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A CONTRIBUTION TO THE CURRENT DEBATE ABOUT THE ADEQUACY OF THE LINEAR-NO-THRESHOLD (LNT) MODEL FOR THE RISK RESULTING FROM RADON EXPOSURE

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Abstract. *The Linear-No Threshold Hypothesis (LNT) states that risk from ionizing radiation is linearly related to dose with no dose threshold below which there was no risk. The LNT is an important fundament in practical radioprotection and for assessment of population risk, e.g., of estimating lung cancer risk or incidence attributable to exposure to indoor radon. The popularity of the LNT stems largely from its mathematical simplicity and therefore, its practicability. It seems that this has obscured the question of whether it is physically true, or "only" a useful practical rule. Distribution of exposure and dose to radon through the population is strongly right-skew, with the bulk of dose low. Therefore, attribution of risk, i.e., mainly lung cancer incidence, depends strongly on the risk model for low dose. As long as no micro-dosimetric model exists which causally relates incident radiation flux or exposure to radon progeny to a sequence of effects, starting on sub-cellular level, which results in clinical evidence, it is impossible to make statements on the effect of very low doses, since it is in principle impossible to extend empirical epidemiological inference to arbitrarily small doses. Therefore, epidemiological findings are extrapolated towards low doses. The most quoted large-scale epidemiological radon meta-study is Darby et al. (2006), which concludes that the LNT model is statistically compatible with the findings. This has been essentially corroborated by newer studies. However, with availability or more data, there seems to be increasing evidence that the model may not be applicable to estimate risk for low doses, which represent the bulk of exposure, if the objective is assessment of population risk. We review literature about the strongly debated question about validity of the LNT. Data are not publicly available, therefore statistical re-analysis is impossible. However, published information in the form of graphs and statistics allows some hypotheses alternative to the LNT. The debate is so serious because of the political consequences regarding radon abatement policy. We refrain from stating any "alternative truth" but investigate the possible consequences for risk assessment and what they entail for radon regulation and policy, resulting from different risk models.*

Keywords: *detriment due to radon exposure, geographical distribution of radon induced detriment, Linear-No Threshold, alternative hypotheses*

1. INTRODUCTION

High doses of ionizing radiation cause acute symptoms while lower doses increase the probability of health effects such as cancer. Therefore, one speaks of stochastic effects. For Rn exposure (more precisely, exposure to Rn progeny by inhalation), this mainly concerns lung cancer, see e.g., [1]. The functional relationship between dose and the probability is used to estimate the risk caused by radiation exposure. On an individual level, risk denotes the chance to suffer a detriment, on the collective level, the size of a detriment to the society, for example, the estimated number of cancer fatalities. Epidemiological studies are performed to acquire the data from which the function is estimated.

The dose-risk model allows estimation of the detriment if the geographical and demographic distribution of exposure or dose are known. From this, conclusions about radiation protection policy are drawn. For Rn, this has been laid down in regulation, codified by the IAEA [2] and similarly by the European Union [3]. The latter requires by its Member State to establish National Radon Action Plans to mitigate the effect of Rn exposure by stating Rn concentration reference levels and action to be taken in areas with high Rn levels. Remember that radiation protection has two objectives: protection of individuals against high exposure, even if these are few persons and therefore contribute little to the total detriment to society, and to the society by mitigating the overall detriment, even if individual risk is relatively low. If the distribution of exposure is strongly right-skew, as is the case for Rn (visualized as a map, e.g., in the European Atlas of Natural Radiation [4]), the bulk of overall detriment is contributed by many cases of low individual risk, whereas cases of high individual risk exist, but are comparatively few. This explains the political relevance of the choice of risk model.

1.1. Scope and outline of this study

This paper is about investigation of a *possible effect of the choice of risk models on estimation of the detriment caused by radon*. In other words, *we do not attempt to quantify the absolute detriment according*

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to a certain model, nor do we decide about the correctness or the applicability of particular risk models. We also do not discuss their plausibility on radio-biological grounds. Furthermore, we do not present new epidemiological data, nor do we re-evaluate existing ones. This is important to emphasize, because discussion about risk models is controversial and sometimes appears almost a matter of faith. However, since the LNT seems almost undisputed in the Rn community, to our observation, we present a number of references questioning this position and discuss their possible implications in radon risk mapping.

