Late (Delayed) Effects of Radiation

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General

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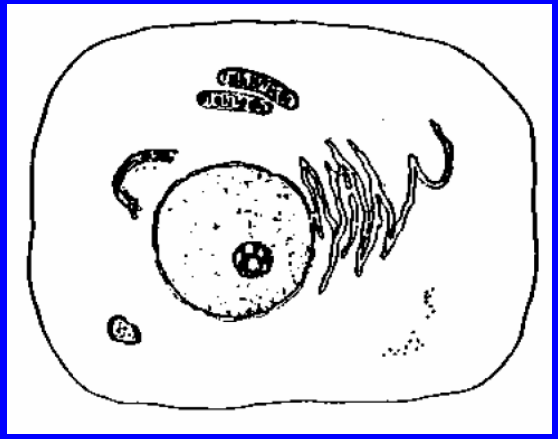
Late Effects (Delayed Effects)

Delayed effects are usually considered to be those effects that appear more than 2 months (in many cases, years) after the exposure.

Depending upon the effect, they can be produced by acute or chronic exposures.

One type of delayed effect is considered possible even with the smallest of exposures. The other types only occurs if the dose exceeded a threshold value



Radiation damage to the cell goes unrepaired. The most important target for such damage is believed to be chromosomal DNA.

Cell Death

Mainly due to DNA double strand breaks and resulting chromosomal aberrations. Cells might survive but are nonfunctional (fibrotic).

Early Effect

Death of cells with short cell cycle. The more rapidly the cells divide, the shorter the latent period.

Late Effect Deterministic

Death of slowly dividing cells. Cells survive longer since a longer time passes before cell divides. This creates a long latent period.

Cell Lives

Damage insufficient to kill cell, but cell metabolism impaired. Cell divides and produces many malfunctioning cells.

Late Effect Stochastic

General

Types of Late Effects (Delayed Effects)

Late effects are classified as: Somatic

Hereditable (genetic)

Fetal (*in utero*)

They can also be classified as: Stochastic

Deterministic

General

Types of Late Effects (Delayed Effects)

Somatic Effects

These occur in the exposed individual.

Hereditable (genetic) Effects

These occur in the offspring of the exposed individual.

Fetal (*in utero*) Effects

These effects occur in those who were exposed *in utero*

General

Types of Late Effects (Delayed Effects)

Somatic Effects might be: Stochastic

Deterministic

Hereditable (genetic) Effects are: Stochastic only

Fetal (*in utero*) Effects might be: Stochastic

Deterministic

General

Important References

BEIR VII. Health Risks from Low Levels of Ionizing Radiation (2005) .

ICRP Publication 103. The 2007 Recommendations of the International Commission on Radiological Protection. 2007.

NCRP No.126. Uncertainties in fatal Cancer Risk Estimates used in Radiation Protection. 1997.

NCRP No. 136. Evaluation of the Linear-Nonthreshold Dose-Response Model for Ionizing Radiation. 2001.

General

Important References

UNSCEAR 2001 Report. Hereditary effects radiation.

UNSCEAR 2006 Report Volume I. Effects of Ionizing Radiation. Annexes A and B.

UNSCEAR 2006 Report Volume II. Effects of Ionizing Radiation. Annexes C,D and E.

Stochastic Effects

Stochastic Effects

General

The only stochastic somatic effect is cancer.

Stochastic effects are those whose probability of occurrence increases with dose. We cannot say that a stochastic effect will definitely occur or not occur, we can only give the probability of such an effect occurring.

The greater the dose, the greater the number of exposed cells and the greater the chance one of them will survive and ultimately result in cancer. This is why the probability of these effects is related to dose.

Stochastic Effects

General

For the purpose of radiation protection, stochastic effects are assumed to be possible at the smallest of doses.

In other words, stochastic effects are not assumed to have thresholds, i.e. doses below which the effects will not occur.

Whether or not this assumption is valid is not known.

The severity of a stochastic effect is not increased by the dose. The severity of a stochastic effect (i.e. cancer) only depends on its location and type, something that is independent of the dose.

Stochastic Effects

General

Since the exposed cell must survive for these effects to occur, the probability of stochastic effects decreases above the dose at which cell killing becomes significant. This dose is often on the order of 300 to 400 rads. At such high doses, acute effects are a more immediate concern than cancer.

The U.S. regulatory agencies limit the possibility of stochastic effects for workers by restricting the annual total effective dose equivalent to 5 rem (0.05 Sv).

The ICRP recommendation is to limit exposures to 0.1 Sv (10 rems) over five years which more or less equates to 2 rem per year.

Stochastic Effects

The Nature of Cancer

Cancer is a non-contagious disease involving malignant tissues of potentially unlimited growth. These tissues usually form discrete masses called tumors. In the case of leukemia, a special type of cancer, no tumors are formed.

Approximately one in five will die of cancer. Roughly twice that many develop cancer.

The older someone is, the more likely they are to die of cancer.

Not all types of cancer appear to be induced by radiation. When a cancer has been induced by radiation, there is nothing unique about it that identifies radiation as the cause.

Stochastic Effects

The Nature of Cancer

Cancer is a non-contagious disease involving malignant tissues of potentially unlimited growth. These tissues usually form discrete masses called tumors. In the case of leukemia, a special type of cancer, no tumors are formed.

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Deterministic (Non-Stochastic) Effects

Deterministic (Non-Stochastic) Effects

General

Deterministic (non-stochastic) effects are those whose severity, but not probability, increases with dose.

Deterministic effects primarily result from cell killing, but delays in cell division, cell sterilization, severe changes in cell function, replacement of functional cells with fibrotic tissue, and damage to the supporting blood vessels can also play an important role.

The result is the gradual atrophy of the tissue. In some cases the loss of function may eventually reverse, e.g., a decrease in glandular secretion may only be temporary if the damaged tissue can be replaced by fully functional cells.

Deterministic (Non-Stochastic) Effects

General

Deterministic effects have a threshold dose below which they will not occur. In other words, it is possible to say that the effect will definitely not occur at doses below the threshold and the effect will definitely occur above a certain dose.

This “threshold” is the dose (or range of doses) above which the severity of the effect is large enough to be diagnosed.

For example, the severity of cataracts depends on the number of cells in the lens of the eye that are killed. Cells might killed below the threshold, but if not enough have died for cataracts to be observed we say that the effect has not occurred.

Deterministic (Non-Stochastic) Effects

General

Early (acute) effects are non-stochastic because they are pimarily brought about by cell death.

