# Anhang/Appendix A

## 6. HEALTH RISK ASSESSMENT (CANCERS)

### 6.1. OVERVIEW OF THE BEIR, UNSCEAR AND ICRP RECOMMENDATIONS: BASES FOR ESTIMATIONS; ADJUSTMENT TO PROGRESS IN KNOWLEDGE[1]

Information on the effects of low doses of radiation in humans is regularly evaluated and published by major scientific committees. The International Commission on Radiological Protection (ICRP) was established in 1928 with the initial name of International X-ray and Radium Protection Committee, following a decision by the Second International Congress of Radiology. The ICRP intends with its recommendations (see e.g. references 8 and 9) to help regulatory and advisory agencies at national, regional and international levels, mainly by providing guidance on the fundamental principles on which appropriate radiological protection can be based. In the early 1950's, the testing of nuclear weapons provoked public concern in the United States about the potential effects of ionizing radiation on human populations. In response to this concern the American National Academy of Sciences in 1955 appointed a group of scientists to conduct a continuing appraisal of the Biological Effects of Atomic Radiation on living organisms. These studies led to a series of reports generally referred to as BEAR reports. In 1964 an Advisory Committee with the slightly modified name the Committee on Biological Effects of Ionizing Radiations (BEIR) was established with the task to review and evaluate the problems of radiation exposure and protection and to issue reports of its deliberations (see e.g. references 1 and 2).

Also in 1955, The General Assembly of the United Nations established the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). The major aims of UNSCEAR are to assess the consquences to human health of a wide range of doses to ionizing radiation and to estimate the dose to people all over the world from natural and man-made radiation sources (see e.g. references 18 and 19).

Biological effects of ionizing radiation are of a deterministic or stochastic nature. Stochastic effects can be divided into somatic effects (carcinogenesis) and genetic effects. In this section attention is focused on the estimation of somatic stochastic effects.

#### 6.1.1. SOMATIC STOCHASTIC EFFECTS

For deterministic effects the severity of the effect depends on the dose and they are not observed below a threshold. Many, but not all, deterministic effects involve cell killing. Deterministic

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effects are perceived only at relatively high doses and they are therefore hardly ever observed in occupational exposure or in diagnostic radiology.

Genetic effects and tumour induction due to exposure to radiation belong to the category of stochastic effects. For stochastic effects not the severity but the likelihood of occurrence of the effect depends on the dose. In radiation protection it is generally assumed that a threshold dose does not exist for stochastic effects. This implies that there is a certain probability that they occur even after exposure to very low doses of radiation. The incidence of stochastic effects has a random character and the probability of occurrence increases with increasing dose.

Direct evidence for the occurrence of somatic stochastic effects in men has only been obtained for doses in excess of 0.5 Gy. The risk at lower doses has to be derived from extrapolation. Animal studies have provided information on the different shapes of dose-effect relationships (3). When all appropriate corrections have been applied the dose response curves for radiation induced cancer can in their most general way be described as:

$$I(D) = (I_0 + a_1D + a_2D^2) \cdot \exp(-b_1D - b_2D^2)$$

where I(D) is the incidence after exposure to an absorbed dose D, $I_0$ is the spontaneous incidence, $a_1$ and $a_2$ are coefficients for the linear and quadratic term of cancer initiation and the exponential term is the probability of survival of transformed cells. For most radiation induced tumours in animals the dose response curves are linear-quadratic for low-LET radiation and only quadratic for high-LET radiation.

Some possible relations between the excess risk of tumour induction and absorbed dose are shown in Figure 6.1. The ICRP assumes in publication 60 a simple linear relation as shown in Figure 6.1(a). This might be a conservative approach since a linear extrapolation probably yields an upper limit for the risks of tumour induction at low doses. Also in the risk model of BEIR V linear extrapolation is applied for all cancers except leukaemia. For leukaemia BEIR V assumes a linear-quadratic relation between absorbed dose and risk (Figure 6.1(b)). Alternative models are for example the linear model with threshold (Figure 6.1(c)). This model assumes negligible risks below a certain threshold dose. Nuszbaum and Köhnlein (13) mention that supra-linear, dose effect relations could result in a better fit to the Japanese cancer mortality data than a linear relation. The results on mortality due to all cancers except leukaemia provide a hint of an increased slope for the dose range from 0 to 0.19 Gy compared to that for the range of 0.06 to 0.99 Gy. The hormesis model (Figure 6.1(d)) assumes a slight beneficial effect of low doses of radiation (12, 20).

Both the ICRP and BEIR committees acknowledge that a correction may have to be introduced when the linear model is used for the extrapolation of the radiation risks of low LET radiation to low dose and low dose-rate levels. This correction is referred to as a dose and dose rate effectiveness factor (DDREF), according to the ICRP (8) or a dose rate effectiveness factor (DREF), according to BEIR (2). Both committees suggest that the numerical value of this factor might be

2 (Figure 6.1(a)) although they recognize that this value has a large uncertainty. The committees have different views in the practical application of this factor. ICRP recommends a DDREF of 2 for all absorbed doses below 0.2 Gy and for higher absorbed doses when the dose rate is less than 0.1 Gy per hour. According to the ICRP model, the DDREF should always be applied. Contrarily, BEIR does not recommend the application of a DREF for non-leukaemia cancer incidence. The decision not to apply a DREF, is justified by the BEIR committee taking into account that most human exposures to ionizing radiation involve relatively low energy x rays. The high energy gamma rays to which the atomic bomb survivors were exposed are supposed to be only half as effective as the low energy x rays and thus BEIR concludes that it is likely that these two factors, i.e. DREF and the relative biological effectiveness of gamma rays compared to x rays, are cancelling each other out. In the leukaemia model of BEIR the choice of a linear-quadratic model already implies that at low doses the effectiveness of radiation is smaller than at high doses.