In the rest of the introduction, we present some basics of statistical terminology. In Sect. 2, we present different risk models. Sect. 3 shortly presents the Rn data which are used to exemplify the effect of different risk models. In Sect. 4, we show European maps of estimated detriment caused by Rn relative to the estimated detriment assuming a baseline model (the LNT, see below). Conclusions are drawn in Sect. 5.

1.2. Relative risk and statistical limitations

Individual and collective risk are by nature sigmoidal functions of exposure or dose because they lie in the intervals (0, 1) and (0, n), respectively, where n denotes population size. More convenient are relative risk measures. Most common is the quantity *relative risk* (RR), defined as the "The rate of disease in an exposed population divided by the rate of disease in an unexposed population" (e.g., ICRP 103, p. 24 [5]). the quantity RR-1 is called *excess relative risk*, i.e., the additional risk created by the exposure, on top of some "background risk". For the RR, "detection limits" can be calculated, which give the lowest effect which can be detected with given confidence. Essentially, it depends on the number of observations or cases investigated and evidently the necessary number of observations N is the higher, the smaller the effect is that shall be detected. Since N cannot be increased at wish, deliberately small effects cannot be detected. (See e.g., [13], annex A, par. A18f. p.37 for discussion.) This is an important point in the discussion about whether monotonous risk models (such as the LNT) can be extrapolated "downwards", i.e., towards small exposure (see [13], annex A, par. A76f, p.56).

Another limitation of epidemiological studies of the effect of low doses is that for physical reasons no zero-dose control group can exist. We are always inevitably subject to low exposure to cosmic rays and internal radiation of the body (by ^{40}K). For Rn, exposure below outdoor Rn concentration (typically 2 - 20 Bq/m^3) is practically impossible.

2. RISK MODELS

The simplest function that relates dose and effect is a constant, which would indicate no effect. According to most authors, this contradicts epidemiological evidence for Rn doses which are encountered in areas with enhanced Rn occurrence. There seems to be agreement that health effects can be detected above doses of about 50 - 100 mSv, which corresponds to exposure to indoor Rn of roughly 100 Bq/m^3 over several decades. In this sense, there is not controversy about the detrimental effect of radon exposure at high radon concentrations, which has been proved for uranium miners [6]–[9]). The discussion is focused therefore on low doses.

2.1. The Linear No-Threshold Hypothesis, LNT

The second simplest function is a straight line through the origin, RR = b × dose, called LNT model or hypothesis. It has first been formulated by Hermann Muller, Nobel price lecture 1946 [10], but it has been also challenged from the very beginning [11]. Nevertheless, the LNT is currently the most commonly used model of Rn risk. This hypothesis suggests that any dose will produce damage in the body. The most relevant publication is [12], which determined an increase in the excess relative risk (ERR) of lung cancer of 8.4% (95% CI 3.0%-15.8%) per 100 Bq/m^3. (Accounting for uncertainty of exposure, i.e., correcting for regression dilution, leads to almost the double ERR estimate, also discussed in [13]. Considering further uncertainty may lead to even higher values [57][2]). The main limitation of the LNT theory is that it has been derived from the exposure to uranium miners (i.e., mostly high radon doses) and its application to most residential exposure (i.e., low radon doses) remains uncertain. Recent analysis of contemporary miners, which are exposed to lower radon concentrations due to prevention measurements at workplace and better radon characterization, still suggest a linear positive relationship with no threshold [6]. The UNSCEAR, in its 2012 report [13], already acknowledged this issue and described some alternative dose-response relationships.

2.2. Critique of the LNT and alternative Models

In spite of the popularity of the LNT, doubts about its correctness and in consequence reasonability and applicability have arisen for years. A number of researchers have expressed their doubts [7], [14], [15]. They found non-linear dose-response models that may also fit the data. Similarly, Tubiana et al. [11] question the LNT based on epidemiological and biological evidence. Another recent review of LNT critical positions is found in [12].

Here we summarise the most relevant, all of them with some scientific support that should not be directly dismissed: i) linear with threshold (LT), ii) superlinear, iii) sublinear, iv) U-shape, and v) hormesis. Several models are shown schematically in Fig. 1 (similar to [13], annex A, par.20f. and Fig.I, p.27). Arguments and references for the different positions are quoted below. For the rationale of alternative models, see also [13], annex A, par. A82 ff, p.57.

