

We can do better than effective dose for estimating or comparing low-dose radiation risks

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Abstract—The effective dose concept was designed to compare the generic risks of exposure to different radiation fields. More commonly these days, it is used to estimate or compare radiation-induced cancer risks. For various reasons, effective dose represents flawed science: for instance, the tissue-specific weighting factors used to calculate effective dose are a subjective mix of different endpoints; and the marked and differing age and gender dependencies for different health detriment endpoints are not taken into account. This paper suggests that effective dose could be replaced with a new quantity, ‘effective risk’, which, like effective dose, is a weighted sum of equivalent doses to different tissues. Unlike effective dose, where the tissue-dependent weighting factors are a set of generic, subjective committee-defined numbers, the weighting factors for effective risk are simply evaluated tissue-specific lifetime cancer risks per unit equivalent dose. Effective risk, which has the potential to be age and gender specific if desired, would perform the same comparative role as effective dose, be just as easy to estimate, be less prone to misuse, be more directly understandable, and would be based on solid science. An added major advantage is that it gives the users some feel for the actual numerical values of the radiation risks they are trying to control.

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1. INTRODUCTION

Effective dose (ICRP, 1977) represents an attempt to provide a quantity which is proportional to the radiobiological ‘detriment’ from a particular low-dose radiation exposure – detriment representing a balance between carcinogenesis, life shortening,

and hereditary effects. Specifically, it is the sum of the equivalent doses to a number of radiosensitive organs/tissues, with each organ/tissue being weighted by a committee-determined tissue weighting factor. Effective dose was designed to facilitate comparisons of the generic risks associated with different radiation fields. More commonly these days, it is used either as a surrogate of cancer risk or to estimate cancer risk, particularly in the medical context.

The use of the effective dose concept inherently involves a number of problematic assumptions and issues. Perhaps the most important are:

- The tissue weighting factors represent a subjective balance between the different stochastic endpoints of cancer incidence, cancer mortality, life shortening, and hereditary risk. These weighting factors change every decade or so (ICRP, 1977, 1991; Streffer, 2007). For example, the weighting factor for the gonads dropped from 0.25 in 1977 (Streffer, 2007) to 0.08 in 2007 (Streffer, 2007); and the carcinogenesis endpoint was represented by cancer mortality for the 1990 weighting factors (ICRP, 1991), but by cancer incidence for the 2007 weighting factors (Streffer, 2007). The reasons for such changes are generally less to do with improved knowledge about radiation risks, and more because different groups of experts will naturally have somewhat differing views on the relative importance of the different endpoints that comprise the ‘detriment’ (Streffer, 2007).
- Effective dose is defined to be independent of age at exposure, whereas it is well established that radiation risks are highly age dependent; for example different cancer sites exhibit very different dependencies on age at exposure (e.g., Preston et al., 2007). To assume, as is implied in the effective dose concept, that the age dependencies for all endpoints are the same is clearly problematic.
- Effective dose is often confused with equivalent dose. Both equivalent dose (which refers to a given tissue) and effective dose (which is a weighted average over the entire body) are measured in sieverts. As such, it is no surprise, for example, that the literature on computed tomography (CT) is replete with examples where effective dose and equivalent dose have been confused with one another. This is not a minor matter of semantics; for a typical CT scan, the effective dose is typically about one-third of the maximum equivalent dose.
- Perhaps the most important problem is that effective dose is increasingly being misused. While it was designed to provide comparisons between one radiation field and another, there are increasing numbers of papers in the radiological literature in which effective dose is calculated, and then lifetime cancer risks are estimated from these effective doses.

2. A SOLUTION TO THE PROBLEM

It has often been argued that effective dose is ‘all we’ve got’ to provide an easy-to-use estimate or comparison of the risks associated with low-dose exposures (Martin, 2007; Brenner and Huda, 2008; Dietze et al., 2009). Rather than tolerate this flawed concept, it is possible to define a new, simple, less-confusing, easy-to-estimate

quantity, based on solid science, which estimates and/or compares the risks associated with different inhomogeneous low doses of radiation more directly (Brenner, 2008). Specifically, let us consider modifying the effective dose concept as follows:

- Focus only on cancer risks: there is no logical way that cancer risks and hereditary risks can be combined into a single number, as is attempted in the effective dose definition. Because we are only concerned, in this context, with low doses, it is entirely reasonable to drop hereditary risks altogether, and focus only on cancer risks. A choice can then be made between considering cancer incidence, cancer mortality, and years of life lost, but some meaningless average should not be used. Here, for the sake of discussion, let us choose cancer incidence.
- Make the quantity dependent on age at exposure; this would not be difficult. Table 1 shows the current BEIR-VII (NRC, 2005) estimates of lifetime-attributable, tissue-specific, age-specific cancer incidence risks. ICRP risk weighting factors could be based on such evaluated cancer risk estimates. Arguably, the quantity could also be made gender dependent. Gender does not have as large an effect as age, and there are arguments both ways here: it is simpler to use a single averaged value, but women are significantly more sensitive than men, and the illogicalities of, for example, including the female breast risk for a male population would be avoided.