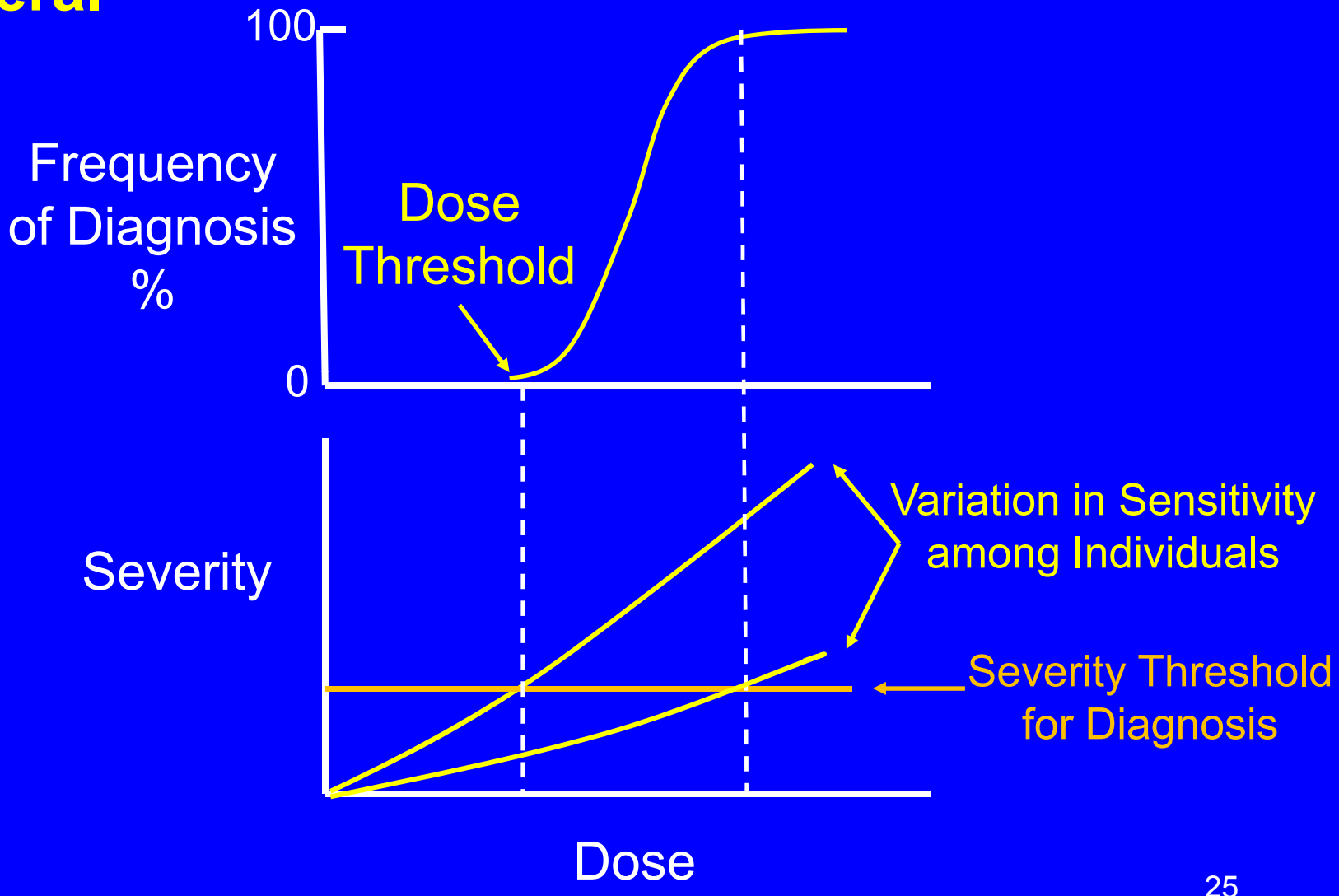
Late (delayed effects) can be deterministic or stochastic. Deterministic late effects involve the death of slowly dividing cells, e.g. those in the liver.

The U.S. regulatory agencies prevent deterministic effects for workers by restricting the annual dose equivalent to a single tissue to 50 rem. The dose equivalent to the lens of the eye is restricted to 15 mrem.

The ICRP does not employ deterministic limits.

Deterministic (Non-Stochastic) Effects

General



Somatic Stochastic Effects

Somatic Stochastic Effects

Hiroshima and Nagasaki Life Span Study

Our current risk estimate of developing and/or dying of cancer due to an external exposure is primarily based on studies of the survivors of those exposed at Hiroshima and Nagasaki, i.e., the life span study (LSS).

In the year 2000, 45% of the 86,611 survivors were still alive.

Of the 10,127 deaths due to solid cancers, 479 were estimated to be due to radiation.

Of the 296 deaths due to leukemia, 93 were estimated to be due to the radiation exposure.

Somatic Stochastic Effects

Hiroshima and Nagasaki Life Span Study

The advantages of the LSS include:

- The large number of individuals (in the tens of thousands)
- The study group represents a typical population, i.e., male and female, young and old, healthy and infirm
- We have excellent information concerning the medical history of the exposed individuals, i.e., what they die from and when.

Somatic Stochastic Effects

Hiroshima and Nagasaki Life Span Study

Sources of uncertainty in the LSS include:

- The individuals were exposed to large doses in a short time (acute exposures). Typical exposures to workers and the public are much lower and delivered over a long time
- Many of the exposed individuals experienced severe physical and mental trauma, disease and malnutrition.
- There is some uncertainty concerning the individual doses.
- The study is not complete (the entire study group must die) before the study can be completed.

Somatic Stochastic Effects

Hiroshima and Nagasaki Life Span Study

Sources of uncertainty in the LSS include:

- The possibility that the risks observed to the Japanese population are different than those that would apply to other ethnic groups.

  The table on the next slide indicates the annual death rate per 100,000 people in three countries (Shull 1995). Note, these are baseline risks that do not involve radiation exposure.

  The major contributor to the differences is undoubtedly lifestyle (e.g., diet, smoking, etc.), but a genetic contribution cannot be ruled out.

Somatic Stochastic Effects

Hiroshima and Nagasaki Life Span Study

Cancer	Sex	Japan	United Kingdom	United States
Bladder	Male	3.1	13.2	5.5
	Female	1.4	1.4	2.6
Breast	Male	-	-	-
	Female	9.0	52.3	33.5
Colon	Male	11.3	21.1	19.6
	Female	11.1	24.3	19.6
Leukemia	Male	5.4	7.8	8.1
	Female	3.8	6.5	6.2
Liver	Male	20.0	1.9	2.0
	Female	6.3	0.8	0.8
Lung	Male	40.6	100.6	73.5
	Female	14.6	41.9	35.9
Rectum	Male	8.5	2.8	3.4
	Female	5.8	10.0	2.9
Stomach	Male	50	22.7	6.8
	Female	29.0	14.7	4.4

Somatic Stochastic Effects

Mathematical Model of Risk

The total risk of developing or dying of a particular a particular cancer is the baseline risk (zero exposure) plus the excess risk.

The excess risk is the risk due to the radiation exposure.

$$R = R_0 + \text{Excess Risk}$$

R is the total risk (e.g., cancer deaths/rem)

R_0 is the baseline risk

Somatic Stochastic Effects

Mathematical Model of Risk

We sometimes estimate the risk of cancer at a specified age, i.e. during a particular year or years:

$$R(a) = R(a)_0 + \text{Excess Risk}$$

R(a) is the total risk of the specific cancer at age a

R_0 is the baseline risk of the cancer at age a

Somatic Stochastic Effects

Mathematical Model of Risk

There are many mathematical ways to model risk. In their simplest form they fall into two broad categories:

1. Absolute Risk Model
   (aka time-constant absolute or time-constant additive)

2. Relative Risk Models
   (aka time-constant relative or time-constant multiplicative)

   This is the preferred model.

Somatic Stochastic Effects

Additive (absolute) Risk Model

The excess risk (risk at age a) does not change, e.g., following an exposure at age 30, the excess risk is the same at ages 50 and 70 even though the baseline risk is greater at age 70.

No excess risk is attributed during a defined latent period (often 10 years). Mathematically, the excess risk is the product of the dose and a risk factor:

$$R(a) = R_0(a) + D \times \text{RISK FACTOR}$$

D is the dose, e.g. rad, Gy

Risk factor is the risk of cancer per unit dose, e.g. rad^{-1}, Gy^{-1}

Somatic Stochastic Effects

Multiplicative (relative) Risk Model

The excess risk increases with age in the same way that the baseline risk increases with age.

Therefore, the baseline risk $R_0(a)$, is a component of the excess risk term in the following linear equation:

$$R(a) = R_0(a) + R_0(a) \times D \times \text{RISK FACTOR}$$

This is the basic structure of the linear equation.

The risk factor can depend on the sex (s), age at exposure (e) and attained age (a).

Somatic Stochastic Effects

Multiplicative (relative) Risk Model

The linear quadratic version of this equation might look like something the following:

$$R(a) = R_0(a) + R_0(a)\,[\alpha\, D + \beta\, D^2]$$

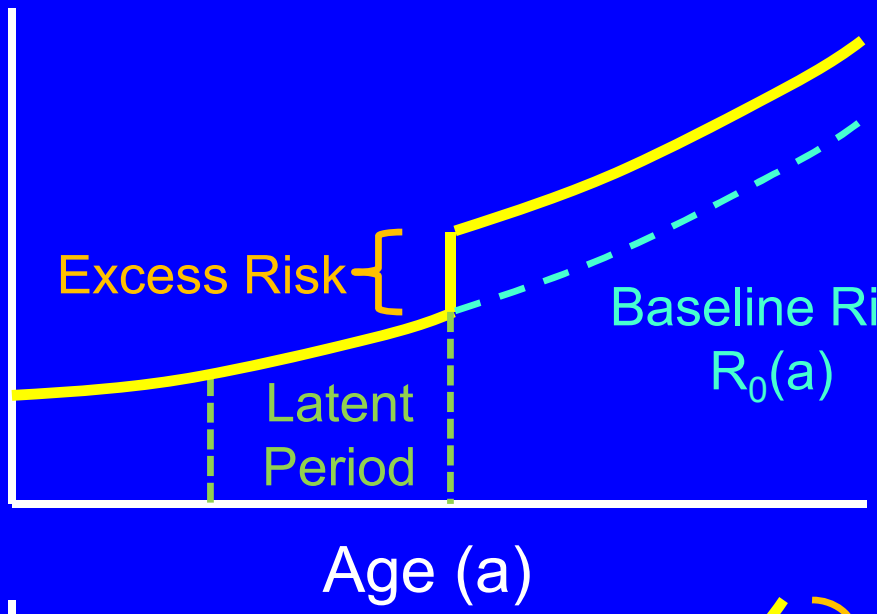
Where the risk factors are indicated by α and β.