One main argument of the adherents of the LNT is that it fits well to epidemiological data and modification does not provide improvement of measures of such as r^2 or AIC. Modification resulting in formally more complex models would not be justified in view of the parsimony principle (Ockham's razor). This is countered by the fact that measures of fit are no valid argument in the range of the explanatory variable (dose) in which no data of the dependent variable are available (i.e., epidemiological data for low doses); the parsimony principle serves as a guideline but cannot override

[2] We thank one reviewer for pointing to these adjustments.

evidence (remember Einstein's succinct dictum that a theory should be simple, but not too simple). We want to emphasize again, that the case cannot be resolved based purely on epidemiological evidence, because of the statistical constraints addressed in section 1.2.

On the other hand, one should not succumb to the fallacy that because one model cannot be sufficiently proven with data, a certain other one must be correct. After all, the same statistical limitations apply to all dose-response models below a certain dose.

i) Linear with threshold (LT): this model proposes that below certain doses (threshold) no damage is produced. It may happen due to both: i) a minimum number of damage cells required for producing cancer, and ii) a stimulation of the immune system for eliminating cancer cells [13], [16], [17]. In this regard, Dobrzynski et al [18] support the existence of a threshold for radon exposure. They concluded that the relative risk (RR) of lung cancer is independent of radon concentrations lower than ~850 Bq/m³ by using Bayesian analysis on data from 34 epidemiological studies. To our understanding, a threshold to long-term exposure of 850 Bq/m³ seems too high, and more realistic values (if it were the case) could be in the order of 50 – 100 Bq/m³ based on the findings of other studies regarding the dose-effect relationship for radon exposure.

The arguments for a LT-type relationship have also been forwarded regarding exposure to ionizing radiation other than Rn: For example, a number of studies in recent years have found no increase in the risk of cancer or leukaemia from enhanced natural or anthropogenic background radiation [19]. In [20] and [21] the authors claim that the LNT is also questionable for atomic bomb survivors.

ii) Superlinear: Under this model, the risk increases under low and very low doses. Two main hypotheses support this effect. On the one hand, a bystander effect, where non-irradiated cells can also suffer radiation-induced damages [20], may be more important under low doses than for moderate/high doses [13]. On the other hand, the efficiency of DNA recovery may be low under low doses [13], [22].

iii) Sublinear: These models suggest an adaptative response to low doses, which stimulate DNA reparation [23]. Therefore, the risk under low doses is lower than the expected under a LNT model. In Fig 1 an LT model is shown as special case of sublinear models (black curve) an another one which starts at a threshold an approximates the LNT with increasing dose.

iv) U-shape: it is a special case of sublinear model where a minimum value of ERR is reached. The values are still positive in these theories, meaning that any exposure to radon would generate adverse health effects, although an adaptative response would make it lower that the risk assumed under a LNT model. However, under very low doses, the risk may be compared to moderate dose exposures. An example of these models can be found in Rosenberger et al [24]. After evaluating the link between indoor radon and lung cancer in a sample of 8,927 cases and 5,562 controls, they found a minimum risk at a concentration of approximately 58 Bq/m³, with no linearity, at least, up to 200 Bq/m³.

v) Hormesis: This also is a special class of sublinear model, which suggest that the adaptative protection from low doses generates a positive effect, and thus a beneficial health effect. This would be reflected in negative values of ERR [25]. Under this mode, the exposure to low radon concentrations will generate a positive impact due to the adaptative response of the body, reaching a minimum value over with the slope of the curve will be positive, for example, Thompson [27] reported that this change in the slope may happen at radon concentrations of approximately 70 Bq/m³. Finally, with higher concentrations there would be a switch from negative to positive values of ERR, which may happen for concentrations higher than 150 Bq/m³ according to [26], [27].

The first studies suggesting a hormetic effect of Rn seem to data back to B. Cohen in the 1990s, [28], [29]. Since the studies were of ecological type they were criticized as biased because confounding effects like smoking habits are difficult to consider, e.g., [58]. (Today the preferred design of epidemiological studies is instead of case-control or cohort type which has higher statistical power but is much more difficult to realize.). Cohen later responded to the critique, [30], but the debate appears ongoing.