The prediction of an attributable death probability rate after exposure at a certain age of a person of a certain sex imposes another problem. After a latency period, the excess risk can be described by an additive model (as in the earlier ICRP publication 26 of 1977, see Figure 6.2(a)) or by a multiplicative model (as proposed by the ICRP in publication 60 of 1991, see Figure 6.2(b)). With the additive model the excess risk remains constant with time after the minimum latency period. In ICRP 60 a multiplicative model is used in which the excess risk is the product of a constant factor to be applied to the incidence rate of natural cancers. In BEIR V a slightly modified multiplicative model is used in which the factor by which the incidence rate has to be multiplied is not constant but depends on the number of years after exposure.

Risks associated with the exposure to ionizing radiation can be derived from epidemiological studies. The most important study is the Life Span Study. The cohort of the Life Span Study includes 93,000 survivors of the atomic bombings in Japan and 27,000 persons who lived in Hiroshima and Nagasaki in 1950 but who were not in the cities at the time of the bombings (control group). The Life Span Study suggests that between 1950 and 1987 roughly 300 of the 6,900 cancer deaths in the cohort are associated with radiation exposure. Of the 231 first leukaemia cases about 76 are in excess of expectation (19). These numbers are rather small. They are, however, large enough to estimate a dose-effect relation. It should be realized that the corresponding uncertainties remain rather large.

Additional information on the incidence and mortality of cancer due to exposure to ionizing radiation is derived from two other epidemiological studies notably a cohort of 14,000 patients undergoing radiotherapy of the spine for ankylosing spondylitis in the United Kingdom and 83,000 women treated for cancer of the uterine cervix in a number of countries (18). The latter two studies have resulted in lower risk factors than those estimated for the group of Japanese survivors (4). Nevertheless the ICRP has taken a safe starting point by basing its risk factors on

the A-bomb survivors. Table 6.1 provides a survey of the risk factors for adult workers and for the whole population.

Table 6.1. Nominal probability coefficients for stochastic effects (ICRP 60; 9)[1].

| *Exposed population* | *Detriment ($10^{-2}$ $Sv^{-1}$)* | | | |
|---|---|---|---|---|
| | *Fatal cancer*[2] | *Non-fatal cancer*[2] | *Severe hereditary effects*[2] | *Total* |
| Adult workers | 4.0 | 0.8 | 0.8 | 5.6 |
| Whole population | 5.0 | 1.0 | 1.3 | 7.3 |

1) A dose and dose rate effectiveness factor (DDREF) of 2 has been included in the probability coefficients.
2) The probability coefficients for non-fatal cancer and severe hereditary effects have been weighted by multiplying the probability coefficient for non-fatal cancers and hereditary effects with weighting factors smaller than 1. The weighting factor is higher when the detriment of the non-fatal cancers or the hereditary effects is more severe. The probability coefficients for fatal cancers are given a weighting factor of 1.

## 6.1.2. MATHEMATICAL MODELS FOR RISK ESTIMATION OF TUMOUR INDUCTION

In this section risks will be expressed as the attributable life-time risk (the risk of induction of a fatal tumour induction after exposure to ionizing radiation) and the number of years of life lost (9).

The attributable life-time risk and the number of years of life lost can be assessed from the age specific death rate from all causes (including induction of fatal cancer) in a non-exposed population q(a) and the age specific attributable excess death rate for a population exposed to ionizing radiation h(a) where a indicates the age.

The fractional survival in a non-exposed group ($L_{non\text{-}exposed}$ (a)) can be denoted as:

$$L_{non\text{-}exposed}(a) = L_{non\text{-}exposed}(a\text{-}1) \bullet (1\text{-}q(a))$$

and the fractional survival in an exposed group ($L_{exposed}$ (a)) as:

$$L_{exposed}(a) = L_{exposed}(a\text{-}1) \bullet (1\text{-}(q(a)+h(a)))$$

These expressions are used to calculate the attributable death probability rate (r(a)) and the fractional excess death due to exposure to ionizing radiation (s(a)):

$$r(a) = h(a) \bullet L_{exposed}(a)$$

$$s(a) = L_{non\text{-}exposed}(a) - L_{exposed}(a)$$

Finally the attributable life-time risk and the average number of years life lost (YLL) can simply be calculated by summation of r(a) and s(a) respectively:

$$\text{Attributable life-time risk} = \sum_{a} r(a)$$

$$\text{YLL} = \sum_{a} s(a)$$

A risk evaluation can thus be performed when q(a) and h(a) are known. The age specific death rate from all causes in a non-exposed population q(a) can be obtained from national registries. Unfortunately considerable uncertainties are involved in the assessment of h(a): the age specific attributable excess death rate for a population exposed to ionizing radiation. The models described by the ICRP in publication 60 are a simple additive model and a simple multiplicative model. For the additive model, $h_A(a)$ has a constant value after a latency period of m years following exposure to dose $D_A$ at the age of exposure A in years:

$$h_A(a) = \begin{cases} 0 & \text{for } a < A + m \\ D_A\, C_{additive}(A) & \text{for } a \geq A + m \end{cases}$$

For the multiplicative model, after a latency period of m years, the constant multiplicative factor $h_A(a)$ has to be applied to the annual age specific cancer rate for a non-exposed population, $q_{cancer}(a)$:

$$h_A(a) = \begin{cases} 0 & \text{for } a < A + m \\ D_A\, C_{multiplicative}(A).q_{cancer} & \text{for } a \geq A + m \end{cases}$$

In ICRP publication 60 the cancer rate per year for a non-exposed population is approximated by:

$$q_{cancer}(a) = \alpha.a^{\beta} + \gamma$$

In Figure 6.3 this parametric model is compared to the mortality rate of the Dutch population, for leukaemia and non leukaemia cancers, as published by the Netherlands Central Bureau of Statistics (6). At ages higher than 65 years the Dutch mortality rate is in excess of the parametric ICRP model.