If we want to account for cell killing or sterilization at high doses, the following linear quadratic exponential equation is employed:

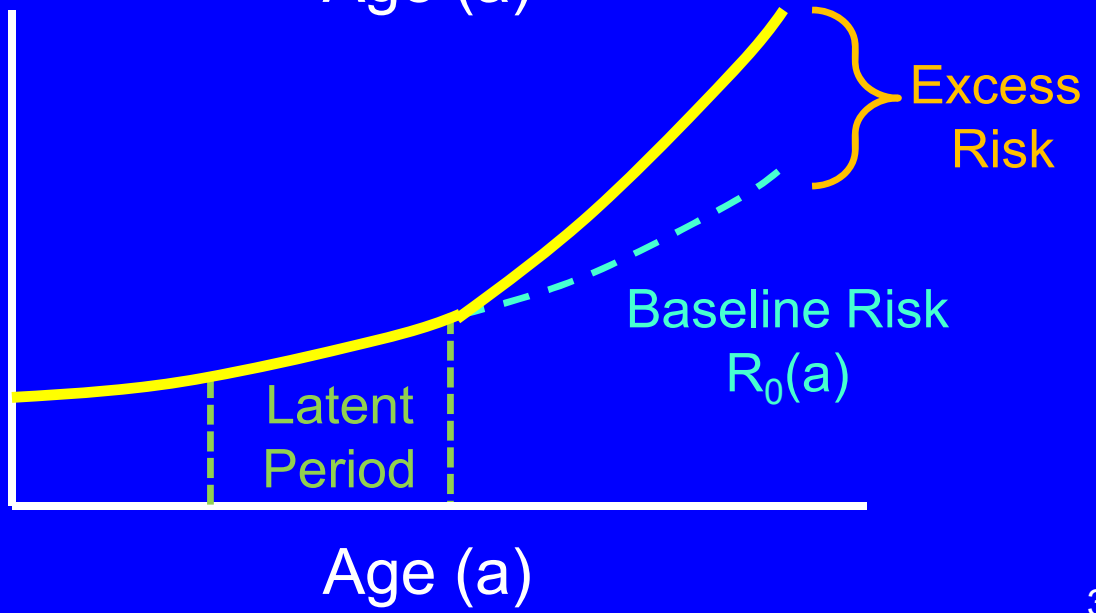
$$R(a) = R_0(a) + R_0(a)\,[\alpha\, D + \beta\, D^2]\, e^{\gamma D}$$

Somatic Stochastic Effects

Additive
Risk Model



Multiplicative
Risk Model



Somatic Stochastic Effects

Dose Response Relationship

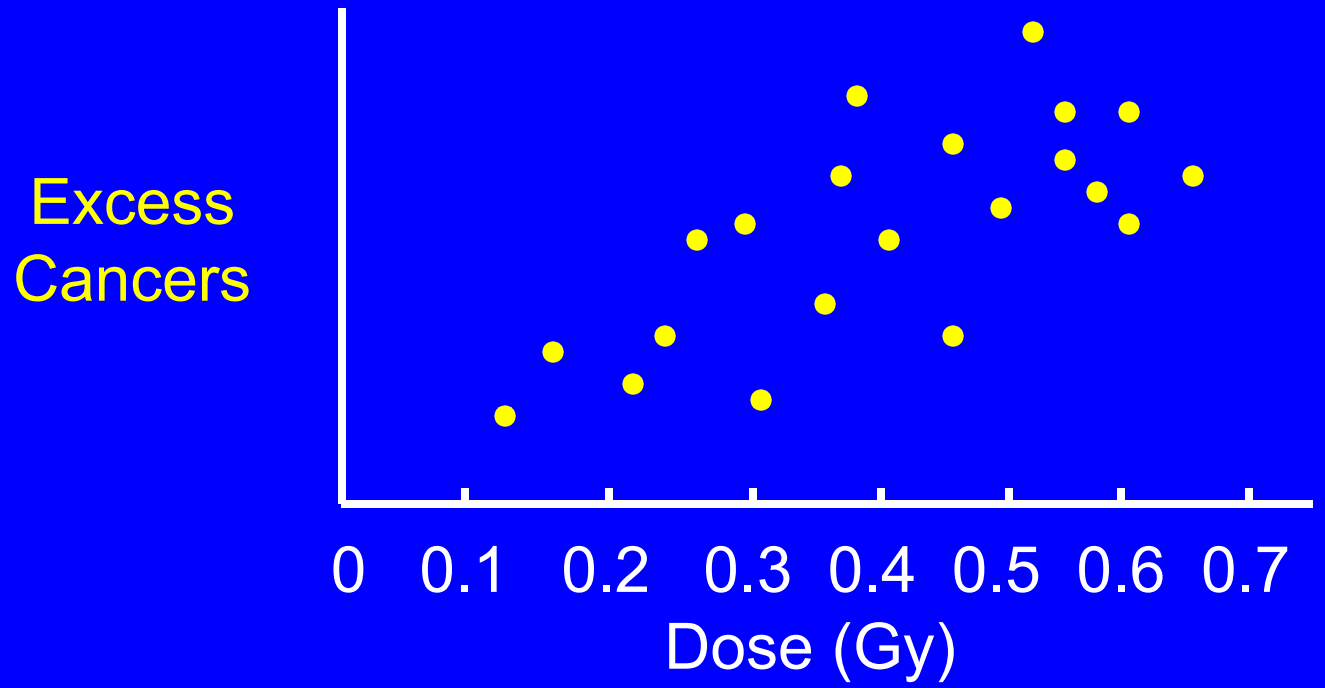
In the atomic bomb survivor life span studies (LSS) a statistically significant increase in deaths was observed above 0.1 Gy (10 rads) or so.

An increase in deaths was observed below 0.1 Gy but there is no consensus as to whether or not they were statistically significant.

The plot on the next slide is intended to convey the problem we are faced with when interpreting the data seen at Hiroshima and Nagasaki – it is not based on real data.

Somatic Stochastic Effects

Dose Response Relationship



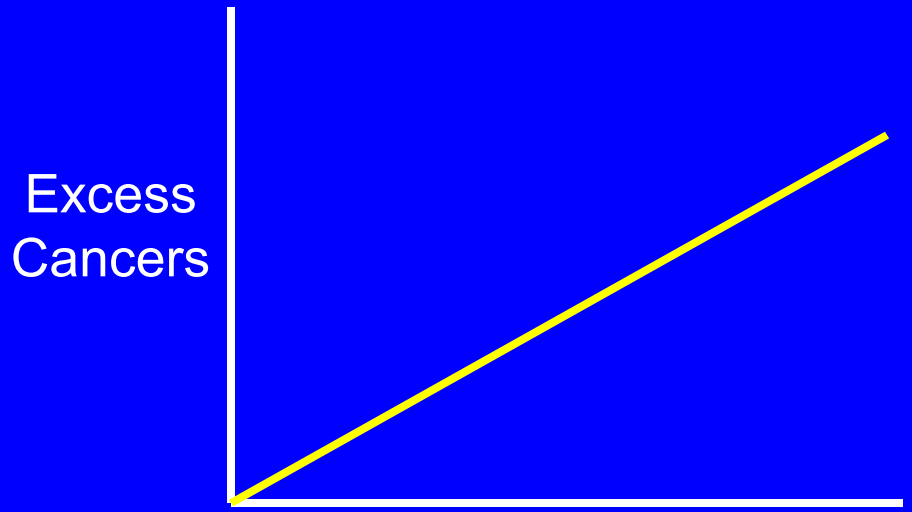
We are primarily interested in the risk at low doses (e.g., < 0.05 Gy), but there is no statistically significant data here!