Becker et al [31] presented an interesting compilation of radon data from Central Europe with a cost-benefit analysis of radon reduction programmes. The results indicate possible biopositive effects of low exposures and support the hypothesis of a nonlinear human response to low and intermediate radon exposures. A fierce advocate of hormesis is Calabrese [32]. Sanders [33] has published an entire book promoting radiation hormesis, discussing numerous examples. For some years, the "Polish school" of Dobrzynski et al. [18] pleaded for hormesis of Rn exposure. A summary of their work is given in [34].

An often-quoted example is that in some areas of Ramsar (Iran) known as HBRA (High Background Radiation Area), radon concentrations are up to 3700 Bq/m³. The study published elsewhere evaluated the relationship between radon concentrations and lung cancer incidence and found a negative relationship between these variables [35].

Studies of the association between ^{222}Rn and lung cancer incidence on the island of Guam described by Denton and Namazi suggest a hormetic effect between the two variables. Possible confounding effects related to smoking and ethnicity were also considered and found not to be significant [36].

A much-disputed subject is radon therapy (for a very brief description: [37]), often used in favour of radiation hormesis, because a positive therapeutic effect for certain diseases seems to be proven. A dosimetric investigation of the Gastein (Austria) Rn therapeutic facility is given in [38]. Reviews of possible therapeutic effects are given in [39], [40]. Among studies which found positive effects of Rn therapy we name [41], [42]. A strongly pro-hormesis review has been provided by [43]. Since Rn therapy is business, one must be careful about biased publicity, as shown in [44].

The German Radioprotection Authority (BfS) has issued a skeptical comment about hormesis: "Possible positive effects of ionising radiation refer to individual cases and must not be transferred to the population." (www.bfs.de/EN/topics/ion/effect/hormesis/hormesis.htm, last visited 30 Nov 2023).

A summary of the controversy about the LNT can be found in Wikipedia [45].

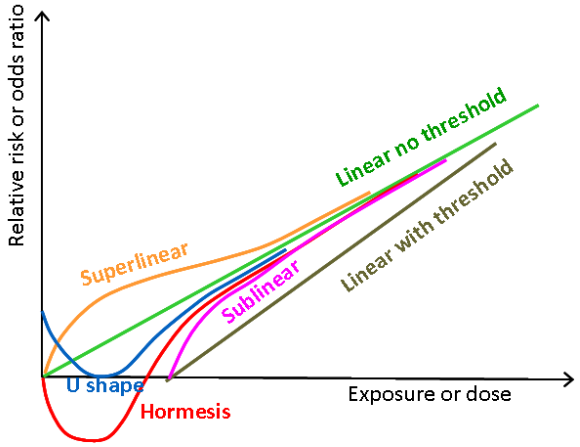

Figure 1. Possible models of the dose - risk relationship.

An important consequence of the uncertainty of the risk related to low dose is the following. Since the majority of people is exposed to low doses, namely in the "uncertain range", the detriment or collective risk, Σ dose × (number of persons exposed do this dose) × (risk factor for the dose, which is uncertain) is also uncertain. Nevertheless, such reasoning is often assumed (e.g., [46]), although the resulting numbers can be misleading, and are in any case extremely uncertain. This applies not only to Rn, but for example to the statement about fatality after the Chernobyl accident: indeed, many people were exposed, but the majority only to very low dose. Application of the LNT in this dose range is clearly problematic. Therefore, the ICRP - while maintaining the LNT pending better knowledge - has nevertheless stated that computing collective doses for collective risk estimation is "inappropriate" (ICRP 103, executive summary (j, k), p.13 [5])

3. Radon data

The data for this study are taken from the European Indoor Radon Map, part of the Atlas of Natural Radiation [4]. For this paper, the version (2019) of the database was used. It comprises about 1.2 million measurements, ground floor rooms, aggregated into 10 km × 10 km cells. It consists of the following cell wise statistics: AM (arithmetical mean), SD (standard deviation), AM and SD of ln-transformed data, minimum, median, maximum, and N (data per cell). In total, there are 29,539 non-empty cells. The original data are not openly accessible, but protected property of the National Competent Authorities, which contributed to the map. They aggregated the data into the cells and sent them to the JRC of the EC for further joint processing, mapping, and possible further evaluation. Also, the aggregated data are not openly accessible, but can be obtained for defined purposes from the EC on justified request. The latest classed-post map of the data (version 2021) is shown in Fig. 2.

