

## EFFECTIVE DOSE: A USEFUL CONCEPT IN DIAGNOSTIC RADIOLOGY?

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### INTRODUCTION

Effective dose ( $E$ ) was introduced by the International Commission of Radiological Protection (ICRP) to provide a summation of radiation doses to tissues and organs for radiological protection. For a non-uniform irradiation,  $E$  is designed to provide an estimate of the corresponding uniform whole-body dose that would result in the same stochastic detriment.  $E$ , which has the same units as equivalent dose, is obtained by summing individual organ equivalent doses ( $H_T$ ) multiplied by the corresponding tissue weighting factors.

$$E = \sum_T W_T H_T \text{ with } \sum_T W_T = 1$$

where  $w_T$  are dimensionless tissue weighting factors characterising the relative sensitivity of various tissues with respect to the endpoints, such as cancer induction and mortality. Twelve tissues and organs are specified in ICRP report 60 (see Table 1) with individual weights  $w_T$ , and an additional ‘remainder’ tissue is defined. A revised set of tissue weighting factors is proposed in the ICRP 2006 Draft Recommendations (see Table 2).

Of course diagnostic radiology deals almost exclusively with non-uniform radiation exposures. Is the concept of  $E$  useful in diagnostic radiology? Does it provide a quick but useful estimate of the overall detriment of a non-uniform low dose? Or is it confusing and more trouble than it is worth?

The question raised in the present debate serves to examine the use and possible misuse of the quantity  $E$  in diagnostic radiology. The participants in our debate have been involved in the applications of radiation dosimetry for many years, and they will argue for and against our proposition.

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## WALTER HUDA

### FAVOURING THE PROPOSITION:

#### Argument

Diagnostic medical examinations that use ionising radiation generally produce a 3-D pattern of energy deposition within the patient. It is important to note that in virtually all of these procedures, individual organ doses will be well below the threshold dose of  $\sim 2$  Gy for the induction of deterministic effects such as erythema and epilation<sup>(1)</sup>. Accordingly, any patient radiation risks relate to the stochastic processes of carcinogenesis and the induction of genetic effects. Use of the effective dose in diagnostic radiology permits the radiation dose of diverse diagnostic procedures to be quantified using a common measure. Since the effective dose ( $E$ ) is directly related to the stochastic risk,  $E$  for a given diagnostic procedure also permits determination of relative and (approximate) absolute estimates of the examination risk.

**Table 1.** Taken from ICRP Recommendations for tissue weighting factors,  $w_T$ , in Publication 60 (1991)<sup>(1)</sup>.

Organ/tissue	$w_T$	$\sum_T w_T$
Gonads	0.20	0.20
Lung, Stomach, Colon, Bone marrow	0.12	0.48
Breast, Thyroid, Oesophagus, Bladder, Liver, Remainder	0.05	0.30
Bone surface, Skin	0.01	0.02
Total		1.0

**Table 2.** Taken from values of tissue weighting factors,  $w_T$ , proposed in ICRP Draft 2006<sup>(2)</sup>.

Organ/tissue	$w_T$	$\sum_T w_T$
Lung, Stomach, Colon, Bone marrow, Breast, Remainder	0.12	0.72
Gonads	0.08	0.08
Thyroid, Oesophagus, Bladder, Liver	0.04	0.16
Bone surface, Skin, Brain, Salivary glands	0.01	0.04
TOTAL		1.0

Diagnostic examinations that are increasingly being performed using digital systems offer operators a choice of radiographic technique (kV and mA s) and X-ray beam filters. Changing the way a radiographic examination is performed impacts on the patient dose as well as the corresponding image quality. Optimisation of diagnostic imaging is an important topic that involves finding the technique that offers the lowest patient dose when image quality (i.e. diagnostic performance) is kept constant.  $E$  is an ideal parameter for use in optimisation efforts. A plot of  $E$  as a function of X-ray tube voltage (kV) at constant image quality, for example, will permit the kilovolt that minimises  $E$  to be identified<sup>(2)</sup>. For a given diagnostic task, this kilovolt value will be optimal provided deterministic effects can be neglected.