This forces us to extrapolate the risks at high doses to the low dose region. How we do this depends on our "politics."

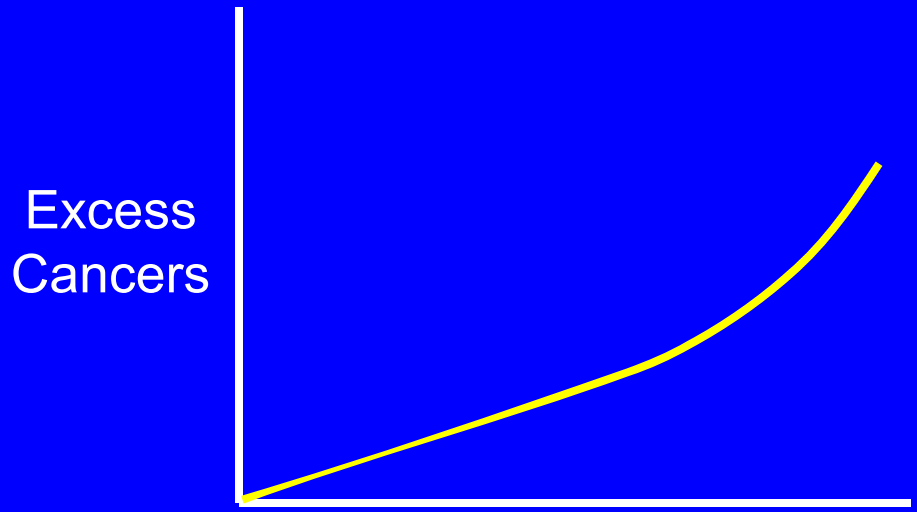
Somatic Stochastic Effects

The "Standard" Extrapolation Approach

The standard approach preferred by regulatory agencies and advisory bodies (e.g., ICRP) is to employ the linear no threshold approach. For some cancers (e.g., leukemia), the linear quadratic approach provides a better fit.



Linear No Threshold (LNT)

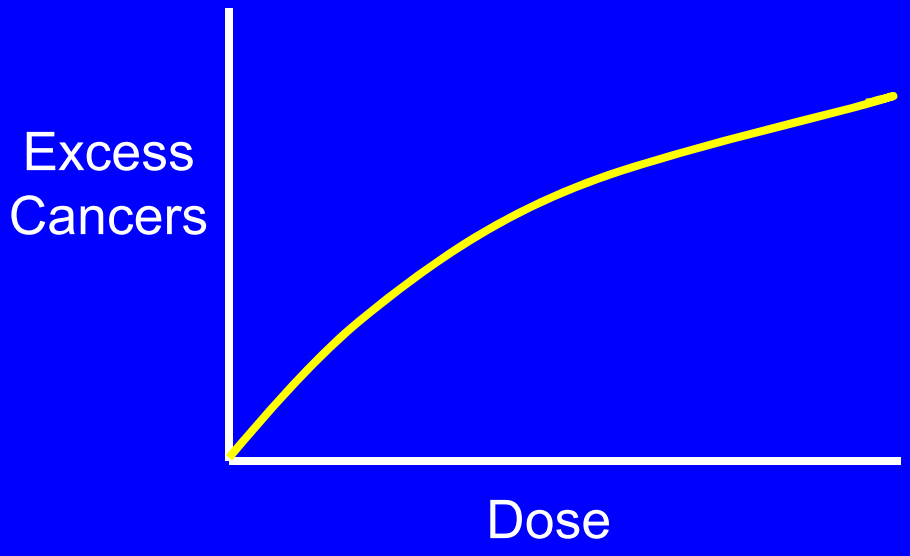


Linear - Quadratic

Somatic Stochastic Effects

An "Anti-nuke" Extrapolation Approach

An anti-nuke might prefer to extrapolate using the supralinear model. In this case, the risk, which is the slope of the line (deaths/Gy) , is greater at the low doses to which the public is exposed than it is at the higher doses seen at Hiroshima and Nagasaki.

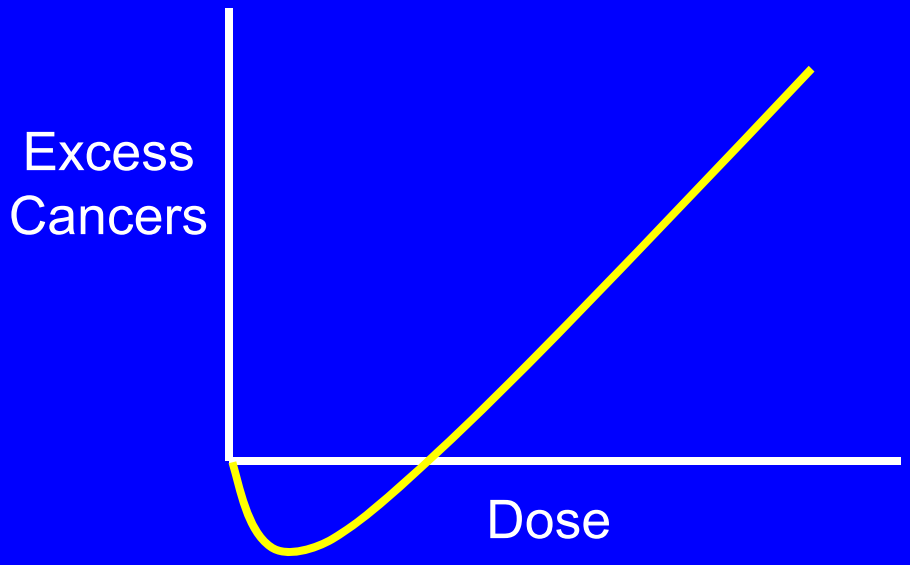


Supralinear

Somatic Stochastic Effects

A "Pro-nuke" Extrapolation Approach

A pro-nuke might prefer to extrapolate as follows. At doses below some value (usually unspecified) the number of excess deaths goes negative. In other words, at low doses, radiation exposures reduce the risk of cancer. This is known as radiation hormesis.



Radiation Hormesis

Somatic Stochastic Effects

Dose and Dose Rate Effectiveness Factor

Unless corrections are made, the linear dose-response model assumes that the risk per unit dose (e.g., excess deaths per rem) is the same at low doses as at the high doses and dose rates seen at Hiroshima and Nagasaki.

There is reason to believe the cellular repair mechanisms are more efficient at low doses and dose rates. If true, the risk per unit dose is lower at low doses.

To account for this, and more accurately estimate the risk at low doses, it is common to divide the risk observed in the life span study (LSS) by the dose rate effectiveness factor (DREF).

Somatic Stochastic Effects

Dose and Dose Rate Effectiveness Factor

The ICRP prefers to call this correction factor the dose and dose rate effectiveness factor (DDREF).

The DDREF (or DREF) is the magnitude of the risk overestimate at low doses. ICRP Report 60 recommended a value of 2 (ICRP 60, p.18). The more recent ICRP 103 continues to employ a DDREF of 2. The BEIR VII report prefers a value of 1.5.

Unless stated otherwise, risks presented in the literature have not been adjusted with the DDREF. It is left to the reader to decide whether or not to divide the stated risk by the DDREF.

Somatic Stochastic Effects

Dose and Dose Rate Effectiveness Factor

The DDREF is intended to be used with low LET radiation (e.g., gamma rays, x-rays, beta particles and electrons), not high LET radiation (e..g., neutrons).

What constitutes the low doses and dose rates at which the DDREF can be applied?

The ICRP considers a dose under 0.2 Gy (20 rads) as low and they consider low dose rates to be those under 0.1 Gy/hr (10 rads per hour).

The BEIR VII report considers low doses to be those below 0.1 Sv (10 rems).

Somatic Stochastic Effects

Dose and Dose Rate Effectiveness Factor

The use of the linear-quadratic dose response relationship in a model automatically assigns a lower risk per unit dose at low doses.

The DDREF should not be used with such a model.

Somatic Stochastic Effects

Risks to Workers

The risks to radiation workers tend to be 50 - 60% of those to the general public.

The main reason is that the workforce has a smaller representation of the most sensitive age group, those under 20. In addition, the working population is healthier than the general population (healthy worker effect).

As a result, the risks to the public are estimated separately from the risks to workers.