Risk coefficients , $C_{additive}(A)$ and $C_{multiplicative}(A)$, which depend on age at exposure and dose and the parameters $\alpha$, $\beta$ and $\gamma$ are given in ICRP 60 for leukaemia and for other cancers. These risk coefficients have been estimated from the death rate in the Japanese survivors from the atomic bombing of Hiroshima and Nagasaki. For risk calculations in this section age and sex specific risk factors have been derived from the ICRP publication 60 (additive and multiplicative model) and from BEIR V (multiplicative model). A difference between the ICRP and the BEIR V models is that in the ICRP model only a distinction in two categories, i.e. leukaemia and "other cancers", is made whereas in the BEIR V model leukaemia, respiratory cancers, digestive cancers, breast cancer and "other cancers" are modelled separately.

### 6.1.3. COMPARISON OF THE ICRP AND BEIR MODELS FOR RISK ESTIMATION

Figure 6.4 provides information on the age and sex specific attributable life-time risk for three different models: the additive and multiplicative ICRP 60 models and the multiplicative BEIR V model. The discontinuities in the graphs occur due to limitations of the models: information on age specific risk coefficients is provided for intervals spanning several years. In the additive model at low ages there is a tendency for an increasing attributable life-time risk with age. Only at ages above 40 years there is a continuous decrease of the attributable life-time risk. Both multiplicative models show a tendency of decreasing risks with increasing age, the only exception is a slight increase in the risk coefficient for males between age 40 and age 60 in the BEIR V multiplicative risk model (Figure 6.4(c)). The lowest attributable risks are observed for the additive model, risk coefficients are considerably higher in the multiplicative models, especially at low ages. Age specific attributable life-time risk in the multiplicative models of ICRP 60 and BEIR V is of the same order of magnitude.

The risk of incidence of fatal cancers cannot without further due be compared with other fatal risks such as accidental death. Cancer death occurs a considerable number of years after the exposure to ionizing radiation whilst accidental death might occur immediately or relatively soon after the accident. The number of years of life lost will therefore differ in both cases. A quantity that considers the probability of an event (e.g. the incidence of a fatal malignancy) as well as the years of life lost is the (unconditional) average number of hours of life lost. The average number of hours of life lost after exposure to 1 mSv whole body dose is shown in Figure 6.5.

The attributable death rate after exposure to ionizing radiation at ages of 5, 25 and 45 years respectively is finally shown in Figure 6.6. Again the curves for the ICRP 60 and the BEIR V multiplicative risk models are similar (Figures 6.6(b) and 6.6(c)): in these graphs the attributable death rate becomes significant only at ages above 40 years, i.e. at ages at which the spontaneous cancer rate becomes significant (see Figure 6.3). The figures illustrate that, especially after exposure at young ages, the maximum attributable death rate occurs many years after the exposure. Risks are, as in figure 3.4, smaller for the additive model than for the multiplicative models.

BEIR V provides an estimation of the errors in the excess risk estimates for fatal tumour induction. The excess risk estimates and corresponding errors (90% confidence intervals) are shown in Figure 6.7. The figure demonstrates that large uncertainties are involved in the assessment of radiation induced fatal cancers.

### 6.1.4. ALTERNATIVE MODELLING AND DOSE ASSESSMENT

In the analysis of the Japanese survivors different time courses were observed for leukaemia and for solid cancers. The excess leukaemia cases appeared predominantly between 5 and 10 years

after exposure. For the other cancers the lateny periods are long. Since the excess rates for these tumours would be proportional to the age specific spontaneous rates, most excess cases would arise at old age. According to the ICRP and BEIR models, the factors of proportionality, and thus the attributable risks are assumed to be markedly higher for young ages at exposure. Kellerer and Barclay (10) argue that there is no firm support for this interpretation. They postulate that the Japanese data are equally well fitted by a model that does not assume a dependence of sensitivity on age at exposure but merely accounts for a dependence of the excess risks on dose and on age attained, here referred to as the "age attained model".

In its 1991 recommendations the ICRP (9) stated a fatal probability coefficient of 8 x $10^{-2}$ $Gy^{-1}$ for the sum of all malignancies in a population of both sexes and of working age at high dose and high dose rates of low-LET radiation. The corresponding value for the whole population including children is estimated as 10 x $10^{-2}$ $Gy^{-1}$. Risk estimates based on the "age attained model" are about half those obtained with the "age at exposure model". Kellerer and Barclay therefore conclude that one could abandon the controversial DDREF of 2, recommended by the ICRP, thus arriving at the same risk coefficients for fatal cancers at low doses is shown in Table 6.1.

Table 6.2. Estimated excess cancer risks in a UK population of all ages and both sexes from low-LET radiation as derived from Japanese A-bomb survivors (15)

| | *Fatal risk, $10^{-2}$ $Gy^{-1}$* | | | | |
|---|---|---|---|---|---|
| | *Based on lifetime projection of relative risks* | | *Based on excess deaths to date in Japanese survivors* | | |
| *Cancer type* | *High dose rate* | *Low dose rate* | *High dose rate* | *Low dose rate* | *ICRP 1977 adults only* |
| Leukaemia | 0.84 | 0.28 | 0.84 | 0.28 | 0.2 |
| Lung | 3.5 | 1.2 | 1.15 | 0.38 | 0.2 |
| Lower large intestine/colon | 1.1 | 0.37 | 0.38 | 0.13 | 0.1 |
| Stomach | 0.73 | 0.24 | 0.14 | 0.05 | 0.1 |
| Remainder other than breast, thyroid, liver, and bone | 5.0 | 1.63 | 0.565 | 0.20 | 0.2 |
| Total* | 12.9 | 4.5 | 3.93 | 1.4 | 1.25 |

* Includes breast, thyroid, liver and bone cancer risks derived from other than Japanese A-bomb survivors.

The DS86 dosimetry system as incorporated in the risk calculations of ICRP and BEIR, was at the time of its completion recognized as a clear improvement over the older dosimetry estimates. Ever since, however, a substantial number of additional neutron activation measurements have been performed in mineral and metal samples from Hiroshima (16). New findings on $^{36}Cl$ due to neutron activation extended the measurements range to more than 1.7 km from the epicenter. At this

distance, which is most relevant for risk evaluation, about 10 times more neutron activation was measured than estimated by DS86. Straume (17) indicated that the finding of high neutron dose would mean that the gamma-rays could actually be a factor of three less effective at doses below 0.5 Sv than assumed on the basis of DS86. These findings indicate that the dosimetry and risk evaluation of the Hiroshima survivors should be further investigated.

