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THE USE OF TOTAL DETRIMENT IN RADIATION
PROTECTION AND ITS POTENTIAL EXTENSION
TO OTHER HAZARDS

J. R. Johnson   J. M. Selby
P. S. Stansbury

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Pacific Northwest Laboratory
Richland, Washington 99352

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Its Potential Extension to Other Hazards

J. R. Johnson, J. M. Selby, and P. S. Stansbury

Pacific Northwest Laboratory
Richland, Washington

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Abstract

Before publication of the 1977 recommendations of the International Commission on Radiological Protection (ICRP), radiation protection standards were based on dose limits to single organs. These dose limits were only loosely linked to the expected genetic effects in the first two generations from gonadal doses and to the risk of fatal cancer from doses to specific organs. In 1977, the ICRP recommended the use of the "effective dose equivalent (EDE)," which is a method of summing the doses (weighted with relative risk coefficients) to all organs and tissues, and recommended an annual limit for EDE. The EDE annual limit was based on the total fatal cancer risk to an individual, combined with risk of the total serious deleterious genetic effects in the first two generations of offspring.

Since the 1977 recommendations were published, a "total risk" or total detriment approach has been extended to include nonfatal cancers and genetic effects for all subsequent generations, i.e., the total health detriment from low doses of ionizing radiation. This paper discusses the development of this total health detriment from ionizing radiation exposures, and explores potential methods for using it with other hazards (such as exposures to other physical agents, hazardous chemicals, and fatal and nonfatal accidents) in calculating the total detriment to a worker.

Introduction

It is recognized that there are a large number of agents in the workplace, at home, and in the environment that can cause adverse health effects in humans. Table I gives an illustrative but not comprehensive list of such agents and undesirable health effects associated with one or more of the agents, depending on the nature and duration of exposure.

Exposure to harmful agents or agents suspected of being harmful is usually regulated and controlled on a case-by-case basis. Often the degree of control and expense associated with limiting exposures to different hazards is not consistent with the potential health effects of the different hazards involved. For example, in some industries much more effort is applied to control worker exposure to ionizing radiation than to chemical carcinogens; conversely, often more effort is applied to the control of hazardous chemicals in the environment.

This paper reviews the development of the "total detriment" concept by the International Commission on Radiological Protection (ICRP) for the purpose of controlling exposure to ionizing radiation. This concept could be extended to include assessment and the comparison of the potential health effects of exposures to all hazards. As such, total detriment could be used as a rational method to compare the relative safety of different practices, technologies and industries, to analyze

the changes in safety and health over time, and to optimize the use of limited resources to improve safety and health of an industry or among all industries.

The concept of total detriment is often used to assess all effects caused by a practice, technology or industry. However, as used in this paper total detriment is restricted to the quantification of total "harm" to an individual from all exposures to hazards at work. For total detriment to be useful, one must be able to quantify exposures (via measurement or estimation) to all hazards in the workplace, and be able to convert these quantified exposures to common units of "harm" (i.e., an "index of harm" must be defined). This paper will outline the ICRPs development of such an index and will suggest how the concept of total detriment could be extended to hazards other than ionizing radiation.

Early Developments

Almost concurrently with the discovery of radiation it was noted that exposures caused undesirable biological effects and that such effects were somewhat proportional to the absorbed dose (energy density) in biological tissues. As more information on these effects accumulated, it was noted that different types of radiations (e.g., gamma rays, neutrons, and alpha particles) caused the same biological effect at different levels of absorbed dose and this led to the development of

the dose equivalent concept (essentially the first index of harm for radiation exposures). This development allowed the summation of absorbed doses to an organ from radiations with different "radiation quality" by multiplying absorbed dose by a "Quality Factor"^(1,2) to get an organ dose equivalent.

Formal recognition by the ICRP of the need to limit the risk from radiation exposures when more than one organ received a significant dose equivalent came with ICRP Publications 1⁽¹⁾ and 6⁽²⁾ and was discussed in more detail in 1966 with the revision of their recommendations.⁽³⁾

While ICRP Publications 1, 6, and 9 recognized the need to limit the exposure when more than one organ received a significant dose equivalent, it wasn't until 1974 that a method of summing doses to organs was proposed. Jacobi presented a system⁽⁴⁾ for summing doses at a Commission of the European Communities Symposium that was essentially adopted in 1977 by the ICRP in its Publication 26.⁽⁵⁾ This sum of the weighted dose equivalents to different organs, later called "effective dose equivalent (EDE)," was defined by:

$$H_E = \sum_t W_t H_t \quad (1)$$

$$\sum_t W_t = 1$$

where H_E is the effective dose equivalent, H_t is the dose equivalent to organs or tissues t and W_t is the weighting factor for each organ or

tissue.⁽⁵⁾ The weighting factors recommended in ICRP Publication 26 were only appropriate for use at dose levels below that which cause gross tissue damage and "deterministic" effects. Or stated differently, the weighting factors could be used only when the risk was thought to be linearly proportional to the dose equivalent with a zero intercept.