Somatic Stochastic Effects

Primary Cancers Induced by Radiation

The LSS of the survivors of Hiroshima and Nagasaki observed an excess in a wide range of cancers.

Nevertheless, the overall increased risk is primarily due an excess of four types of cancer:

- Leukemia
- Breast cancer
- Lung cancer
- Cancer of the digestive system

Somatic Stochastic Effects

Leukemia

Leukemia is a malignant disease of the blood forming organs.

It involves a large increase in the population of leukocytes (granulocytes and lymphocytes). There is also a substantial reduction in the number of erythrocytes and platelets which leads to an increased risk of hemorrhage and anemia.

Despite the increase in the white blood cells, there is also an increased susceptibility to infection.

Leukemia is unique in that it doesn't involve the production of solid tumors.

The baseline incidence is ca. seven per thousand individuals.

Somatic Stochastic Effects

Leukemia

The excess risk of leukemia begins to rise approximately two years after the exposure.

It peaks around 5 years after the exposure and then declines.

After 25 years or so, the rate of leukemia becomes identical for exposed and unexposed populations.

The short latent period and the significant decrease in the risk with time following the exposure are not typical of other cancers.

Somatic Stochastic Effects

Leukemia

Acute myeloid leukemia, the most commonly occurring type in the general population, is the most likely to result from radiation exposures. On the other hand, chronic lymphatic leukemia has never been associated with radiation exposure.

Assuming a linear-quadratic dose response relationship, the BEIR V committee estimated an excess risk probability of approximately 1×10^{-4} per rem. In other words, an exposure of one rem carries a risk of approximately one in ten thousand of developing a fatal case of leukemia.

Somatic Stochastic Effects

Leukemia

The UNSCEAR 2006 report estimates the risk of developing a fatal case of radiation induced leukemia at two dose ranges:

At a dose of 0.1 Sv: $3 \text{ to } 5 \times 10^{-3}$ Sv^{-1} (0.3-0.5% Sv^{-1})
10 rems: $3 \text{ to } 5 \times 10^{-5}$ rem^{-1}

At a dose of 1 Sv: $6 \text{ to } 10 \times 10^{-3}$ Sv^{-1} (0.6-1.0% Sv^{-1})
100 rems: $6 \text{ to } 10 \times 10^{-5}$ rem^{-1}

These estimates have implicitly accounted for the DDREF.

Somatic Stochastic Effects

Breast Cancer

Breast cancer is the most common type of cancer in women. Approximately one in nine develop it, although it rarely occurs in women younger than twenty-five.

One concern with breast cancer is the possibility that it might metastasize to another part of the body. Breast cancer is especially prone to spreading to the skeleton.

The latent period for radiation induced breast cancer is estimated at 10 years with the rate of incidence peaking 15 to 20 years after the exposure. The time at which the incidence of death peaks is 20 to 25 years after the exposure. After this period, there is a slow decline in the risk.

Somatic Stochastic Effects

Breast Cancer

The BEIR V committee estimated the risk for those exposed under the age of 20 to be four times the risk to those between 20 and 40 and roughly ten times the risk to women over the age of 40.

Women who have borne children and those who experienced late menarche or early menopause are at lower risk.

The BEIR V report estimated the excess risk at 0.7×10^{-4} per rem (0.7% Sv^{-1}) while the UNSCEAR 88 report put the risk at 0.6×10^{-4} per rem (0.6% Sv^{-1}).

Somatic Stochastic Effects

Lung Cancer

Lung cancer is a common form of cancer that can be induced by any number of chemical or physical irritants. It almost never occurs in those under forty.

Approximately 1.1% of male and 0.6% of female non-smokers die of lung cancer.

For male smokers the risk is 12.3% and for female smokers it is 5.8%.

If caught in the very early stages, the tumor might be successfully removed with surgery.

Somatic Stochastic Effects

Lung Cancer

Regarding radiation-induced lung cancer the age at exposure is not a major factor. The risk decreases with time after the exposure, however.

BEIR V estimated the risk at 1.9×10^{-4} per rem ($1.9\ \% \ Sv^{-1}$) for males and 1.5×10^{-4} per rem ($1.5\ \% \ Sv^{-1}$) for females.

The UNSCEAR 1988 average risk for males and females is 1.5×10^{-4} per rem ($1.5\ \% \ Sv^{-1}$).

Somatic Stochastic Effects

Cancer of the Digestive Tract

The baseline incidence of stomach cancer appears to be influenced by both lifestyle and genetics. Diets rich in smoked, pickled and salted foods predispose one to stomach cancer.

After leukemia, it showed the largest excess among the survivors of Hiroshima and Nagasaki.

ICRP 60 estimated the risk of fatal stomach cancer at 1.1 x 10^{-4} per rem (1.1% Sv^{-1}). The excess relative risk at 100 rem (1 Sv) is estimated at 0.23 and the corresponding relative risk is 1.2.

Somatic Stochastic Effects

Cancer of the Digestive Tract

Colon cancer can be characterized by anemia, loss of weight and changes in bowel habits. It is most commonly observed in those over forty. Roughly twice the incidence of colon cancer was observed among the survivors of Hiroshima and Nagasaki as among the control population. ICRP 60 estimated the excess risk for colon cancer at 0.85×10^{-4} per rem ($0.85\%\ Sv^{-1}$).

ICRP 60 estimated the risk for all components of the digestive system to be 2.25×10^{-4} per rem ($2.25\%\ Sv^{-1}$). The BEIR V report estimated the risk at 1.7×10^{-4} and 2.9×10^{-4} per rem for males and females respectively, with an average value of 2.3×10^{-4} ($2.3\%\ Sv^{-1}$).

Somatic Stochastic Effects

UNSCEAR 1988 Risk Estimates

The overall risk of fatal cancer is 11 x 10^{-4} per rem (11% per Sv). This does not account for a decreased risk at low doses.

UNSCEAR 2000 Risk Estimates

Male solid tumor fatalities: 9 x 10^{-4} per rem (9% per Sv)
Male leukemia fatalities: 1 x 10^{-4} per rem (1% per Sv)
Female solid tumor fatalities: 13 x 10^{-4} per rem (13% per Sv)
Female leukemia fatalities: 1 x 10^{-4} per rem (1% per Sv)

For chronic exposures, they stated that the estimates could be reduced by 50%. In other words, the DDREF was not applied to the indicated risks.

Somatic Stochastic Effects

UNSCEAR 2006 Risk Estimates

The following fatal cancer risks, averaged over both sexes, are provided for two dose ranges. They are based on linear quadratic models so that an application of the DDREF is not appropriate:

Acute Exposure of 0.1 Sv (10 rems)

Solid tumor: 3.6 to 7.7 x 10^{-4} rem^{-1} (3.6 to 7.7% per Sv)
Leukemia: 0.3 to 0.5 x 10^{-4} rem^{-1} (0.3 to 0.5% per Sv)

Acute Exposure of 1 Sv (100 rems):

Solid tumor: 4.3 to 7.2 x 10^{-4} rem^{-1} (4.3 to 7.2% per Sv)
Leukemia: 0.6 to 1.0 x 10^{-4} rem^{-1} (0.6 to 1.0% per Sv)

Somatic Stochastic Effects

BEIR V Risk Estimates

For a mixed population and an acute exposure, BEIR V estimated the risk of dying of radiation induced cancer to be about 8×10^{-4} per rem (0.8% per 0.1 Sv).

"For low LET radiation, accumulation of the same dose over weeks or months, however, is expected to reduce the lifetime risk appreciably, possibly by a factor of 2 or more."

Somatic Stochastic Effects

BEIR V Risk Estimates

Males

The risk for all cancers: 7.7×10^{-4} per rem ($7.7\%\ Sv^{-1}$).

Broken down, the risk for leukemia was 1.1×10^{-4} per rem and for non-leukemia cancers was 6.6×10^{-4} per rem.

Females

The risk for all cancers: 8.1×10^{-4} per rem ($8.1\%\ Sv^{-1}$).

Broken down, the risk for leukemia was 0.8×10^{-4} per rem and for non-leukemia cancers was 7.3×10^{-4} per rem.