### 6.1.5 INFLUENCE OF RADIATION QUALITY

It is a well known fact that equal doses of radiation of different quality do not necessarily lead to the same level of biological damage. Radiobiological studies have shown that the relative biological effectiveness (RBE) is dependent on factors such as the nature of the detrimental effect, the absorbed dose, the dose rate and physiological conditions. For radiation protection applications emphasis should be placed on the risk for late stochastic effects after low-dose irradiation. For this condition the ICRP (8) introduced the radiation weighting factor $w_R$ which was slightly amended in the recent recommendation (9). For photons and electrons $w_R = 1$, for $\alpha$-particles $w_R = 20$ and for neutrons $w_R$ varies between 5 and 20, with the highest value in the energy range of 0.1 - 2.0 MeV.

For specific endpoints in annimals and in man RBE values in excess of 100 have been reported (3, 13). It is imporant to verify the type of reference radiation. When $\gamma$ rays would be used as the reference radiation, instead of X rays, appreciably higher RBE values would be derived, especially at low doses, since the RBE of $\gamma$ radiation can be appreciably smaller than unity. It should further be realized that higher RBE values of neutrons do not necessarily imply an extremely high risk factor per unit dose for this radiation modality but that they reflect more the low efficacy for tumour induction by low-LET radiation. This can be exemplified by the results of studies on mammary carcinogenesis after exposure to Cs-137 gamma radiation (5). The effect of fractionated irradiation with 120 fractions of 2.5 and 10 mGy (at intervals of 12 hours) was compared with that of a single acute exposure with doses of 0.3 and 1.2 Gy. A mathematical analysis of the relative excess hazard reveals quadratic dose-response curves without a significant linear component for the induction of carcinomas. On the basis of the dose-response curves for acute gamma irradiation in comparison to the linear dependency for 0.5 MeV neutrons (see Fig. 6.8) the RBE would approach infinity in the region of very low doses (below 0.3 Gy $\gamma$ rays). The recommended $w_R$ values (9) can be considered as a safe approach for radiation protection. It was recently suggested (7) that the $w_R$ of 5 for protons with energies in excess of 2 MeV and of 20 for alpha particles from radon progeny are both too high by at least a factor of two.

Animals studies have not yet provided an unambiguous answer about the influence of fractionation or protection of the dose administration on the carcinogenic effects of high-LET radiation and the epidemiological data from human exposures do not provide any reliable answer either. In

view of its importance for radiation protection this aspects will certainly need further investigation in a variety of animal species. For low-LET radiation, however, there is general consensus that the number of cancers induced per unit dose at low doses and low-dose rates, is reduced from that obtained at high doses and high-dose rates by a factor in the range of 2 - 10 (12). The ICRP (9) called this reduction factor the dose and dose-rate effectiveness factor (DDREF). For the studies on induction of mammary carcinoma in rats shown in Figure 6.8, a DDREF of 4.5 ± 2.5 can be derived. For high-LET radiation UNSCEAR (18) recommendeds a DDREF of 1.

## 6.1.6. CONCLUSION

The 1991 ICRP risk estimates (9) for radiation-induced cancer in man were based on the Japanese survivors considering the DS86 dosimetry, a longer observation period and the projection to life-time by a multiplicative model. The revision is mainly based on an increase in the number of solid malignancies from 135 observed in 1975 to a number of 260 in 1985 on a total population of more than 90,000 exposed persons. The consequencs of the increased risk estimates applied to the age distribution of the UK population are shown in Table 6.2 (15). Extrapolation of the observed radiation risk over the remainder of the lifes span yields appreciably higher risk factors than those derived solely from excess deaths observed in 1988 in the Japanese population. Stather et al. (14) applied a reduction factor close to 3 to derive risk estimates at low doses and low dose rates . Not all the values in the table originate from the epidemiology of the Japanese survivors. The risk factor at low dose rate based on excess deaths in Japan, does not differ significantly from the 1977 risk estimate of the ICRP (8). With reference to the assessment of the new risks the following objections can be expressed. Risk factors are based on the Japanese population; the medical studies revealed lower values. The lifetime projection with a multiplicative model is probably not applicable to all types of tumours. Alternative risk approaches, such as the age attained model yield fifty per cent lower risk factors. The findings of an appreciably reduced contribution from neutrons in Hiroshima imply a reduction by a factor of three in the risk factor for gamma rays. Consequently, the risk coefficient of $5 \times 10^{-2}$ $Sv^{-1}$ at low doses and low dose rates is probably an overestimation. The new dose limits for different categories of the population (9) can therefore be classified as conservatively safe recommendations.

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LEGENDS TO THE FIGURES

Figure 6.1. Dose-effect relations for the induction of fatal tumours used for extrapolation to low doses; (a) linear model and linear model with application of a dose and dose rate effectiveness factor (DDREF = 2); (b) linear-quadratic model; (c) linear model with threshold; (d) hormesis model. The points and error bars in the graphs are hypothetical.

Figure 6.2. Additive (a) and multiplicative (b) risk projection model. In the additive model, the annual excess risk due to irradiation remains constant with time after the latency period (i.e. 10 years) whereas in the multiplicative model the excess risk is the product of a constant factor with the age-dependent incidence rate of natural cancers.

Figure 6.3. The age specific spontaneous mortality rate of non leukaemia cancers (a) and leukaemia (b). Solid line: Dutch data, dotted line: parametric model of the ICRP.

Figure 6.4. The age and sex specific attributable life-time risk for fatal tumour induction after exposure to low doses. (a) Additive ICRP model with DDREF = 2; (b) multiplicative ICRP model with DDREF = 2; (c) multiplicative BEIR model.