These considerations suggest a simple strategy to replace effective dose with a similar, but scientifically more defensible quantity in which the tissue weighting factors are none other than evaluated age-specific (and perhaps gender-specific) cancer risks, such as those in Table 1. So instead of summing the product of the equivalent dose to each tissue and the appropriate ICRP tissue weighting factor, one would instead sum the product of the equivalent dose to each tissue and the tissue-specific life-time cancer risks per unit equivalent dose, such as those in Table 1. Clearly this calculation would be no more difficult or complicated than calculating effective dose.

The quantity defined by this approach, termed ‘effective risk’ (Brenner, 2008) is easy to calculate, fulfils exactly the same role as effective dose for comparing the risks associated with different inhomogeneous exposures, but is based on solid science, and avoids all the confusions associated with effective dose vs equivalent dose. Another major advantage is that it would give the users some feel for the actual numerical values of the risks they are trying to control.

In summary, effective dose is currently defined as:

$$E = \sum_T w_T H_T \quad (1)$$

where H_T is the tissue-specific equivalent dose for tissue T , and w_T is the committee-defined dimensionless tissue-specific weighting factor. In an analogous way, effective risk (Brenner, 2008) is defined ¹ as:

¹ An identical formalism to this effective risk formalism (Brenner, 2008) has subsequently been published by Li et al. using the name ‘risk index’ (Li et al., 2011).

Table 1. Current estimates of tissue-, age-, and gender-specific lifetime-attributable cancer incidence risks per unit equivalent dose in a Western population. It is proposed that data similar to these could be used for the quantity r_T in calculating effective risk, R , from Eq. (2).

Organ	Age at exposure (years)										
	0	5	10	15	20	30	40	50	60	70	80
<i>Males</i>											
Stomach	76	65	55	46	40	28	27	25	20	14	3
Colon	336	285	241	204	173	125	122	113	94	65	30
Liver	61	50	43	36	30	22	21	19	14	8	3
Lung	314	261	216	180	149	105	104	101	89	65	34
Prostate	93	80	67	57	48	35	35	33	26	14	5
Bladder	209	177	150	127	108	79	79	76	66	47	23
Thyroid	115	76	50	33	21	9	3	1	0.3	0.1	0.0
Leukaemia	237	149	120	105	96	84	84	84	82	73	48
Other	1123	672	503	394	312	198	172	140	98	57	23
<i>Females</i>											
Stomach	101	85	72	61	52	36	35	32	27	19	11
Colon	220	187	158	134	114	82	79	73	62	45	23
Liver	28	23	20	16	14	10	10	9	7	5	2
Lung	733	608	504	417	346	242	240	230	201	147	77
Breast	1171	914	712	553	429	253	141	70	31	12	4
Uterus	50	42	36	30	26	18	16	13	9	5	2
Ovary	104	87	73	60	50	34	31	25	18	11	5
Bladder	212	180	152	129	109	79	78	74	64	47	24
Thyroid	634	419	275	178	113	41	14	4	1	0.3	0.0
Leukaemia	185	112	86	76	71	63	62	62	57	51	37
Other	1339	719	523	409	323	207	181	148	109	68	30

Adapted from Table 12D-1 of the 2006 BEIR-VII report (NRC, 2005).

$$R = \sum_T r_T H_T, \quad (2)$$

where r_T is the lifetime tissue-specific cancer risk (per unit equivalent dose to tissue T), such as those shown in Table 1. Clearly R is no more difficult to calculate than E , and, as argued here, it is much more defensible scientifically.

As discussed above, it would make more sense to define the effective risk quantity, R , to be age and gender dependent (which, of course, it is). This would be easy to do using the r_T data, such as in Table 1.

3. CONCLUSIONS

There is a need for a quantity that compares or estimates the risks from different inhomogeneous low dose exposures. However, effective dose is confusing and is based on flawed science. For radiation protection, one could perhaps make an argument for the continued use of effective dose, flawed and confusing as it may be. In practice, however, effective dose is nowadays largely used to quantify risks associated with low dose (mainly radiological) exposures, and its use cannot be justified in this

area. Consideration should be given to replacing it with another quantity, ‘effective risk’ (Brenner, 2008), that is just as easy to estimate, does the same job, is less prone to misuse, is more directly understandable, and is based on solid science.

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