer and hereditary effects, LNT model. This relationship can be written as "Risk = Nominal risk coefficient x Organ dose" and is valid at low dose and low dose rate, below 100 mSv (or 100 mGy) [1].

The quantities in the equation are: the radiation risk (R) is the probability of a certain group of the population for becoming ill or for dying due to the consequences of having been additionally exposed to ionizing radiation; the risk coefficient for organ or tissue (R_T), also called detriment, is the probability of the occurrence of a stochastic effect per dose and per time or age interval; the average absorbed dose in the tissue or organ T, D_T, is the total energy transmitted to a tissue or organ by its mass. Total detriment is the

sum of the damage to each organ part of the body (tissues and / or organs), [2].

The number of radiation-induced fatal cancer cases can be calculated from the mortality rate λ_r (t) of a population group at age t. The mortality rate is the number of radiation induced cancer cases dN_r (t), in the time interval t + dt, divided by the number N_o (t) of members of a population group who live of the time t, and by the time interval dt, i.e., dN_r (t) = N_0 (t)·λ_r (t).dt. The analogue of this equation for the rate of spontaneous cancer due to the background (index s) in the group of unirradiated population is of the form dN_S (t) = N_0 (t)· λ_S (t)·dt.

Using the linear dose-effect (survival) relationship, the risk of radiation-induced cancer, assumed to increase linearly with the radiation dose, is defined by equation (1),

$\lambda_r(t) = a\ D(t)$, (1)

The absolute risk coefficient (AR), $r_a \equiv a$, and the relative risk coefficient (RR), or r_r, are defined by the relations (2)

$$r_a \equiv \frac{d\lambda_r(t)}{dD(t)} = \frac{dN_r(t)}{DN_0(t)dt};\ r_r \equiv \frac{r_a}{r_s} = \frac{dN_r(t)}{DdN_s(t)}. \quad (2)$$

The excess absolute risk (EAR), sometimes called the attributable risk, is defined as EAR = Incidence in exposed population minus Incidence in unexposed population. The radiation risk determined at high doses can be extrapolated to low doses using the additive or absolute risk projection model (ARP model), with the dose-dependent and age-independent probability rate. In case of exposure to SARS Cov-2 virus, for the Romanian population of approximately 1,911,546 people tested for two years, the number of deaths is 98490. It results that the mortality rate is 1,911,546 / 57696 x 2 y = 0.01509 = 1.51 x 10^{-2} / PY, [3].

For the unexposed population, the probability per unit time of dying from cancer at time t, λ_s (t) from the background, added to the radiation death rate, λ_r (t), is λ (t) = λ_s (t) + λ_r (t). After a single irradiation at dose D and an age t_e (age at exposure), the total death rate in the population at time $t \geq t_e + \tau$, τ being a constant time lag, can be written as follows

$\lambda(t, t_e) = \lambda_s(t)\ [1 + r_r(t_e)\ D)]$, (3)

Where $r_r = r_a / \lambda_s = \lambda_r / \lambda_s D$, is the excess relative risk coefficient (ERR), equal to Relative Risk (RR) minus 1. The relative risk (RR) is the Incidence in exposed population per Incidence in unexposed population. Lifetime risk estimation is done, for example, using the multiplicative or relative risk projection model (RRP model). In this model, the probability per unit time of dying from radiation-induced cancer at a certain age is that after reaching this age (conditional probability) it increases with age at the same rate as the rate of cancer due to the background (λ_s (t) = k t^5).

The absorbed dose, D (t) = $\dot{D} \cdot t$, fundamental quantity of dosimetry, is defined in conditions of electronic equilibrium by equation (4),

$$D\ (Gy) = 1.6 \times 10^{-10}\ \Phi\ (cm^{-2}\ sec^{-1})\ E\ (MeV)\ \frac{\mu_{ab}}{\rho}\ (cm^2\ g^{-1})\ t\ (s), \quad (4)$$

Where 1.6 x 10^{-13} is the number of Joules in 1 MeV, 10^3 is the conversion factor from g^{-1} to kg^{-1}, E (= hv) is the energy of the photon, in MeV, (μ_{ab} / ρ) is the mass energy - absorption coefficient of in (cm^2 / g), t is the irradiation time in seconds (s), and Φ is the fluence rate in particles per cm^2·seconds, for 1 Gy, defined in equation (5),

$$\dot{\Phi} = \frac{6.25 \times 10^9}{E\ (MeV)\ \frac{\mu_{ab}}{\rho}\ (cm^2/g)}\ \left(\frac{particles}{cm^2 s}\right). \quad (5)$$

Example: Fluence rate Φ = 2.04 x 10^{11} photons / cm^2·s for E = 1 MeV and (μ_{ab} / ρ) = 0.0307 (cm^2 / g).