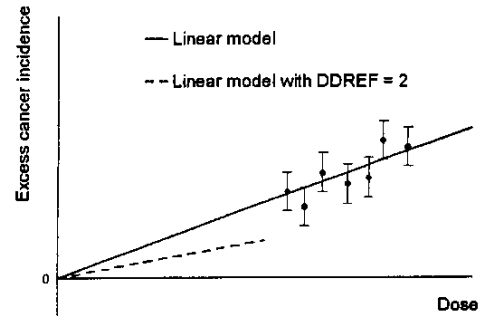
Figure 6.5. The age and sex specific (unconditional) average hours of life lost after exposure to low doses. (a) Additive ICRP model with DDREF = 2; (b) multiplicative ICRP risk model with DDREF = 2; (c) multiplicative BEIR model.

Figure 6.6. The age and sex specific attributable death probability rate after exposure to low doses at the age of 5, 25 and 45 years respectively. (a) Additive ICRP model with DDREF = 2; (b) multiplicative ICRP model with DDREF = 2; (c) multiplicative BEIR model. The attributable death rate is averaged for males and females.

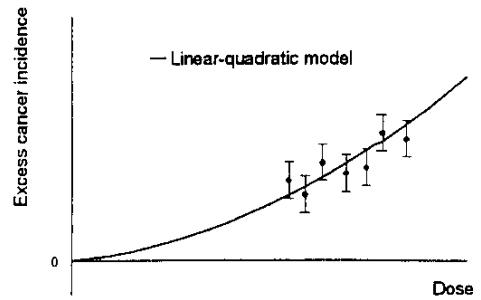
Figure 6.7. Excess risk estimates of BEIR V for fatal tumour induction with 90% confidence intervals. The figures correspond with an exposure of 100,000 males of each age to 100 mSv. Age ranges from 5 to 85 years in 10 year intervals.

Figure 6.8. Relative excess hazard as a function of the total absorbed dose for the induction of mammary carcinomas in WAG/Rij rats after irradiation with 0.5 MeV neutrons and Cs-137 gamma rays. Curve 1, fractionated irradiation: curve 2, single-dose irradiation at 17 weeks of age; curve 3, single-dose irradiation at 8 weeks of age (5).

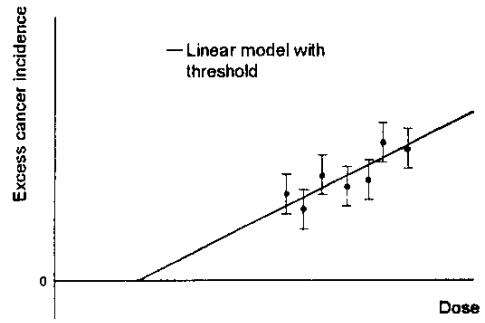
# Anhang/Appendix A




6.1a




6.1b

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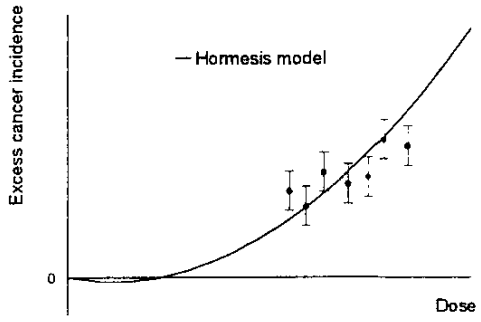
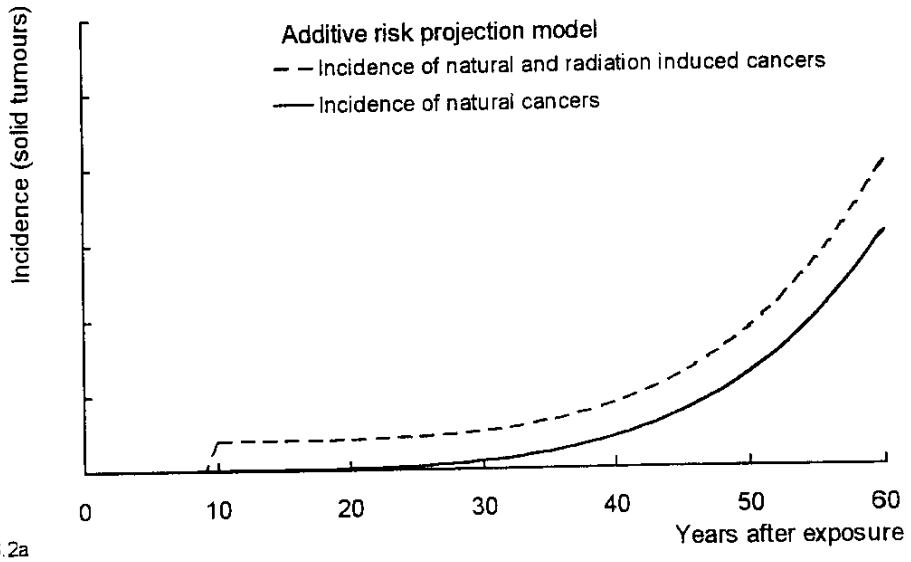
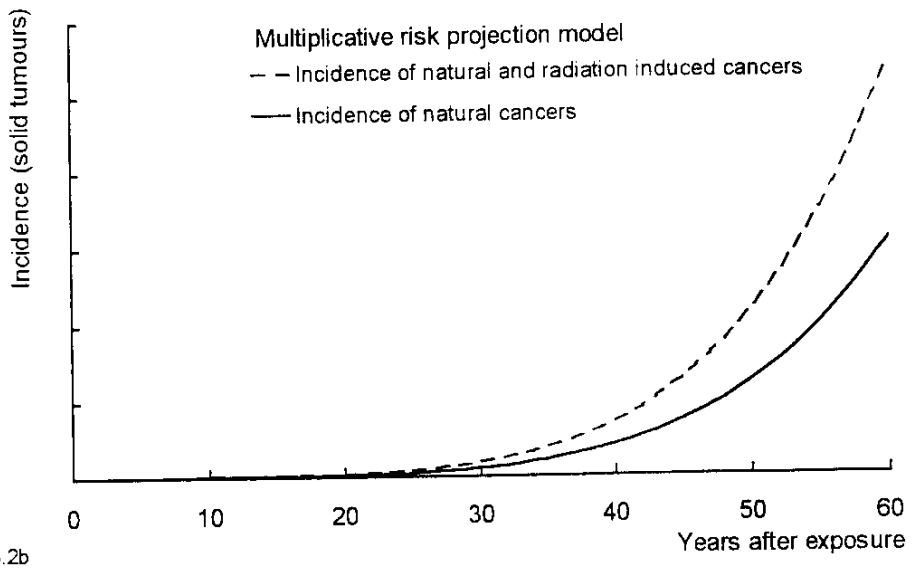