Patients undergo a range of diagnostic procedures that employ X-rays or radionuclides, and it is useful to know how much radiation a patient may receive from different procedures<sup>(3, 4)</sup>. Knowledge of patient doses improves our understanding of these diagnostic tests; for example, knowing that a typical chest CT examination ( $E \sim 5$  mSv) is  $\sim 100$  times larger than a two view chest X-ray examination ( $E \sim 0.05$  mSv) alerts practitioners to the high doses associated with CT<sup>(5)</sup>. Doses from an X-ray exam can also be compared with those in nuclear medicine (NM), and may help identify which test has a lower patient dose (risk). For example, chest CT and radionuclide studies (i.e. ventilation/perfusion) may both be used to rule out a pulmonary embolism in a given patient. Knowledge of the corresponding  $E$ s that depend on the specific protocols used in a given department will quantify the relative radiation risks and help identify which test is the most appropriate for any clinical problem within a given population.

Operators, patients and other involved individuals often seek to better understand the amount of radiation that a patient receives in a given diagnostic test. NM examinations typically deliver  $E$ s of  $\sim 5$  mSv, which should be understandable to X-ray practitioners who work in medical imaging, but will likely mean little to referring physicians or to

patients and their families.  $E$  from a NM study can be compared with a range of benchmark  $E$ s including natural background radiation (cosmic, terrestrial and internal) of  $1 \text{ mSv y}^{-1}$ , and the average radon exposure in the USA of  $2 \text{ mSv y}^{-1}$ <sup>(6)</sup>.  $E$  in diagnostic tests may also be compared with regulatory  $E$  limits for occupational exposure ( $50 \text{ mSv y}^{-1}$ ) as well as those for members of the public ( $1 \text{ mSv y}^{-1}$ ). The current US regulatory dose limit to the fetus of an occupational radiation worker is  $0.5 \text{ mSv month}^{-1}$ , or no more than  $5 \text{ mSv}$  total embryo/fetal dose after the declaration of any such pregnancy. Comparing natural background and regulatory  $E$ s with  $E$ s from diagnostic tests helps put medical exposures into perspective.

It is possible to attempt to convert  $E$ s into an approximate radiation risk to a reference patient. The ICRP currently use a risk of fatal cancer of 5% per Sv when averaged over a whole population, but it is important to recognise that these risk estimates are only very approximate, and sex/demographic factors may need to be taken into account for specific populations<sup>(7, 8)</sup>. For example, a reference child would be expected to have much higher risks per unit  $E$  than the nominal ICRP value, whereas the risks for reference retirees would be considerably lower. Radiation risks in diagnostic radiology may be compared with other medical hazards such as fatal and non-fatal adverse effects following the administration of iodinated contrast agents. Quantitative radiation risks may also be compared with other risks individuals may encounter in every day life. An informed consent statement for subjects participating in research projects could show that an  $E$  of  $1 \text{ mSv}$  has fatality risk that is comparable to smoking  $\sim 20$  packs of cigarettes or dying in an automobile accident when driving  $\sim 1000$  miles<sup>(9)</sup>.

It is, however, important to recognise that there are situations where the  $E$  would not be an appropriate indicator to the dose (risk) that a patient receives. For example, quantification of the 'risks' associated with virtual colonoscopy requires detailed information about organ doses from this examination, as well as demographic specific organ risk estimates to quantify the detriment that needs to be balanced

**Table 1. Typical effective doses in diagnostic radiology.**

Imaging modality	Examination type	Representative effective dose (mSv)
Radiography	Skull	~0.1
	Chest	~0.05
	Abdomen	~0.4
Fluoroscopy	Barium swallow	~1
	Barium meal	~3
	Barium enema	~5
CT	Head	~1
	Chest	~5
	Cardiac	~10
Nuclear medicine	Abdomen/Pelvis	~5
	Planar imaging/SPECT	~5
	Positron Emission Tomography	~10

**Table 2. Typical values of annual effective dose encountered in the USA.**

Source of exposure	Annual effective dose (mSv)	Comments
Natural background	~ 1	Terrestrial + cosmic + internal
Radon (daughters)	~ 2	Individual doses very variable
Radiation worker limit	20	ICRP and NCRP
Highest worker doses	~5	Typical effective dose to IR fellow
Public dose limits	1	Excludes background and X-rays
Radiation worker conceptus	5	No more than 0.5/month

against any anticipated benefit.  $E$ , which is computed assuming a reference population, would be inappropriate for use in this type of cost-benefit analysis.