For external exposure to environmental radionuclides (air, water, and soil), it is possible to determine the maximum duration of standing (or irradiation) in those contaminated environments. This is obtained using the equation "Dose rate = Estimated concentration × Dose rate coefficient". To solve this equation, there are reports with tabulated dose rate coefficients for submersion in contaminated air, for immersion in contaminated water, and for exposure to contaminated soil, [4]. By dividing the effective limit dose (Sv) in the chosen environment, at the calculated dose rate (Sv / s), the maximum stationary time, T (s), is obtained in the contaminated environment (air, ground water) using equation (6),

$$T\ (s) = \frac{Dose\ limit\ (Sv)}{Dose\ rate\ \left(\frac{Sv}{s}\right)}. \quad (6)$$

Regarding internal exposure to noble gases, epidemiological studies have confirmed the risk of fatal lung cancer associated with inhalation exposure of Radon (^{222}Ra origin ^{238}U, $T_{1/2}$ = 4.5 x 10^9 y) and Thoron (^{220}Th origin ^{232}Th, $T_{1/2}$ = 1.4 x 10^{10} y) in homes and at work. We mention that Radon ($T_{1/2}$ = 3.8 days) and Thoron ($T_{1/2}$ = 55.6 sec) are fixed in the lung. The detriment per unit exposure to radon daughters is 1.41 x 10^{-4} per (mJ h m^{-3}) or 5 x 10^{-4} per WLM for long-term cancer risk for workers and the same for members of the public. Conversion from exposure to dose it was done based on equal detriment. The dose coefficients are 12 mSv per WLM (or 18.8 mSv per MBq h m^{-3}) for workers and 9 mSv per WLM (or 14.1 mSv per MBq h m^{-3}) for the population [1].

Regarding COVID 19, the symptoms associated with this virus include, among other things, a massive inflammatory reaction that will destroy the epithelial cells of the lung and lead to respiratory complications for infected patients [5]. The lung-specific epithelial cells are placed between the environment and the body and have several roles, among which: the initiation of the immunization action, the protective barrier, the repair of possible damage caused by the appearance of viruses [6].

The total age-specific rate of lung cancer for chronic smokers (index s) and chronic non-smokers (ns index) is given by equation (7),

$\lambda_s(t) = \lambda_{0,\ ns}(t)\ [1 + S(t)] \cdot [1 + \bar{r}\ E(t - \tau]$, (7)

where $\bar{\Gamma}$ is the age-averaged, relative excess risk coefficient, and E (t - τ) = $\dot{E}$ [t - τ] is the cumulative exposure to radon daughters up to age t -τ. The function S (t) characterizes the enhancement of this rate by smoking, [7].

Method and Materials

Malignant tumors are exposed to beams of ionizing radiation of a certain dose D (energy per unit mass of tumor) to destroy cancer cells. The best description of the tumor's response to the incident radiation is their survival fraction (SF), i.e., the ratio between the number of surviving cells (N) and the number of initial cells (N_0), $SF \equiv N / N_0$.

In the dose range (0 - 5 Gy), the probability of survival, p (survival), is described by the linear-quadratic equation (LQ) model, presented in Figure 1 [8], for the parameters $\alpha = 0.1$ $Gy^{-1,}$ $\beta = 0.0125$ Gy^{-2} and $\alpha / \beta = 8$ Gy,

$$p(survival) \equiv SF = \exp(-\alpha D - \beta D^2) \quad (8)$$

The linear coefficient, α (Gy^{-1}), with values between 1 x 10^{-1} and 5 x 10^{-1}, describes the linear component of the cell's sensitivity to killing on a graphical representation of the survival fraction (on the logarithmic axis) as a function of dose (on the linear axis). The quadratic coefficient, β (Gy^{-2}), with values 1 x 10^{-1} and 5 x 10^{-2} Gy^{-2}, describes the increased sensitivity of the cell to higher radiation doses. The α / β ratio (Gy), with values between 1 and 10 Gy, represents the dose at which the linear contribution (αD) is equal to the quadratic contribution (βD^2) in killing the cell.

The dose required to sterilize a malignant tumor is, for example, 60 Gy. Medical practice shows that it can be administered in 30 fractions at the absorbed dose of 2 Gy per fraction, LQ model.