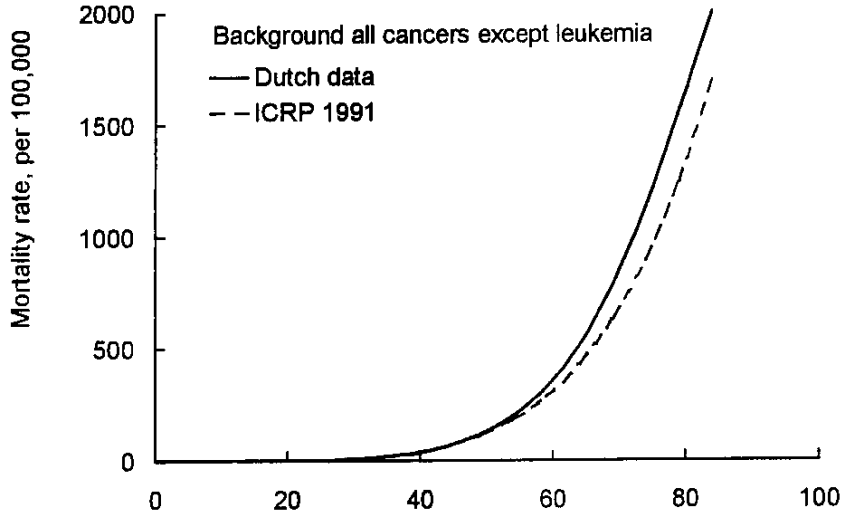

6.1d


Additive risk projection model
– – Incidence of natural and radiation induced cancers
—— Incidence of natural cancers
Incidence (solid tumours)
0
10
20
30
40
50
60
Years after exposure

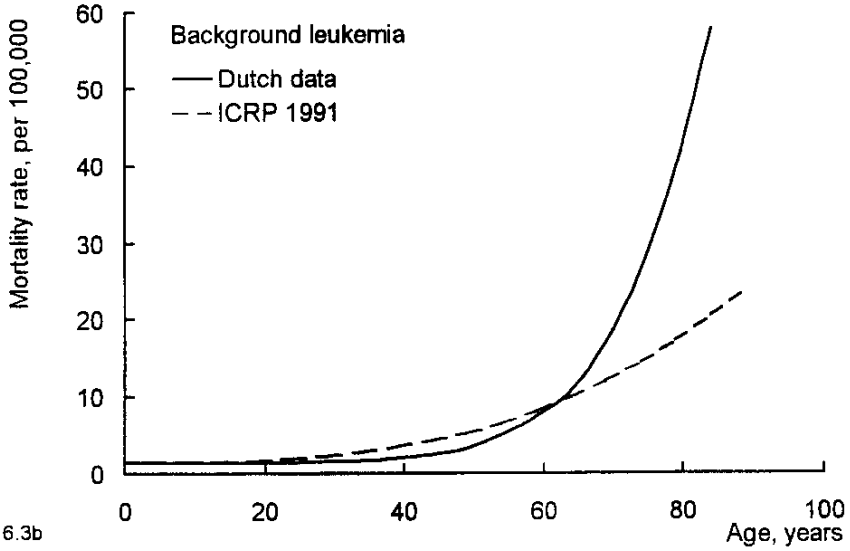
6.2a


Multiplicative risk projection model
– – Incidence of natural and radiation induced cancers
—— Incidence of natural cancers
Incidence (solid tumours)
0
10
20
30
40
50
60
Years after exposure

6.2b

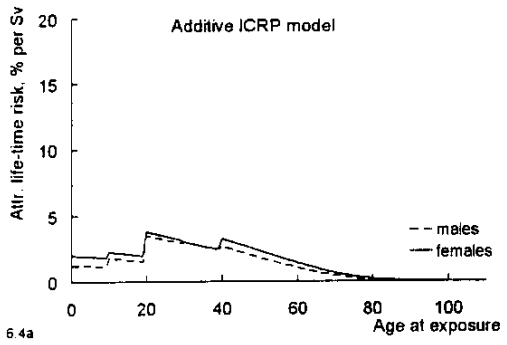

Background all cancers except leukemia
Dutch data
ICRP 1991
Mortality rate, per 100,000
2000
1500
1000
500
0
0
20
40
60
80
100
Age, years

6.3a

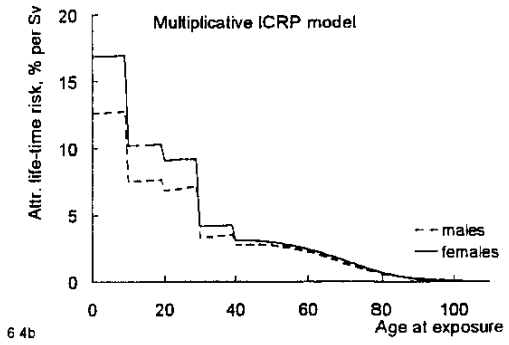

Background leukemia
Dutch data
ICRP 1991
Mortality rate, per 100,000
60
50
40
30
20
10
0
0
20
40
60
80
100
Age, years