In conclusion, it is important to recognise that the most uses of the  $E$  in radiology do not require the explicit use of any quantitative radiation risk estimates, since direct comparisons are made between two exposures for the same individual. Converting  $E$ s to radiation risk and detriment estimates is possible, but must be performed with care. In 25 y of work in a clinical radiology environment, I personally have found that using  $E$  to quantify radiation dose from X-ray studies as the most effective way to communicate about X-ray doses within the radiology community, as well as with other medical practitioners and members of the public.

### Rebuttal

I disagree that  $E$  is ‘...designed to provide a measure of the “stochastic detriment” of a radiation exposure’. The principal benefit of using  $E$ s in diagnostic radiology is to quantitatively compare different types of non-uniform exposures. This can be illustrated by the data in Table 1 that show typical  $E$ s in diagnostic radiology, and the data in Table 2 that show benchmark values of  $E$ . Data presented in Tables 1 and 2 quantify patient doses in a simple

and understandable way, and for this reason are beneficial to the medical imaging community.

I also disagree that  $E$ s cannot be used to provide quantitative radiation risks. Consider a young adult undergoing a chest CT examination that results in the patient receiving an  $E$  of 5 mSv (Table 1). The fatal cancer risk averaged over a typical population, as recommended by the ICRP, is 5% per Sv. This population average risk value will be close to the value for young adults, so this patient’s fatal cancer risk is ~2.5 per 10 000. The genetic risk will be negligible, since the gonads are not irradiated in chest CT scan, and non-fatal cancer risks are expected to be comparable to fatal cancer risks. If the patient was an infant, the fatal cancer risk would be about three times higher, whereas for a retiree the corresponding risk might be a factor of three times lower. This example shows that the  $E$ s can be converted to (approximate) risks when required, with no confusion between organ dose (mGy), organ equivalent dose (mSv) and (whole-body)  $E$  (mSv).

I am not enthused with the suggested new quantity of effective risk in diagnostic radiology. There are non-trivial problems of obtaining quantitative risks to all exposed organs, particularly the category describes as ‘other’. Quoting radiation risk of fatal cancer does not address other radiation risks (non-fatal cancer and genetic risks), and also fails to address the large uncertainties in risk at the doses

normally encountered in diagnostic radiology. Any differences in the numerical value of Dr Brenner's suggested quantity 'effective risk', and the corresponding risk estimate obtained directly from the  $E$  (see above), would likely be small in comparison with uncertainties in organ doses and their corresponding risk factors. Replacing patient  $E$ s with numerical effective risks would do a little to enlighten the medical imaging community (radiologists, technologists and physicists), as well referring physicians and their patients, regarding patient doses in diagnostic radiology.  $E$  conveniently quantifies the radiation received by patient's undergoing diagnostic examinations, and I wholeheartedly support its continued use for this purpose by medical imaging community.

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## David Brenner

### OPPOSING THE PROPOSITION:

#### Argument

The effective dose is designed to provide a measure of the 'stochastic detriment' of a radiation exposure. As defined, it represents an attempt to provide a

single number that characterises cancer incidence, cancer mortality, life shortening, as well as hereditary risk. But each of these endpoints generally corresponds to different tissue weighting factors. Any weighted average of these endpoints is necessarily both arbitrary and subjective. Should a fatal cancer represent twice the stochastic detriment of a non-fatal cancer? Ten times the detriment? How should a birth defect be weighted relative to a cancer? The numerical values of the tissue weighting factors depend entirely on subjective decisions such as these, so it is no wonder that the ICRP weighting factors keep changing.

Just one example: the weighting factor for the gonads has dropped from 0.2 in 1991 to 0.08 in 2007, a drastic decrease in the relative significance of hereditary effects. One could argue that the tissue weighting factors should be changed periodically to reflect increased knowledge of radiation risks, but in fact we do not know much more about the relative importance of somatic vs. hereditary effects than we did in 1991. Rather, a different committee has made a different judgment based largely on the same data. The bottom line is that there is no logical way to combine all types of stochastic detriments within a single quantity.