The weighting factor W_t for the gonads (both male and female, chosen to be 0.25), represented the relative risk of hereditary effects in the first two generations following exposure of the parents, and the weighting factors W_t for the other tissues (sum = 0.75) were selected to reflect the risks of developing radiation induced fatal cancer per unit dose equivalent in those tissues. Although the weighting factors selections were somewhat arbitrary, the effective dose equivalent provided a common index of harm for comparing and limiting all doses (from both external and internal sources) of ionizing radiation.

Development of Measures of Total Detriment

The ICRP recognized that fatal cancer and genetic effects did not account for the total detriment from exposures to ionizing radiation and wanted to be able to compare the total harm (detriment) caused by ionizing radiation to that from all other workplace hazards. As a result, Publication 27 -- "Problems Involved in Developing an Index of Harm" was published in 1977.⁽⁶⁾ The rate and age distribution of occupational fatalities and the frequency of injuries and their severity

in terms of lost days of work were examined in the report. In addition, occupational diseases were compared to accidents in terms of lost days of life and lost days at work. It was noted in that report that in most industries the effects of accidents far outweigh those of occupational diseases by either of these measures of harm. However, this publication did not provide any definitive measure for comparison of different hazards; instead it was intended "to help formulate rather than answer questions."⁽⁶⁾

The ICRP continued to address the question of a unified index of harm and published "Quantitative Basis for Developing a Unified Index of Harm" in 1985⁽⁷⁾. In that report, the work in Publication 27 was extended to quantitatively address accident rates, radiation induced non-fatal cancers, deterministic effects such as radiation induced cataract formation and hereditary detriment in terms of lost years of life, or the equivalent, in terms of years of impaired health.

ICRP Publication 60

The ICRP published a major review of its recommendations in 1991.⁽⁸⁾ The EDE concept introduced in Publication 26 was extended to include non-fatal cancer and estimates of hereditary effects for all subsequent generations, and the weighting factors for Equation (1) were explicitly derived from the index of harm proposed in 1985.⁽⁷⁾ Also in Publication 60 the ICRP simplified the EDE nomenclature, by designating "equivalent

dose" as the average dose to an organ weighted for radiation quality summed over all types of radiation. The sum of the products of all equivalent doses to organs with the Equation (1) weighting factors was defined as the "effective dose."

In Publication 60 the effective dose weighting factors are derived by accounting for non-fatal cancers and genetic effects along with fatal cancers on the basis of equivalent years of life lost. The ICRP expresses total detriment as:

$$\text{Total Detriment} = F \cdot \ell / \bar{\ell} \cdot (2-k) \quad (2)$$

where F is the number of fatal cancers per unit of equivalent dose to organs, $\ell / \bar{\ell}$ is the relative length of life lost expected per fatal cancer and $2-k$ is the factor used to account for non-fatal cancers.

The factor ℓ in Equation (2) is the expected life lost (in years) for fatal cancers of various types. Among the cancer sites the ICRP considered the factor ranges from a low of 10 years for bladder cancers to a high of 31 years for bone marrow cancers. Thus the product of F and ℓ in Equation (2) is the expected years of life lost per unit radiation exposure, assuming that the radiation exposure is uniform in time over a working life. The average expected loss of life from all cancer sites considered is 15.0 years, is defined as $\bar{\ell}$, and is used to

remove the unwanted dimension, years, from Equation (2). Thus $\ell/\bar{\ell}$ is the relative expected life lost per fatal cancer.

The 2-k factor for non-fatal cancer is derived as follows: k is the fraction of cancers of a particular type which is fatal i.e., the lethality fraction. The total number of cancers is then F/k and the number of non-fatal cancers is $(1-k) F/k$. The ICRP uses a weighting factor for non-fatal cancers of k. Although the selection of this weighting factor is arbitrary, it has the right qualitative features. Cancers which are hard to cure and hence have a low cure rate, such as lung cancer, have a large impact on the quality of life, whereas cancers which are easy to cure, such as most skin cancers and thyroid cancer, will have a smaller impact on the quality of life. Thus the total weighted cancers are:

$$\text{Total "weighted" cancers} = F + k(1-k)F/k \quad (3)$$

or

$$= F(2-k)$$

The ICRP's recommended values for the factors F, $\ell/\bar{\ell}$, and 2-k, for radiogenic cancers are given in Table II. Genetic effects are accounted for in the same table as follows: the F factor is chosen to be a hundred to reflect that 100 severe genetic disorders in all future generations per 10^4 Sieverts (Sv) of equivalent dose that would be expected from irradiation of a population of parents of both sexes at a

uniform rate over their working lifetime. The equivalent years of life lost for each severe genetic disorder was taken to be 20 years (i.e., $\ell/\bar{\ell} = 1.33$) reflecting both early death caused by some disorders and the lifelong deleterious effects of others.

The products of the factors, summarized in Table II, implied much more precision than is warranted and therefore the ICRP simplified the results of these calculations with the new factors given in Table III. The sum of these factors equals unity, and the effective dose calculated using these weighting factors represents a new index of harm.