6.3b

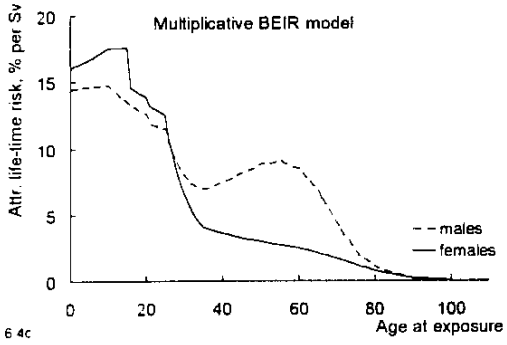
# Anhang/Appendix 4



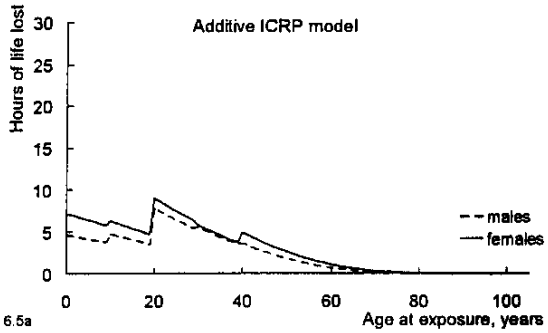
6.4a



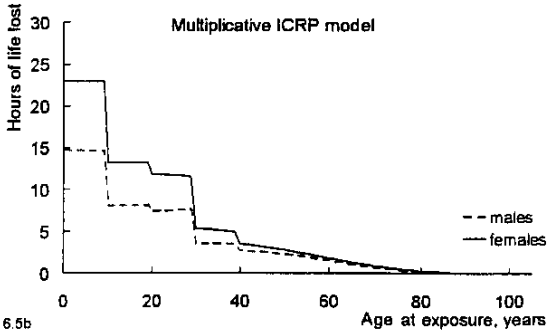
6.4b



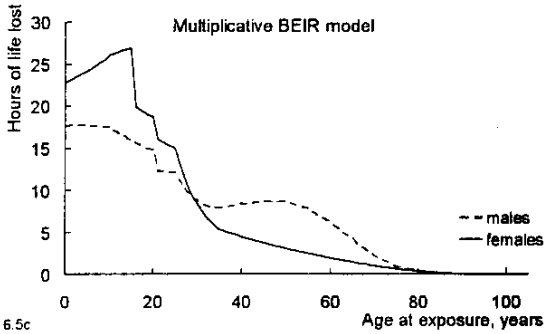
6.4c


Additive ICRP model
Hours of life lost
30
25
20
15
10
5
0
0
20
40
60
80
100
Age at exposure, years
males
females

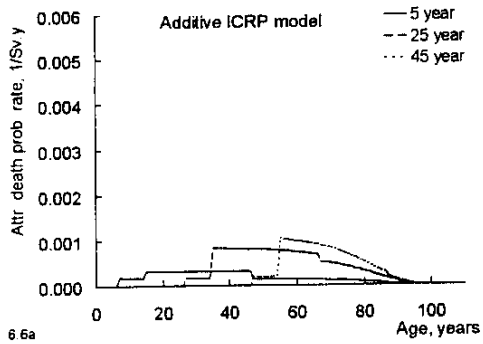
6.5a


Multiplicative ICRP model
Hours of life lost
30
25
20
15
10
5
0
0
20
40
60
80
100
Age at exposure, years
males
females

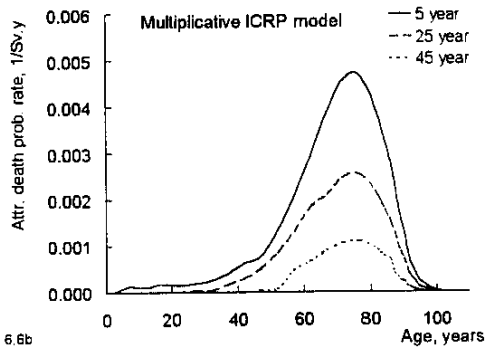
6.5b


Multiplicative BEIR model
Hours of life lost
30
25
20
15
10
5
0
0
20
40
60
80
100
Age at exposure, years
males
females

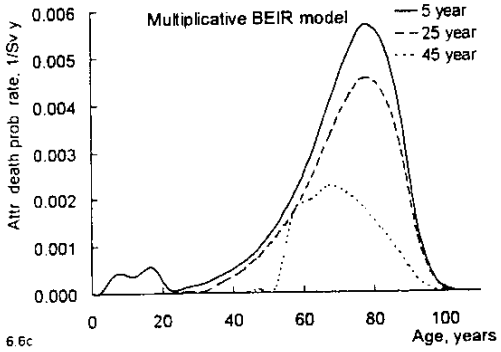
6.5c


Additive ICRP model
5 year
25 year
45 year
Attr. death prob. rate, 1/Sv.y
0.006
0.005
0.004
0.003
0.002
0.001
0.000
0
20
40
60
80
100
Age, years

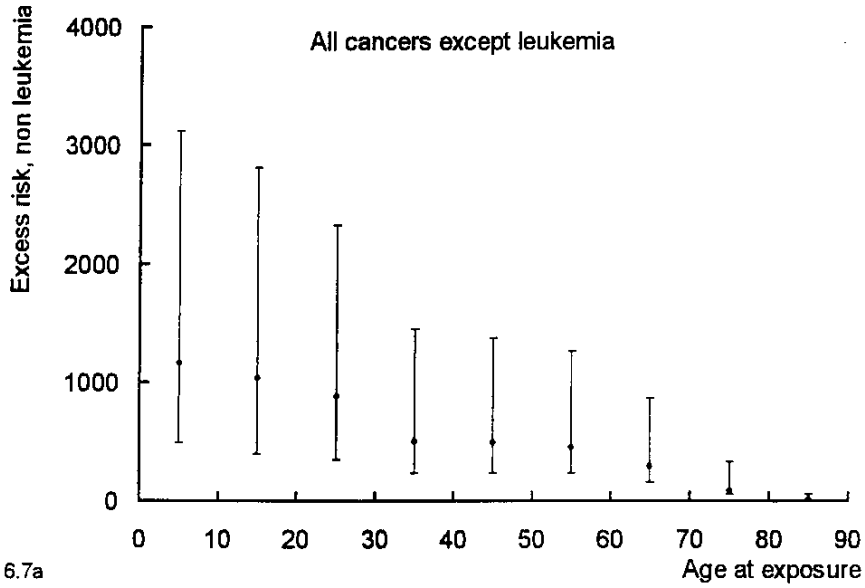
6.6a


Multiplicative ICRP model
5 year
25 year
45 year
Attr. death prob. rate, 1/Sv.y
0.006
0.005
0.004
0.003
0.002
0.001
0.000
0
20
40
60
80
100
Age, years