The second (but fixable) major problem with the effective dose is that it is defined to be independent of age, whereas in fact the relative radiosensitivity of different organs changes greatly with age. To take, for example, the breast and the lung, two of the most important radiosensitive organs: for the endpoint of lifetime cancer mortality, the latest BEIR report<sup>(1)</sup> has the lung about twice as sensitive as the breast for a neonate, and has the lung ~20 times more sensitive than the breast for a 60-y-old woman. Yet within the framework of the effective dose concept, the relative sensitivity of the lung vs. the breast is described by a single age-independent value.

So the effective dose is based on flawed science. But leaving this aside for a moment, does it do its job of providing a simple easy to understand framework for comparing the risks of one inhomogeneous dose distribution with another? I would argue that it does not, but rather it has led to a great deal of confusion. An example: This author recently gave a Grand Rounds in the radiology department of a major hospital on the topic of CT-based screening. At one point, the potential cancer risks associated with CT-based lung screening were being discussed, specifically for a 4.5 mSv equivalent dose to the lung, associated with a 60 mA s setting on a particular scanner. A polite arm went up in the audience stating that she had a published paper in her hand showing that 60 mA s using that scanner corresponded to 1.5 mSv, not 4.5 mSv, so that all the risk estimates should be three times lower. It turned out

that the paper was quoting effective dose, rather than equivalent dose to the lung (though it was described in the paper merely as 'dose'). Subsequent perusal of 20 papers in the peer-reviewed literature in which CT risks were mentioned, revealed that eight confused the distinctions between organ dose, effective dose and equivalent dose. Of course one could argue that better education is the answer here, not a change in the effective dose concept. But in that the effective and the equivalent doses have the same units, they are always likely to be confused with each other.

What is to be done? Can we come up with a simple, less confusing, easy-to-estimate quantity, based on solid science, which does the job of comparing the risks associated with different inhomogeneous doses? I think we can. Let us consider modifying the effective dose as follows:

- (1) Separate out cancer risks from hereditary risks. There is no way they can be logically combined. Given what we now know, we might perhaps drop hereditary risks altogether or, if that is not acceptable, provide two different numbers, one related to somatic risks and one to hereditary risks. Then make a choice between considering cancer incidence and cancer mortality, but do not use some meaningless average of the two. Let us say, for the sake of discussion, we choose cancer mortality.
- (2) Make the quantity age dependent. This is not hard to do, Table 1 shows the current BEIR-VII<sup>(1)</sup> estimates of lifetime attributable organ- and age-specific cancer mortality risks. We could just replace the ICRP weighting factors with something based on these. Arguably, we could also make the quantity gender dependent. Gender is not as big 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.

But given that we have systematic organ- and age-specific cancer risk estimates available, such as those in the BEIR-VII Report (Table 1), why not do away with effective dose altogether, and instead simply calculate the lifetime cancer risk directly from this Table? It would be no more difficult or complicated than calculating effective dose: instead of summing (over different organs) the product of the organ dose and the tissue weighting factor, we would sum (over different organs) the product of the organ dose and the organ-specific lifetime cancer risk-per-unit-dose given in this Table.

The single resulting quantity, perhaps one could call it the 'Effective Risk', would be easy to

calculate, would fulfil exactly the same function as the effective dose, i.e. comparing stochastic risks associated with different inhomogeneous exposures, but it would be based on solid science, and it would do away with all the confusions associated with effective dose vs. equivalent dose. And, perhaps its major advantage is that it would give the users some feel for the actual numerical values of the risks that they are trying to control.

The bottom line is that there is indeed a need for a quantity which simply compares the risks from different inhomogeneous dose distributions. But the effective dose is confusing and is based on flawed science. Let us consider replacing it with a quantity that is just as easy to estimate, an effective risk, that does the same job, is less prone to misuse, is more directly understandable, and is based on solid science.

### Rebuttal

Dr Huda and I clearly have the same goal, We both want an easily calculable quantity that gives the user a quick measure of the whole-body stochastic risk.