An interpolated European Rn map has been generated by Elío et al. [48]. Briefly, its method is lognormal regression kriging. The regression step used geology (scale 1: 5 million), uranium and potassium concentrations (also taken from the Atlas database) as independent variables and the cell means of the European Rn database as independent variables. The residuals were subjected to ordinary kriging. As final step, the result was back-transformed into the original linear scale by taking advantage of log-normality. (The method implies certain bias, but this appeared tolerable given data uncertainty. The adjusted r^2 for data against predictions is 0.20.) The resulting map, whose resolution are the same 10 km × 10 km cells as the ones of the database is shown in Fig. 3. The cell entries of the interpolated map were used as inputs to the next step, section 4.

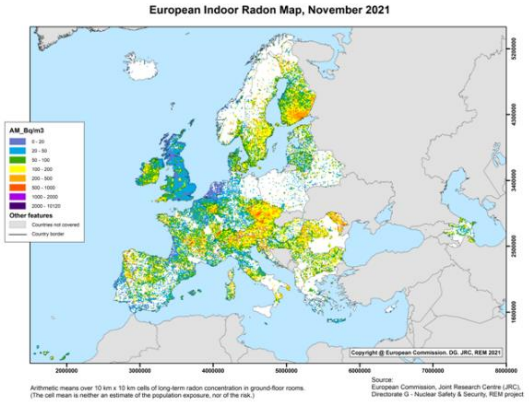

Figure 2. European indoor Rn map, arithmetical means per cell, according to the database underlying the European Atlas of Natural Radiation.

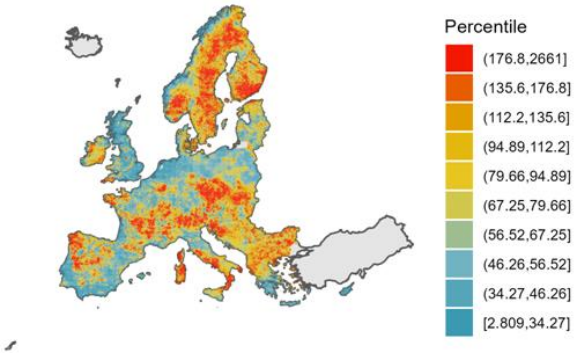

Figure 3. Interpolated indoor Rn map of Europe. For technical details, see text.

The histograms of the Rn concentrations per cell derived from the interpolated map, Fig. 3, are strongly right-skew, as shown in Fig. 4 (a). This applies even much more to the population-weighted concentrations (computed as Rn concentration × population per cell), Fig. 4 (b). The reason of this obvious difference is that most people live in areas with comparatively low Rn hazard. In most cases, cities, where most people live, are built in sedimentary basins with low Rn hazard.

Fig. 5 shows qualitatively the same information: about 37.4% of Europeans live in cells with AM(Rn)≤50 Bq/m³; about 75.7% in cells ≤100 Bq/m³; 95.8% ≤200 and 99.1% in cells ≤300 Bq/m³ (the maximum reference level permitted by the European BSS; at this point we want to note that the spatial variability of indoor Rn concentration is very high, geometrical standard deviation about 2 (e.g., [49]), which means that in a cell with mean 300 Bq/m³, houses with much higher and much lower concentrations may be encountered. In many EU countries, the definition of radon priority areas is based

on a certain probability to exceed the reference level, but not on the mean concentration).

This means that over 70% of Europeans are exposed to Rn at levels where the dose-risk relationship is uncertain, and could be very different from the LNT, according to some authors cited above. Consequently, models deviating from the LNT would cause radically different results of the detriment attributable to Rn exposure.