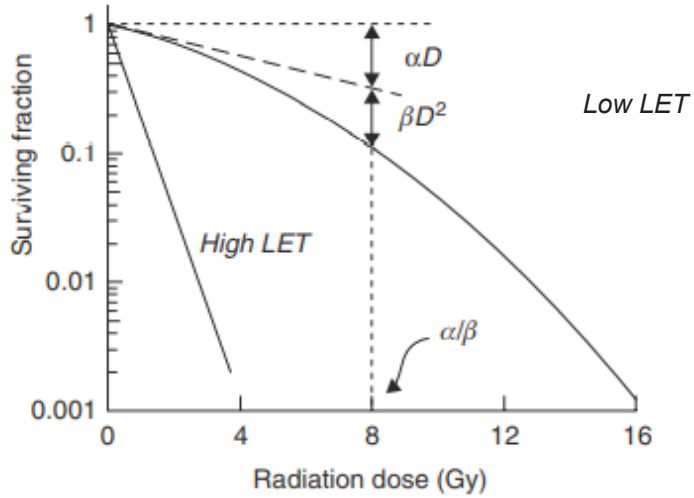

Figure 1: The fraction of surviving cells is plotted on a logarithmic scale against dose on a linear scale. LET- Linear Energy Transfer.

The cell survival fraction, SF_d, at a dose fraction d, is $SF_d = \exp(-\alpha d + \beta d^2)$. Assuming equal effects on each successive fraction, in a series of fractions, the total effect, (E) is given by equation (9),

$$E \equiv -\ln (SF_d)^n = (\alpha + \beta\, d)\, D, \quad (9)$$

Where D is the total radiation dose in n dose fractions d, D = n.d. Equation (9) represents the relationship between total Isoeffect, E, and dose per fraction, used in fractionated radiotherapy and radiobiology.

The ratio E/α in Gy, called the biological effective dose (BED), is a measure of the effect, E, on continuous or fractional irradiation. For a total dose D, in n fractions of size d, BED is defined by equation (10),

$$BED \equiv \frac{E}{\alpha} = D\,[1 + d/(\alpha/\beta)] \quad (10)$$

Radiological protection based on radiation dosimetry (external and internal) limits the absorbed dose in organ and tissue, D_T (Gy), equivalent dose, H_T (Sv), effective dose, E (Sv), and committed effective dose, E (50), to radiobiological and epidemiological established values. For example, for public exposure: 1 mSv / y - to prevent cancer and hereditary diseases, 9 mSv for ^{222}Ra (radon) and ^{220}Rn (thoron) to prevent lung cancer, 15 mSv for eye protection, 50 mSv for skin protection, 500 mSv- hand and foot protection. Moreover, a dose of 4,000 mGy throughout the body represents cancer death [1]. The particles that generate this dose can be, for example: photon (X-ray, γ-ray, bremsstrahlung), electron (10^{-18} m, 9.1 x 10^{29} g), proton, neutron (10^{-15} m; 1.6 x 10^{24} g), the nucleus (10^{-14} m) and the ionized atom (10^{-10} m). Taking these limits as an example to avoid radiation-induced diseases, this paper considers it necessary to have such specific limits for diseases caused by nanoparticles (viruses). Instead of the dose, the particle fluence and fluence rate are proposed. It allows the establishment of fluence thresholds in various stages of the spread of virus diseases.

The dosimetric approach to SARS Cov-2 virus infection, an inert biochemical complex [9,10], valid for any type of lethal virus, is to transform mortality to the detriment, using the number of viruses (or viral cells) per (cm^2. second). Based on the knowledge of the nominal probability coefficient (fatality) for any fatal virus-induced disease, compared to the reference fatal disease due to radiation, a hierarchy of all fatal diseases can be made. There is currently a recommended detriment for cancer of 5.5 x 10^{-5} per mSv for the entire population, 4.1 x10^{-5} per mSv for adult workers and the detriment for radon exposure of 1.41 x 10^{-4} per (mJ h m^{-3}). Also, the detriment for skin cancer of 10^{-4} per mSv, and the estimated total absolute risk at 8.7 x 10^{-4} per person-year-sievert (PYSv), composed of 6.7 x 10^{-4} $(PYSv)^{-1}$ per 3,000 cm^2 of exposed skin and 2 x10^{-4} $(PYSv)^{-1}$ per 15,000 cm^2 of unexposed skin, are also recommended, [11].

In addition, as known data from radiation exposure, we mention that the whole body's exposure to ionizing radiation at values of the effective dose between 3 and 5 Gy represents the death of an adult, due to the loss of bone marrow stem cell function. The lethal dose, $LD_{50/60}$ = 4 Gy, means that half of the number of irradiated individuals would be expected to die in 60 days. The value of this dose represents the density of ionized atoms, of 8.23 x 10^{17} affected

atoms per kg of tissue, compared to the total number 7 x 10^{27} atoms of a reference person. The corresponding photon fluence is Φ = 8.14 x 10^{11} photons per cm^2.