I think we can and should directly estimate the whole-body stochastic risk, and it seems that Dr Huda really wants to do this too, to judge from his comment 'Knowledge of the corresponding effective doses. . . will quantify the relative radiation risks and help identify which test is most appropriate for any clinical problem. . .'.

How could it be otherwise? No one is interested in quantifying a dose for its own sake, only in as much as it is an indicator of risk. If, as argued here, it is just as easy (and scientifically more defensible) to calculate a whole-body stochastic risk, as opposed to an effective dose, then surely that is what we should be doing.

Specifically, Dr Huda wants to estimate the effective dose:

$$E = \sum_T w_T H_T$$

where  $H_T$  are the tissue-specific equivalent doses, and  $w_T$  are committee-defined dimensionless tissue-specific weighting factors; then Dr Huda has the afterthought that 'it is possible to attempt to convert effective doses into an approximate radiation risk to a reference patient'. But why use this scientifically questionable intermediate, effective dose, when one can estimate a whole-body cancer risk directly:

$$R = \sum_T r_T H_T,$$

where  $R$  is the whole-body 'effective risk', and  $r_T$  are the lifetime attributable tissue-specific cancer risks (per unit organ dose) given in Table 1 (above).

**Table 1.** Taken from Table 12 D-2 of BEIR-VII report<sup>1</sup>, giving current best estimates of the organ-, age- and gender-specific lifetime attributable cancer mortality risks per unit organ dose (number of deaths per 10<sup>6</sup> persons exposed to 10 mGy).

Cancer Site	Age at exposure (y)										
	0	5	10	15	20	30	40	50	60	70	80
<b>Males</b>											
Stomach	41	34	30	25	21	16	15	13	11	8	4
Colon	163	139	117	99	84	61	60	57	49	36	21
Liver	44	37	31	27	23	16	16	14	12	8	4
Lung	318	264	219	182	151	107	107	104	93	71	42
Prostate	17	15	12	10	9	7	6	7	7	7	5
Bladder	45	38	32	27	23	17	17	17	17	15	10
Other	400	255	200	162	134	94	88	77	58	36	17
All solid	1028	781	641	533	444	317	310	289	246	181	102
Leukaemia	71	71	71	70	67	64	67	71	73	69	51
All cancers	1099	852	712	603	511	381	377	360	319	250	153
<b>Females</b>											
Stomach	57	48	41	34	29	21	20	19	16	13	8
Colon	102	86	73	62	53	38	37	35	31	25	15
Liver	24	20	17	14	12	9	8	8	7	5	3
Lung	643	534	442	367	305	213	212	204	183	140	81
Breast	274	214	167	130	101	61	35	19	9	5	2
Uterus	11	10	8	7	6	4	4	3	3	2	1
Ovary	55	47	39	34	28	20	20	18	15	10	5
Bladder	59	51	43	36	31	23	23	22	22	19	13
Other	491	287	220	179	147	103	97	86	69	47	24
All solid	1717	1295	1051	862	711	491	455	415	354	265	152
Leukaemia	53	52	53	52	51	51	52	54	55	52	38
All cancers	1770	1347	1104	914	762	542	507	469	409	317	190

Clearly  $R$  is no harder to calculate than  $E$ , and, as argued here, it is scientifically much more defensible.

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 actually is), and which would be easy to do using the  $r_T$  data in Table 1—but this is a detail.

The effective dose concept reflects weak science, and provides an answer which does not, in itself, mean anything. In contrast, a whole-body effective risk ( $R$ ) directly reflects the best epidemiology, we have at our disposal, and provides an answer which can be easily understood and interpreted by scientists and the general public alike. Are we afraid of the possibility that the public could actually understand what we do?

## REFERENCE

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## SUMMARY

Our debaters have examined the nature of effective dose from two viewpoints, and they have provided us with insightful observations and suggestions to improve this protection quantity. The 2007 recommendations of the ICRP will likely be available by the time this debate is published. Therefore, it will be interesting to see if our debaters have foretold possible changes in the ICRP recommendations. The protection quantities, equivalent and effective doses embody complex concepts, and the condition of non-uniform exposure represents a particularly complex situation. It is hoped that this debate will have provided the reader with some useful information and opinions regarding an important topic in medical radiation protection, and radiation protection in general.