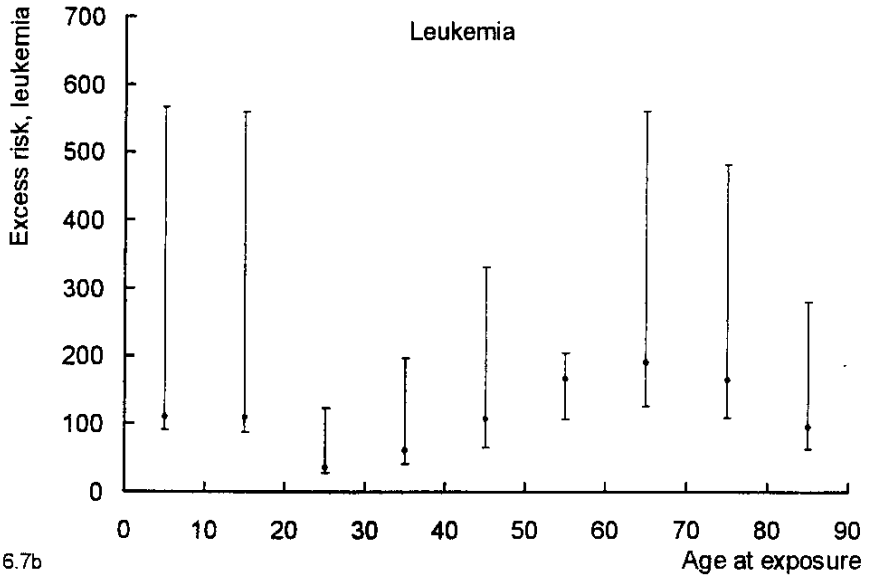
6.6b


Multiplicative BEIR model
5 year
25 year
45 year
Attr. death prob. rate, 1/Sv.y
0.006
0.005
0.004
0.003
0.002
0.001
0.000
0
20
40
60
80
100
Age, years

6.6c


All cancers except leukemia
Excess risk, non leukemia
4000
3000
2000
1000
0
0
10
20
30
40
50
60
70
80
90
Age at exposure

6.7a


Leukemia
Excess risk, leukemia
700
600
500
400
300
200
100
0
0
10
20
30
40
50
60
70
80
90
Age at exposure

6.7b

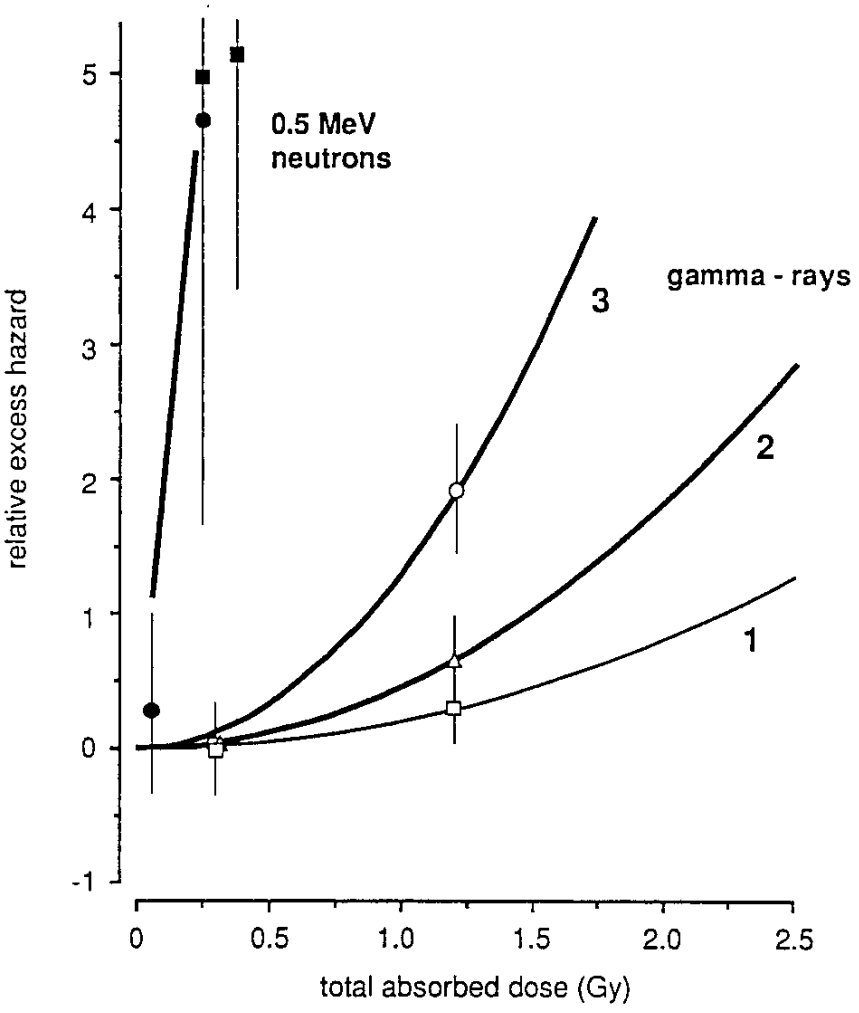

relative excess hazard
5
4
3
2
1
0
-1
0.5 MeV
neutrons
gamma - rays
3
2
1
0
0.5
1.0
1.5
2.0
2.5
total absorbed dose (Gy)

fig. 6.8