Somatic Stochastic Effects

BEIR VII Risk Estimates

Solid cancer mortality averaged for both sexes (DDREF of 1.5)

5.1×10^{-3} per 0.1 Gy (5.1×10^{-4} per rad)

Leukemia mortality averaged for both sexes (DDREF of 1.5):

6.1×10^{-4} per 0.1 Gy (0.61×10^{-4} per rad)

Somatic Stochastic Effects

BEIR VII Risk Estimates

Males

Solid cancer mortality (DDREF of 1.5)

4.1×10^{-3} per 0.1 Gy (4.1×10^{-4} per rad)
2.9×10^{-3} per 1 mGy/y for life (2.9×10^{-3} per 100 mrad/y)

Leukemia mortality (DDREF of 1.5)

6.9×10^{-4} per 0.1 Gy (6.9×10^{-5} per 1 rad)
4.7×10^{-4} per 1 mGy/y for life (4.7×10^{-4} per 100 mrad/y)

Somatic Stochastic Effects

BEIR VII Risk Estimates

Females

Solid cancer mortality (DDREF of 1.5):

6.1×10^{-3} per 0.1 Gy (6.1×10^{-4} per rad)
4.6×10^{-3} per 1 mGy/y for life (4.6×10^{-3} per 100 mrad/y)

Leukemia mortality (DDREF of 1.5):

5.2×10^{-4} per 0.1 Gy (0.52×10^{-4} per rad)
3.8×10^{-4} per 1 mGy/y for life (3.8×10^{-4} per 100 mrad/y)

Somatic Stochastic Effects – BEIR VII Risk Estimates

Lifetime Attributable Risk of Cancer Incidence: cases per 100,000 persons exposed to a single dose of 0.1 Gy (10 rads)

Cancer Site	Age at Exposure (years)					
	0	10	20	40	60	80
Males						
Stomach	76	55	40	27	20	7
Colon	336	241	173	122	94	30
Liver	61	43	30	21	14	3
Lung	314	216	149	104	89	34
Prostate	93	67	48	35	26	5
Bladder	209	150	108	79	66	23
Other	1123	503	312	172	98	23
Thyroid	115	50	21	3	0.3	0.0
All solid	2326	1325	881	564	407	126
Leukemia	237	120	96	84	82	48
All cancers	2563	1445	977	648	489	174
Females						
Stomach	101	72	52	35	27	11
Colon	220	158	114	79	62	23
Liver	28	20	14	10	7	2
Lung	733	504	346	240	201	77
Breast	1171	712	429	141	31	4
Uterus	50	36	26	16	9	2
Ovary	104	73	50	31	18	5
Bladder	212	152	109	78	64	24
Other	1339	523	323	181	109	30
Thyroid	634	275	113	14	1	0.0
All solid	4592	2525	1575	824	529	177
Leukemia	185	86	71	62	57	37
All cancers	4777	2611	1646	886	586	214

Somatic Stochastic Effects – BEIR VII Risk Estimates

Lifetime Attributable Risk of Cancer Mortality: cases per 100,000 persons exposed to a single dose of 0.1 Gy (10 rads)

Cancer Site	Age at Exposure (years)					
	0	10	20	40	60	80
Males						
Stomach	41	30	21	15	11	4
Colon	163	117	84	60	49	21
Liver	44	31	23	16	12	4
Lung	318	219	151	107	93	42
Prostate	17	12	9	6	7	5
Bladder	45	32	23	17	17	10
Other	400	200	134	88	58	17
All solid	1028	641	444	310	246	102
Leukemia	71	71	67	67	73	51
All cancers	1099	712	511	377	319	153
Females						
Stomach	57	41	29	20	16	8
Colon	102	73	53	37	31	15
Liver	24	17	12	8	7	3
Lung	643	442	305	212	183	81
Breast	274	167	101	35	9	2
Uterus	11	8	6	4	3	1
Ovary	55	39	28	20	15	5
Bladder	59	43	31	23	22	13
Other	491	220	147	97	69	24
All solid	1717	1051	711	455	354	152
Leukemia	53	53	51	52	55	38
All cancers	1770	1104	762	507	409	190

Somatic Stochastic Effects

NCRP No. 126 Risk Estimates

The following risk estimates identified in NCRP 126 are the same as in NCRP No. 116. These, in turn, are based on the risk estimates of ICRP 60. They incorporate a DDREF of 2.

5×10^{-2} Sv^{-1} for a population of all ages

4×10^{-2} Sv^{-1} for adult workers

NCRP 126 discusses the uncertainties in these estimates in some detail.

Somatic Stochastic Effects

ICRP 60 Risk Estimates

ICRP 60 rounded the UNSCEAR 88 and BEIR V estimates to 10×10^{-4} per rem (10% Sv^{-1}) for high dose/ high dose rates.

When calculating the risk to the general population from low doses and low dose rates, ICRP 60 used a DDREF of 2.

This produced an overall risk of 5×10^{-4} per rem (5×10^{-2} per Sv) .

For workers, the estimated risk was somewhat lower: 4×10^{-4} per rem (4×10^{-2} per Sv) .

Somatic Stochastic Effects

ICRP 103 Risk Estimates

ICRP 103 provides its risk estimates in terms of the detriment (see Appendix B) rather than the risk of fatal cancer. These risks are higher than the risk of mortality alone because the detriment factors in cancer incidence and hereditable effects.

Population Exposure

5.5×10^{-2} per Sv (5.5×10^{-4} per rem)

Worker Exposure

4.1×10^{-2} per Sv (4.1×10^{-4} per rem)

Somatic Stochastic Effects

ICRP 103 Risk Estimates

"It is therefore the recommendation of the Commission that the approximated overall fatal risk coefficient of 5% per Sv on which current international radiation safety standards are based continues to be appropriate for the purpose of radiological protection."

Somatic Deterministic Effects

Somatic Deterministic Effects

General

At the doses commonly experienced by radiation workers and the public, the severity of non-stochastic effects is so low as to make them undetectable.

Somatic Deterministic Effects

General

The following table is taken from ICRP 60.

Threshold for Selected Non-Stochastic Effects (rad)			
	Tissue/Effect	Single (acute) Exposure	Annual (chronic) Exposure Protracted over many years
Male	Temporary sterility	15	40
	Permanent sterility	350 - 600	200
Female	Permanent sterility	250 - 600	> 20
Lens of Eye	Detectable opacities	50 - 200	> 10
	Visual impairment (cataracts)	500	> 15
Bone marrow	Decrease in blood cell count	50	> 4

Somatic Deterministic Effects

Cataracts

Cataracts are one of the most important of the non-stochastic (deterministic) effects in man.

Cataracts induced by radiation tend to develop on the posterior surface of the lens of the eye (posterior subcapsular) while the cataracts associated with old age, diabetes, etc., tend to occur on the anterior surface.

Posterior subcapsular cataracts can be induced by other causes however, e.g., trauma.

They might appear as soon as 10 months after an acute exposure of 200 rads (2 Gy) or chronic exposures of 1000 rads (10 Gy) from low LET radiation.

Somatic Deterministic Effects

Cataracts

The threshold is lower for high LET radiation: 75 rads (0.75 Gy) or so.

Neutrons, fast neutrons in particular, are especially effective at inducing cataracts.

The relative risk for radiation induced cataracts decreases with age.

Hereditable Effects

Hereditable Effects

General

Hereditable effects are those effects that occur in the descendants of the exposed individual. The are also referred to as genetic effects but hereditable is better since almost all radiation effects are "genetic" in that they involve effects on the genetic material in the chromosomes.

The radiation damage, mutations or chromosomal aberrations, takes place in the reproductive cells of the exposed individual. By reproductive cells we primarily mean the oocytes, spermatogonia and sperm cells. Hereditable effects could occur if this damage is passed on to subsequent generations.

Hereditable Effects

General

Chromosomal aberrations (rings, dicentrics, acentric fragments and inversions) in the reproductive cells are the type of DNA damage that is the most likely to result in readily apparent abnormalities in the offspring.

Effects due to mutations (genetic aberrations) that are dominant or X-linked recessive mostly occur in the first two generations.

Recessive mutations (unless X-linked) have almost no effect in the first two generations but lead to an overall increase in the genetic damage in the gene pool.

Hereditable Effects

General

While animal studies have indicated that genetic damage due radiation exposures can be passed on to subsequent generations, there are no similar human studies.

None of the offspring of survivors of Hiroshima and Nagasaki have shown effects that could be linked to the radiation exposures of their parents.