In the first stage of the virus, the working hypothesis consists in the equality between the fluence rate of the virus field φ (coronavirus - particles / cm^2.s) and the flow rate of the photon field Φ, (photons / cm^2.s). This equality starts from the assumption of the equality of the biological effects of the two particles, virus (150 nm, 1 ag) and photon. The photon is a massless "particle", uncharged, of energy E = h.ν and an associated impulse, P = h·k, where h = 6.63 x10^{34} J·s = Planck's constant, k = wave number. The advantage of this equality is that the photon frequency, ν[Hz], at the beginning of the experiment is known, and then it changes until the two effects coincide. Another advantage is that the photon biologic effect is known.

In the second stage, the hypothesis at the beginning of the studies consists in the equality between the photon fluence and the infected cell fluence, considering that the appearance of the coronavirus infection in cells corresponds with the appearance of the radiation-induced stochastic effects. The results of the radiobiological and epidemiological study, exactly as the radon exposure, "risk / WLM per risk / Sv", will allow to obtain the ratio "risk / coronavirus fluence per risk / photon fluence". This report can be used to scale all fatal diseases. We are currently looking for data on the respiratory tract model and model for gastrointestinal, to be able to give an example of calculation.

The experimental stages can be performed in a radiopharmaceutical production center, equipped with a hot chamber, photonic radiation sources, electron microscope, and virology and radiation biology laboratories. The workspace must have a gradient atmospheric pressure and an activity-measuring device generated by radon and thoron, so that their effect does not add to comorbidities. A Cobalt-therapy unit or a Clinical linac of 10 MeV could be used as a radiation source, which are radiologically protected. The ^{60}Co source parameters are as follows: source activity -15 k Ci, photon average energy - 1.25 MeV, source distance - skin (SSD), variable, between 50 and 100 cm, and standard irradiation field, variable between (4 x 4) cm^2 and (40 x 40) cm^2, [12,13].

Comments

Nuclear decay, just like naturally occurring cancer, is a spontaneous process that happens by chance, so it needs to be looked at from a statistical point of view. While the radioactivity of some elements decreases by disintegration, they can occur by the disintegration of other elements and thus its radioactivity increases. To find out the law of disintegration, at any studied element, by continuously removing all its disintegration products, it is found that its radioactivity decreases exponentially with time. As with spontaneous cancer, the number of decaying atoms, dN, is proportional to the number of radioactive atoms, N, present at time t, with the time interval dt, and with a decay constant λ, specific to each element. i.e., dN = λ N dt. After integration from N_0 to N and from 0 to t, the law of radioactive decay is obtained, N = N_0 exp (-λ t), [14].

The relationship between the generating radionuclide (G) and one derived (D) from the distant ones can be a secular equilibrium ($\lambda_G << \lambda_D$), when after about 10 half-lives of the derived radionuclide, the activities of the two become equal and remain in secular equilibrium, because being in the same test, what one loses wins the other. This situation would correspond to healing at home when the viral load is less than 50% of the survival fraction and remains constant over time. The second situation can be a transient equilibrium ($\lambda_G < \lambda_D$), which is established when the activity of the derivative after the intersection with the activity of the generator becomes higher and its decay rate depends on the rate of the generator. This situation would correspond to the patient with comorbidities. There is also the situation of nonequilibrium ($\lambda_G > \lambda_D$) when the life of the generator is shorter than that of the derivative. This would apply to people who are not infected with lethal virus.

Coronavirus, as parent and the series of progeny (or strains), being natural, compared to the natural phenomenon of radioactive decay, and helps to establish a model for interpreting of the virus route: testing, quarantine, hospitalization and ATI. Another interpretation would be that the last strain of any natural lethal virus is a non-lethal strain, just as, after 17/15/10 progeny radionuclides from the element ^{238}U / ^{235}U / ^{232}Th, a stable element appears, ^{206}Pb / ^{207}Pb / ^{208}Pb.

Coronavirus disease, seen by a dosimetry physicist and a nuclear physicist, is very similar to nuclear decay. The statistical model of nuclear decay helps to understand the virological model more easily. Moreover, this model indicates a gradient of atmospheric pressure in the areas of diagnosis and treatment, as well as monitoring of radioactive gas (Radon and Thoron) generating fatal lung cancers. About half of the natural background dose absorbed by the public per year is due to radon exposure.

Conclusion

The risk of fatal cancer and hereditary diseases induced by radiation, controlled by the absorbed dose may be a model for the risk of lethal viral diseases (coronavirus) controlled for example by fluence of particles (virus and cells).

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