Extension of the Total Detriment Concept

In general, what is needed to extend the ICRP's definition of total detriment to address other hazards is a formalism to convert the potential harm of exposure to expected life lost. For carcinogenic chemicals the following equation would suffice:

$$\text{harm} = (\text{exposure}) \times (\text{risk coefficient}) \times (\text{severity factor}) \quad (4)$$

provided that the exposures could be measured or adequately estimated, that the risk coefficient (i.e., the risk of fatal cancer per unit exposure) was known and was linear over the range of exposures measured or, alternatively the variation of risk coefficient with exposure rate was quantifiable, and that a severity factor accounting for years of

life lost from fatal cancers and a loss-of-life equivalent for non-fatal effects could be derived. To be completely parallel with the ICRP concept of total detriment the severity factor in Equation (4) would have to be expressed in units of years of life lost and divided by an $\bar{e} = 15.0$ years.

For extending the ICRP's total detriment concept to chemicals like lead, deriving the factors in Equation (4) would be more difficult. Lead can cause birth defects and mental retardation in the offspring of exposed parents and the ICRP methodology could be directly adapted to address these effects. To account for the effects of lead on those occupationally exposed, a new system of risk coefficients would have to be developed since lead is known to cause mental illness in a manner that is not stochastic. Perhaps the risk coefficient for lead exposure could be three tiered: 1) governed by reproductive effects below levels known to degrade to the mental health of those occupationally exposed over a lifetime, 2) heavily weighted for annual exposures which would lead to an unacceptably high accumulation of lead in a fraction of the worker's career (say 10 years), 3) weighted intermediately for levels between those in 1) and 3) above. The justification for an intermediate level would be based on the presumption that the effects are not stochastic but individual susceptibility and other factors such as true exposure verse measured exposure are. Clearly much work remains to be done to develop appropriate risk coefficients and severity factors for chemicals and agents with severe, non-carcinogenic effects.

It is presumed that modern industrialized societies do not permit routine, planned exposure to agents which cause known deterministic effects. Never the less there are unplanned events such as exposure to strong acids, ocular exposure to lasers, and other industrial accidents. On an actuarial basis mortality and morbidity can be derived per person-year at risk. Thus deterministic effects could be accounted for with:

$$\text{harm} = \frac{(\text{person-years})}{\text{at risk}} \times (\text{risk coefficient}) \times (\text{severity factor})(5)$$

If the severity factor for Equation (5) could be appropriately derived, normalizing to the equivalent of 15 years lost life expectancy, the harm from Equation (5) could be included in the ICRP's total detriment.

Conclusion

The ICRP method of total detriment could be expanded to assess and limit risk comprehensively among a wide variety of industries. Agents known to cause cancer could be assessed using the ICRP system directly. However better methods of quantifying exposures and predicting the expected health effects of such exposures are needed. Toxic effects of particular agents on specific organs could also be addressed but better quantification of the relationship of the agents and their effects are needed. Furthermore in these and other cases such as non-fatal

accidents, the nature of the effects must be related to harm in terms of equivalent lost life expectancy.

Table I. Some Potentially Harmful Agents and Typical Hazards Associated with Exposure to Harmful Agents

Agents	Hazards
Physical	- Fatal and non-fatal accidents
- Ionizing Radiation	- Fatal and non-fatal cancers
- Non-ionizing radiation	- Genetic effects
- Noise	- Diseases
- Vibrations	- Organ specific effects
- Heat	(kidneys, liver, etc.)
- Mechanical energy	- Reproductive effects
Chemical	- Mental disorders
- Heavy Metals	
- Acids and bases	
- Volatile organic compounds	
- Natural carcinogens	
- Cigarette smoke	
Biological	
- Viruses	
- Bacteria	

Table II. Values Proposed by ICRP⁽⁸⁾ for the Calculation of Total Detriment (see Equation 2)

	F^1	$e/\bar{e}$	2-k	Product
Bladder	30	0.65	1.50	29.4
Bone marrow	50	2.06	1.01	104.0
Bone Surface	5	1.00	1.29	6.5
Breast	20	1.21	1.50	36.4
Colon	85	0.83	1.45	102.7
Esophagus	30	0.77	1.05	24.2
Liver	15	1.00	1.05	15.8
Lung	85	0.90	1.05	80.3
Ovary	10	1.12	1.30	14.6
Skin	2	1.00	2.00	4.0
Stomach	110	0.83	1.10	100.0
Thyroid	8	1.00	1.90	15.2
Remainder	50	0.91	1.29	58.9
Gonads ¹	100 ²	1.33 ³	1.0	133.3

Adapted from Table B-20 of Reference 8

¹ F is the probability of fatal cancer per 10⁴ Sv equivalent dose to the indicated organ, except for the gonads.

² Expressed as 10⁴ Sv equivalent dose per effective dose rate to the gonads for subsequent generations for a lifetime

³ Assumed that if a severe genetic effect occurs it is equivalent to 20 years loss of life

Table III. Weighting Factors Recommended by the ICRP in Publication 60⁽⁸⁾

Organ	W_T	ΣW_T
Bone surface, skin	0.01	0.02
Bladder, breast, liver, esophagus, thyroid, remainder	0.05	0.30
Bone marrow, colon, lung, stomach	0.12	0.48
Gonads	0.20	<u>0.20</u>
Total		1.00

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