Perhaps the best evidence that radiation induced mutations can be passed on to subsequent generations in humans was presented in a report in the journal Science (Dubrova 2002). A gradual decrease in mutations was observed in subsequent generations of the offspring of those who lived near the Semipalatinsk nuclear test site in Kazakhstan.

Hereditable Effects

General

It has been reported that the children of radiation workers at the Sellafield nuclear facility in England have an increased risk of leukemia. Caution must be used when assessing such reports because childhood cancer is rare and it only takes a few cases to give the appearance of a greatly increased risk.

It is possible that the increased leukemia rate, if real, is due to an increase in viral infections created by large scale immigration to the area. When large facilities like nuclear plants are built, the area experiences a wave of immigration and an concurrent increase in viral infections, a phenomenon that has shown a link to the rate of leukemia.

Hereditable Effects

BEIR Risk Estimates

Except for their risk estimates for congenital abnormalities which are based on mouse data, the BEIR committee used the doubling dose concept to come up with their BEIR V and BEIR VII risk estimates.

The doubling dose is the radiation dose that doubles the rate of mutations. In both reports, it was assumed to be 1 Gy (100 rads).

Unlike the ICRP, they limit their risk projections to the first two generations.

Hereditable Effects

BEIR Risk Estimates

Multifactorial diseases are those that have a genetic component but are not “inherited” according to classic Mendelian genetics. They occur because of a combination of genetics and the environment (e.g., diet). Examples include diabetes and heart disease.

Congenital abnormalities are those that are present at birth.

Hereditable Effects

BEIR V Risk Estimates

Risk per Gray per Million Progeny at			
Disease Class	**Baseline Frequency per Million Live Births**	**First Generation**	**Second Generation**
Mendelian			
Autosomal dominant	10,000	600 to 3500	NE
X-linked	400	<100	NE
Autosomal recessive	2,500	<100	NE
Chromosomal	4,400	<600	NE
Multifactorial			
Congenital abnormalities	20,000 to 30,000	1000	NE
Heart disease	600,000	NE	
Cancer	300,000	NE	
Selected others	300,000	NE	

Hereditable Effects

BEIR VII Risk Estimates

Risk per Gray per Million Progeny at			
Disease Class	**Baseline Frequency per Million Live Births**	**First Generation**	**Second Generation**[a]
Mendelian			
Autosomal dominant and X-linked	16,500	750 to 1500	1300 to 2500
Autosomal recessive	7,500	0	0
Chromosomal	4,000	[b]	[b]
Multifactorial			
Chronic multifactorial	650,000[c]	250 to 1200	250 to 1200
Congenital abnormalities	60,000	2000 [d]	2400 to 3000
Total	738,000	3000 to 4700	3950 to 6700
Total risk of baseline per gray as percent		0.41 to 0.64	0.53 to 0.91

Hereditable Effects

BEIR VII Risk Estimates

Notes to previous table:

a. Risk to second generation includes that of the first generation

b. Assumed to be subsumed in part under the risk of autosomal dominant and X-linked diseases and in part under congenital (condition present at birth) abnormalities

c. Frequency in the population

d. Calculated using mouse data on developmental abnormalities without using the doubling dose method.

Hereditable Effects

BEIR VII Risk Estimates

The risk estimate equation used in BEIR VII is:

$$\text{Risk per unit dose} = P \times (1/DD) \times MC \times PRCF$$

P is the baseline frequency of the disease class

DD is the doubling dose

MC is the mutation component for that disease

PRCF is the potential recoverability correction factor

Hereditable Effects

BEIR VII Risk Estimates

Autosomal Dominant and X-Linked

P = 16,500/10^6
DD = 1 Gy
MC = 0.3 for first generation and 0.51 for the second
PRCF = 0.15 to 0.30

Chronic Diseases

P = 650,000/10^6
DD = 1 Gy
MC = 0.02
PRCF = 0.02 to 0.09

Hereditable Effects

UNSCEAR 2001 Risk Estimates

UNSCEAR 2001 estimates the risk of hereditable effects using the same doubling dose approach and equation as BEIR VII. They even use exactly the same values for the doubling dose, baseline frequency, mutation component and potential recoverability correction factor!

Quoting the report: “For a population exposed to radiation in one generation only, the risks to the progeny of the first post radiation generation are estimated to be 3,000 to 4,700 cases per gray per one million progeny; this constitutes 0.4 to 0.6 per cent of the baseline frequency of those disorders in the human population.”

Hereditable Effects

ICRP 60 Risk Estimates

ICRP 60 estimated the detriment for all generations to be 0.5 x 10^{-4} per rem. Dominant and X-linked mutations are believed to be responsible for 80% of these effects.

The detriment for multifactorial effects was estimated at 0.5 x 10^{-4}. Multifactorial effects have a genetic component but do not occur unless certain environmental conditions exist.

Therefore, the total risk estimate for the general population is 1 x 10^{-4} per rem.

When the ICRP took into account the years of life lost by the descendants, the risk increased to 1.3 x 10^{-4}.

Hereditable Effects

ICRP 103 Risk Estimates

As was the case with somatic effects, ICRP 103 provides these risk estimates in terms of the detriment (see Appendix B) which factors in cancer incidence as well as cancer mortality. ICRP 103 uses the same mathematical approach to estimating the risks as BEIR VII and UNSCEAR 2001.

Population Exposure

0.2×10^{-2} per Sv (0.2×10^{-4} per rem)

Worker Exposure

0.1×10^{-2} per Sv (0.1×10^{-4} per rem)

Hereditable Effects

ICRP 103 Risk Estimates

The ICRP 103 risk estimates for hereditable effects are a factor of 6-8 lower than those of ICRP 60.

The reduced genetic risk has resulted in a significant reduction in the tissue weighting factors for the gonads.

Fetal (*in utero*) Effects

Fetal (*in utero*) Effects

General

In order to minimize the risk to the fetus, the U.S. Nuclear Regulatory Commission limits the dose during the entire pregnancy to 0.5 rems (5 mSv).

This section considers the following risks due to a fetal exposure:

Cancer
Mental Retardation
Decreased IQ
Malformations

Fetal (*in utero*) Effects

Cancer

As noted in the BEIR VII report, “In the case of *in utero* exposures (exposures to the fetus during pregnancy, excess cancers can be detected at doses as low as 10 mSv [1 rem]”

Based on the atomic bomb life span study, they noted that the excess relative risk (ERR) per Sv was 2.1. Of the ten individuals in the LSS exposed *in utero* who developed cancer, nine were female.

Summarizing medical exposure data, UNSCEAR 1996 noted that there was a statistically significant increased risk of leukemia and a 40% increase in the risk of childhood cancer at doses of 10 to 20 mSv (1 to 2 rem).

Fetal (*in utero*) Effects

Mental Retardation

The evidence from the LSS indicates that the risk of severe mental retardation is extremely small for exposures during the first seven weeks after fertilization.

The observed risk was greatest during the 8 - 15 week period following fertilization when the CNS neurons are rapidly increasing in number.

During the 16 - 25 week period, the risk of mental retardation still exists but has been substantially reduced.

After 26 weeks the CNS continues to develop but no increased risk was observed for those exposed during this period.

Fetal (*in utero*) Effects

Mental Retardation

ICRP 90 noted that the LSS data indicated a threshold for mental retardation of at least 300 mGy (30 rads) when the exposure was in the most sensitive stage, i.e., the 8 to 15 week period post conception.

Fetal (*in utero*) Effects

Decreased IQ

Treating mental retardation as an either/or phenomenon with a threshold of 20 to 40 rads (0.2 to 0.4 Gy) can be misleading in that it might be taken to imply that doses below this threshold cannot have effects on intelligence.

With regard to the risk of a decreased IQ, ICRP 103 stated "even in the absence of a true dose threshold, any effects on IQ following in-utero doses under 100 mGy [10 rads] would be of no practical significance." They also noted that "data on IQ losses estimated at around 25 points per Gy [100 rads] are more difficult to interpret."

Fetal (*in utero*) Effects

Malformations

At birth approximately 3% of children exhibit some type of significant malformation. Over time, two to three times this number demonstrate some type of abnormality.

The most common malformation observed in humans due to prenatal exposures to radiation is microcephaly (decreased head size). Almost all microcephalic children will also be severely mentally retarded.

Other observed malformations include growth retardation, abnormalities of the eye (cataracts and retinal pigmentation changes), and genital and skeletal malformations.