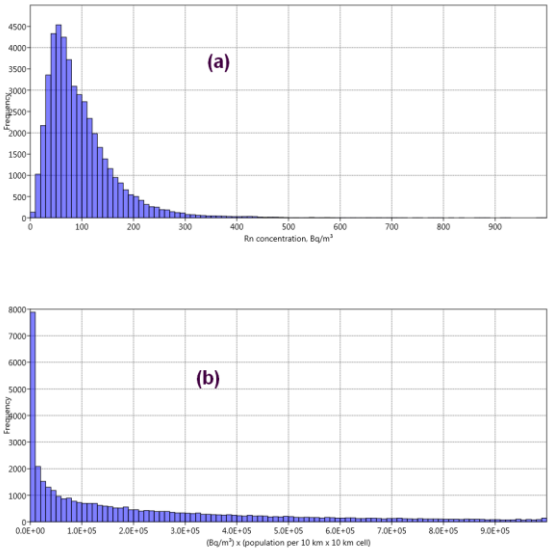

Figure 4. Histograms of (a) the Rn concentrations per 10 km × 10 km cells; (b) the population-weighted Rn concentrations per same cells.

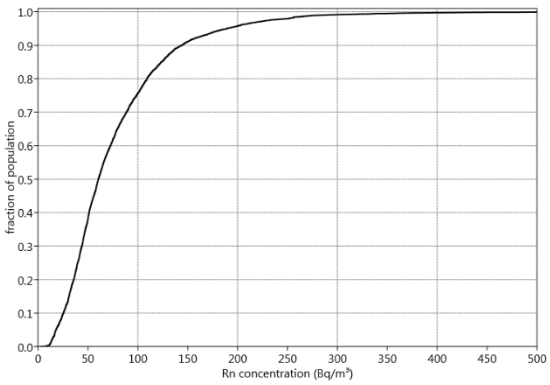

Figure 5. Fraction of the European population living in 10 km × 10 km cells with mean Rn concentration below the value given in the x-axis.

It should be noticed that this result is only an approximation of the true exposure distribution and affected by various sources of uncertainty. Among them are

1) also, within 10 km × 10 km cells, there is considerable dispersion not captured by substituting it by the cell mean.

2) the data are restricted to people hypothetically living in ground floor rooms, while in reality, in higher floor rooms exposure is usually lower.

3) Cell means are uncertain, because data of which they are computed are uncertain and because it is unknown how well they represent indoor Rn in the cell.

4) Here it has been assumed that people spend all time within the cell to which they were assigned. This does not reflect social reality in which mobility is rather the rule than the exception. No statistical information is available about this effect.

4. Results: maps of relative detriment

Briefly remind the notions of hazard, risk and vulnerability (e.g., [50], [51]). Hazard denotes the physical cause of risk and may be called potential risk. Its distribution is the one of its physical constituents (or their proxies); for Rn hazard, these are geology, geochemical concentrations (mainly uranium), soil properties, orography, and possibly many other (see [52], [53] for examples and discussion). Importantly, the European Rn map (section 3) is in essence a Rn hazard maps.

Hazard is linked to risk by vulnerability, which defines the conditions of possible exposure, such as floor level in a building, house construction characteristics; actual exposure, that is the presence of persons who are exposed (if there are no persons, there is hazard but no risk); and the risk factor, which numerically translates exposure or dose into risk and which is a function of exposure or dose. This is where the risk model steps in.

For each 10 km × 10 km cell (x), we computed the detriment $D(x) := AM(Rn)(x) \times$ (number of persons (x)) $\times RF(Rn(x))$. (AM denotes the arithmetic mean.) The vulnerability factor is currently not available to us, i.e., it has formally set to Vulnerability≡1. The number of persons has been taken from the European population density database that underlies also the European Atlas of Natural Radiation [4] (p.161). The relative detriment related to a model, RD(x; model) is defined as D(x; model)/D(x; LNT). This represents the additional collective risk due to Rn, estimated through a model, compared to the one estimated by assuming the LNT.

Analytic forms of the risk factor (defined as excess relative risk RR-1) are assumed as follows:

LNT

$RF(Rn)=\alpha\ Rn$, $\alpha=0.001$ " 10% per 100 Bq/m³

Sublinear

$RF(Rn)=\alpha Rn[1-\exp(-\beta(Rn-\theta)]$ for $Rn>\theta$, else=0. θ=threshold=100 Bq/m³, $\beta=0.016$ (Bq/m³)$^{-1}$

Superlinear

$RF(Rn)=\alpha Rn[1+\gamma\exp(-\delta Rn)]$, $\gamma=7$, $\delta=0.03$ (Bq/m³)$^{-1}$

Hormesis

$RF(Rn)=\alpha\varepsilon[-\zeta^2+(Rn-\zeta)^2]$ for $Rn<\theta$, $\varepsilon=0.03$, $\zeta=50$ Bq/m³, else like sublinear with $\beta'=0.035$ (Bq/m³)$^{-1}$

U-shape

$RF(Rn)=\varepsilon'(Rn-\zeta')^2$ for $Rn<\theta'$, $\varepsilon'=5e\text{-}5$, $\zeta'=50$ Bq/m², $\theta'=75$ Bq/m³, else like sublinear with $\beta''=\beta$, $\theta''=50$ Bq/m³.