Fetal (*in utero*) Effects

Malformations

The lowest dose at which malformations have been attributed to a prenatal exposure is approximately 0.05 Gy (5 rads).

Quoting ICRP 103 “there are age-dependent patterns of in-utero radiosensitivity with maximum sensitivity being expressed during the period of major organogenesis. On the basis of animal data, it is judged that there is a true dose threshold of around 100 mGy [10 rads] for the induction of malformations; therefore for practical purposes, the Commission judges that risk of malformation after in-utero exposure to doses well below 100 mGy [10 rads] are not expected.”

Appendix A
Ways to Express Risk

Ways to Express Risk

Relative Risk (RR)

The relative risk is the ratio of the total risk to the baseline risk at a specified dose, usually 100 rad (1 Gy).

To say that the relative risk is 2 at 100 rad is to say that the total risk due to a 100 rad exposure is twice the baseline risk.

$$Relative\ Risk\ (RR) = \frac{R(a)}{R_0(a)}$$

Ways to Express Risk

Excess Relative Risk (ERR)

The excess relative risk (ERR) is the rate of the effect under consideration in the exposed population divided by the rate in an unexposed population minus 1.

To say that the relative excess is 1.0 at 100 rads means that the excess risk is the same as the baseline risk.

If the excess relative risk is 1.0, the relative risk is 2.0.

$$\begin{aligned} \textit{Excess Relative Risk (ERR)} &= \frac{R(a)}{R_0(a)} - 1 \\ &= \textit{Relative Risk} - 1 \\ &= \frac{\textit{Excess Risk}}{R_0(a)} \end{aligned}$$

Ways to Express Risk

Excess Absolute Risk (EAR)

The excess absolute risk (EAR) is the rate of the effect in the exposed population minus the rate in the unexposed population.

$$Excess\ Absolute\ Risk\ (EAR) = R(a) - R_0(a)$$

Appendix B
ICRP Detriment

ICRP Detriment

General

The detriment is a risk approach wherein the risk of detrimental occurrences are reported rather than the number of deaths.

It is an attempt to recognize that there is more to the risk of radiation than just the risk of death.

The detriment tries to factor in the loss of life expectancy and also the number and severity of non-fatal cancers induced by radiation. Non-fatal cancers are not given equal weight to fatal cancers.

ICRP Detriment

General

The following table is from ICRP 60. It is intended to help explain the concept of the detriment.

The first column indicates the probability of fatal cancers.

The second column shows the risk of hereditable effects.

The third column calculates the relative length of life lost by each cancer type: the years of life lost for the cancer divided by the average years of life lost for all cancer types(15 years).

The fourth column calculates the relative contribution from non-fatal cancers by subtracting the lethality fraction (k) for a cancer from 2.

ICRP Detriment

ICRP 60 Data

	Probability of fatal cancer F (10^{-4} per rem)	Severe genetic effects (10^{-4} per rem)	Relative length of life lost l/l	Relative non-fatal contribution (2 - k)	Product F(l/l)(2 - K) (10^{-4} per rem)
Bladder	0.30		0.65	1.50	0.29
Bone marrow	0.50		2.06	1.01	1.04
Bone surface	0.05		1.00	1.30	0.07
Breast	0.20		1.21	1.50	0.36
Colon	0.85		0.83	1.45	1.04
Liver	0.15		1.00	1.05	0.16
Lung	0.85		0.90	1.05	0.80
Oesophagus	0.30		0.77	1.05	0.24
Ovary	0.10		1.12	1.30	0.15
Skin	0.02		1.00	2.00	0.04
Stomach	1.10		0.83	1.10	1.0
Tyroid	0.08		1.00	1.90	0.15
Remainder	0.50		0.91	1.29	0.59
Gonads		1.0	1.33	--	1.33
Total	5.0				7.25

ICRP Detriment

General

The product of these is the detriment.

Detriment = risk x relative life lost x non-fatal contribution

The total ICRP 60 detriment is 7.25×10^{-4}/rem ($7.25\%\ Sv^{-1}$).

The current ICRP 103 detriment is 5.7×10^{-4}/rem ($5.7\%\ Sv^{-1}$).

Note that the detriment is a little higher than the risk of fatal cancers, but not much.

The ICRP uses these detriments for the individual cancer types to calculate the tissue weighting factors.

Appendix C

Loss of Life Expectancy

Loss of Life Expectancy (LLE)

While it is most common to express the stochastic risks of radiation as the number of cancer deaths per unit dose, it is possible to express the risk as the average number of years of life lost (due to cancer).

The following table (ICRP 60 data) shows, for various types of cancer, the fraction that prove fatal and the average years of life lost per death.

Loss of Life Expectancy

The average years of life lost for all cancers (l) is 15.

The years of life lost because of exposure to the gonads refers to resulting deaths in the offspring.

Tissue	Lethality Fraction k	Life lost (years)
Bladder	0.50	9.8
Bone marrow[1]	0.99	30.9
Bone surface	0.70	15.0
Breast	0.50	18.2
Colon	0.55	12.5
Liver	0.95	15.0
Lung	0.95	13.5
Esophagus	0.95	11.5
Ovary	0.70	16.8
Skin	0.002	15.0
Stomach	0.90	12.4
Thyroid	0.10	15.0
Remainder	0.71	13.7
Gonads	--	20.0
	Average (l)	15.0

[1]leukemia

Loss of Life Expectancy (LLE)

If we take the risk per rem of dying of cancer and multiply it by the average years of life lost per cancer death (15), we obtain the loss of life expectancy per rem:

For acute exposures

10×10^{-4}/rem x 15 years/death = 5.5 days lost/rem

For chronic exposures (assuming a DDREF of 2)

5×10^{-4} /rem x 15 years/death = 2.75 days lost/rem

Loss of Life Expectancy (LLE)

In Regulatory Guide 8.29 the U.S. NRC estimates:

15 days of life would be lost due to an occupational exposure of 0.3 rems/y (3 mSv/y) from age 18 to 65

51 days of life would be lost due to an occupational exposure of 1 rems/y (10 mSv/y) from age 18 to 65

They also note that:

6 years of life would be lost because of smoking 20 cigarettes a day

2 years of life would be lost due to being 15% overweight

1 year is lost due to the average U.S. consumption of alcohol

Life expectancy (in years) on various continents and in various selected nations in 1985–1990 (World Resources Institute 1987).

World	61.1[a]		
Africa	51.3	Asia	61.1
Algeria	62.5	Afghanistan	39.0
Congo	48.5	Bangladesh	49.6
Egypt	60.6	China	69.4
Ethiopia	41.9	India	57.9
Gambia	37.0	Indonesia	56.0
Ghana	54.0	Iran	59.0
Kenya	55.3	Japan	77.2
Libya	60.8	Korea	69.4
Morocco	60.8	Pakistan	52.1
Nigeria	50.5	Saudi Arabia	63.7
Sierra Leone	36.0	Turkey	64.1
South Africa	55.5	Europe	74.0
North America	72.0	France	75.2
Canada	76.3	Germany (W)	74.5
Haiti	54.7	Italy	75.2
Mexico	67.2	Poland	72.4
United States	75.0	Sweden	76.8
South America	65.5	United Kingdom	74.5
Argentina	70.6	Yugoslavia	71.7
Bolivia	53.1		
Brazil	64.9	U.S.S.R.	72.1
Chile	70.7	Oceana	69.1
Peru	61.4	Australia	75.7
Venezuela	69.7	Papua N.G.	54.0

LLE due to sports participation per year of participation. The second column gives the probability per year of being killed (Reif 1981), the third column gives an estimate of the average life expectancy lost by a victim, and the last column gives the product of these × 365, which is the LLE in days.

Sport	Probability of death	Years lost per victim	LLE (d)
Prof. boxing	1:2,200	50	8
Hang gliding	1:560	40	25
Football			
-high school	1:81,000	60	0.3
Football			
-college	1:33,000	55	0.6
Mountain climbing			
-dedicated	1:167	50	110
-all climbers	1:1,750	50	10
Mountain hiking	1:15,700	40	0.9
Parachuting	1:570	40	25
Sail planing	1:1,710	40	9
Scuba diving (amateur)	1:2,400	45	7
Skiing-racing	1:40,000	50	0.5
Snowmobiling	1:7,600	40	2