To be sure, these analytical models, shown in Fig. 6, have been designed only for illustrative purposes and to demonstrate the effect of choice of model. They do not result from data fitting! To relate Rn concentration to dose, very roughly, 20 years with 100 Bq/m³ corresponds to about to 100 mSv (ICRP 137,[47]).

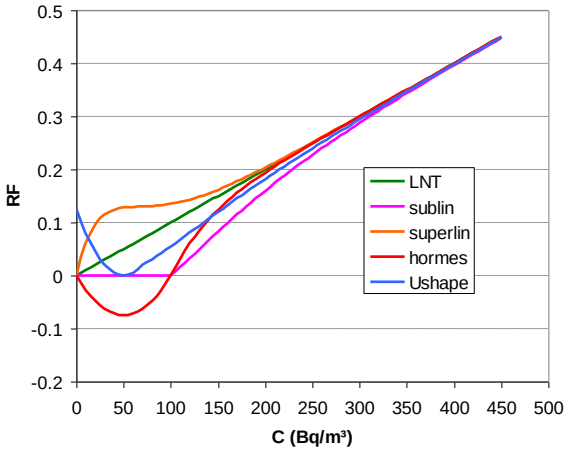

Figure 6. Risk factor as function of Rn concentration

European maps of RD(x; model) are shown in Fig. 7. The sum over all cells (x) of D(x; model) divided by D(x; LNT) gives the total relative detriment, shown in Tab. 1 for the 4 risk models.

Table 1. Relative detriment, equalling the total additional risk over Europe due to Rn according to different models, relative to the one estimated supposing the LNT model.

Model	Relative detriment
LNT	1
Sublinear	0.0014
Superlinear	1.74
Hormesis	-0.23
U-shape	0.59

The *superlinear* model leads to a much larger estimated detriment than the LNT model (Fig. 7, table 1), which is clear because the FR function is above the LNT function, RF(Rn; superlinear)>RF(Rn; LNT) for all Rn doses. On the other hand, the *sublinear* model leads to very small relative detriment, because in the Rn dose range in which most of the population is affected, <100 Bq/m³ (Fig. 5), no Rn risk is assumed. The situation is more complex for the *U-shape model:* While for very low exposure (about <25 Bq/m³), it leads to higher detriment than expected for the LNT, it is lower for all higher doses, which dominates the balance, resulting relative detriment < 1. The *hormesis* model, finally, leads to a negative relative detriment, or in fact a health benefit of Rn exposure. This is because the hormetic range in the assumed risk model (<100 Bq/m³) covers most of the population.

Again, we warn against taking the results literally, because the models were designed for demonstration of the effect only. However, the tendency is clear and similar results, in terms of order of magnitude, can be expected for realistic models - if they exist in reality, that is.

5. Conclusion

Traditionally, most models of radiation risk assume the linear no-threshold (LNT) model. It seems conservative, therefore reasonable for practical radioprotection. Without opting for any specific risk model, we showed that choice of the risk model has large influence on the detriment attributable to radon. If the scientific community should come to the conclusion that a model alternative to the LNT relating Rn exposure and risk should be used, this would have consequences on regulation and on Rn Action Plans. It can therefore be expected that the political impact would be considerable. If hormesis exists, Rn protection policy based on the LNT would even be counter-productive in the hormetic dose range.

In this paper, we showed that the choice of model does make a big difference for assessment of the detriment to the society; this is because - at least in Europe which has been used as an example, but probably not essentially different elsewhere - the large majority of the population is exposed to Rn at levels, for which the dose - risk relationship is highly uncertain.

On the other hand, Rn policy related to areas with high Rn exposure would probably remain by and large unaffected.

In spite of possible impact on Rn policy, we think that the discussion should not be suppressed for reasons of political or ideological unease – whatever the outcome in the future. One may have to revise the frequent statement that Rn is the second cause, or one of the leading causes of lung cancer after smoking. It has been proposed [54] to replace “LNT hypothesis” by “LNT concept” to relax the assumption that the LNT represents physical truth, but instead to indicate that it is a rule adopted for practical radioprotection.

Another possible issue shall be briefly addressed. It has been proposed [50], [55] to complement Rn abatement policy which mostly concentrates on high-hazard areas or areas with expected elevated individual risk, that is, Rn priority areas as defined conventionally, with areas where collective Rn risk is concentrated; these may be areas with low hazard but high number of people affected. Under the LNT, most detriment would occur there, but not in high-hazard areas, if in these population density is low [51]. To avoid that collective Rn risk is assigned to areas with (i) trivial (inevitable) Rn exposure, (ii) un-remediable Rn concentrations (too low to remediate with reasonable effort and cost) or relating to the subject of this paper, (iii) to Rn concentrations for which individual risk is questionable, a lower threshold of risk was proposed [53].

Future tasks will include (1) specification of alternative models according to literature, instead of heuristically exemplified ones as demonstrated in this paper. The “epistemic uncertainty”, that is, the uncertainty resulting from the choice of model (expressed by the variability of model outcomes, Tab. 1), would likely be reduced; (2) accurate consideration of individual exposure, e.g., like in [56], where floor levels are accounted for. (3) Input data of the analysis are affected by uncertainty of different type, whose propagation into the result (relative detriment) is difficult to handle. (4) Discussion of the possible impact on Rn regulation is not explicit subject of this paper but should be started as consequence of the findings presented here.

Finally, we would like to stress once again that this paper is no plea for any particular model - neither LNT nor any other -, but a mere demonstration of the effect which the choice of model has. Our approach may be compactly summarized in the following points to avoid misunderstanding:

- As a matter of fact, the discussion about the validity of the LNT for low doses of Rn exposure exists and seems to be gaining momentum.
- The authors are no experts in radon dosimetry, epidemiology or radiation biology. Our field of expertise concerning Rn is surveying, spatial modelling, mapping and assessing consequences for Rn policy.
- Therefore, we only report existing positions about the dose - risk relationship. We notice that they diverge for low doses, but we do not presume to decide on the correctness of one position or another.
- Hence, the subject of our manuscript is a *scenario study, not an attempt to decide which position is correct*. It is a "what ... if" study, intended as a contribution to the discussion of possible consequences.

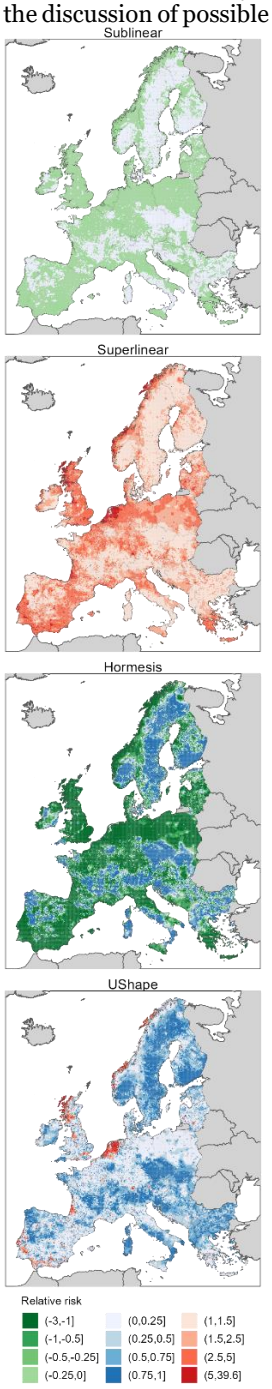

Figure 7. Additional risk due to Rn for different models of relative risk, relative to the LNT model (Green colours: Rn healthy! Blues: little Rn risk < LNT; Reds: Rn risk > LNT)

Declarations, data availability and acknowledgments:

The authors declare no conflicts of interest.

European radon data used in this study are not publicly available but can be obtained on justified request from the European Commission. The EU-JRC also provided the population data, aggregated in grid cells of 10km × 10km.

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