NRPB-M78

HEALTH-MARC: the health effects module in the methodology for assessing the radiological consequences of accidental releases

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ABSTRACT

A methodology for the assessment of the consequences of accidental releases of radionuclides from nuclear facilities has been developed. The methodology consists of a suite of computer programs which predict the transfer of activity from the point of release to the atmosphere through to the population. The suite of programs is entitled MARC - Methodology for Assessing Radiological Consequences. This report describes the health effects models currently incorporated into the module HEALTH-MARC. Models are included to estimate the early and late somatic effects in an exposed population and hereditary effects in their descendants.

The models in the MARC procedure for accident assessment are under continuing review. This memorandum records the models currently included in HEALTH-MARC; additional models and improved procedures will be incorporated, as appropriate, in the future

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As from 1 April 1978 NRPB adopted the International System of Units (SI). The relationship between the new SI units which are used in this report and the previous units are shown in the table below.

Quantity	New named unit and symbol	In other SI units	Old special unit and symbol	Conversion factor
Exposure	-	$C\ kg^{-1}$	röntgen (R)	$1\ C\ kg^{-1} \sim 3876$ R
Absorbed dose	gray (Gy)	$J\ kg^{-1}$	rad (rad)	1 Gy = 100 rad
Dose equivalent	sievert (Sv)	$J\ kg^{-1}$	rem (rem)	1 Sv = 100 rem
Activity	becquerel (Bq)	s^{-1}	curie (Ci)	$1\ Bq \sim 2.7 \times 10^{-11}$ Ci

1. INTRODUCTION

An integral part of any assessment of the risk presented by nuclear installations is the evaluation of the radiological consequences of potential accidental releases of radioactivity identified by safety analyses. Studies of the radiological consequences of accidental releases of radioactivity also provide an important input into the development of siting and design criteria and pre-planning of emergency arrangements. In order to undertake assessments of the radiological consequences of accidental releases of radioactivity to the atmosphere, a series of interlinked models designated MARC - Methodology for Assessing Radiological Consequences - has been developed. The overall methodology and detailed descriptions of some of the various modules making up the methodology have recently been published[1 - 7]. The methodology is shown schematically in Figure 1. This report describes the health effects models that have been incorporated into the module, HEALTH-MARC.

The health effects module, HEALTH-MARC, evaluates the incidence of each of the major types of health effect from the distribution of dose in the exposed population, after taking due account of the application of protective actions. The module is intended to be flexible. Options have been provided to change the values of parameters in the models from those used by default and, in some cases, a choice of models is offered.

The pathways of exposure considered in MARC are as follows:

(a) external β, γ dose from the cloud;
(b) external γ dose from the deposited activity;
(c) internal dose from activity inhaled during the passage of the cloud;
(d) internal dose from the inhalation of resuspended activity;
(e) internal dose from the consumption of foodstuffs contaminated by activity deposited from the cloud.

The estimation of the distribution of doses in the exposed population via these exposure pathways is described in DOSE-MARC[4], and PROT-MARC[7] describes the protective actions that may be invoked to limit the exposure.

Of the many deleterious effects which may result from exposure to ionising radiation following an accidental release of radioactivity, it is sufficient, for the purposes of risk analysis, to limit consideration to three major categories of biological effects. These are early and continuing somatic damage and late somatic damage in the exposed population, and hereditary effects in their descendants. Each category is considered in turn.

2. EARLY AND CONTINUING SOMATIC EFFECTS

Early and continuing somatic effects involve mortality and morbidity. They are usually associated with large acute doses of radiation and often occur within days or weeks of exposure, although sometimes a year or so may elapse. In general, there is a threshold dose below which any significant clinical effect is unlikely. For accidental releases from nuclear installations, the irradiation of

the bone marrow, GI tract and lung, would be the major causes of early death and each is considered in HEALTH-MARC.

Non-lethal radiation effects may also occur soon after exposure. These may require medical attention and some may result in a significant reduction in the quality of life and life shortening. They include lung fibrosis, prodromal vomiting, fertility impairment and pre-natal damage, skin damage, cataracts and hypothyroidism. The most serious effect for postulated releases of activity from reactor accidents is respiratory impairment from irradiation of the lung. This may have a much lower initial impact than early mortality, but may be of considerable importance in the longer term, because of the continuing health care that may be necessary and the existance of a large localised group of people suffering from a similar affliction. The incidence of lung morbidity is evaluated in HEALTH-MARC together with that of prodromal vomiting.

Dose-mortality and dose-morbidity relationships for the effects considered are discussed in more detail in the following sections. In using these relationships, it is essential that the doses to which the relationships are applied are compatible with the doses for which the dose-effect relationships have been derived. Account must also be taken of the relative biological effectiveness (RBE) of the radiation. Appropriate values of RBE for the incidence of early effects are discussed in more detail by Kelly et al[8] and Smith[9]. Values of 1 and 10, respectively, are adopted for β/γ and α radiation as default values in HEALTH-MARC, but other values may be substituted.

2.1 Death due to irradiation of the bone marrow

The human data on the incidence of death within 60 days of the exposure of the whole or a substantial part of the body to penetrating radiation have recently been reviewed[8,9]. The human data are sparse and dose-mortality relationships derived from animal experiments cannot be used directly to evaluate such relationships for man owing to marked species variation. The evaluation of a dose-mortality relationship for man must therefore be associated with considerable uncertainty and reflect a significant measure of personal judgement.

The dose-mortality relationships used in the US Reactor Safety Study (US-RSS)[10] and in a comparable German study[11] are shown in Figure 2. Two curves are shown in the case of the American study; the first assumes no treatment following exposure and the second assumes the provision of simple supportive medical treatment (eg, antibiotics, blood and fluid transfusions). The latter dose-mortality relationship was adopted in the US-RSS as the most appropriate in the circumstances. The provision of simple supportive medical treatment was also assumed in the formulation of the dose-mortality relationship in the German study. The evidence in favour of simple supportive medical treatments enhancing the survival probability is, however, limited; the quantitative data emerge from a few animal experiments but are supported by some clinical experience gained in the treatment of leukaemia and other malignancies.

The extent to which simple supportive treatment will influence the survival at varying levels of dose (in particular for the irradiation of a heterogeneous population, including the old, young and sick whose sensitivity may be greater) is a matter of continuing debate and awaits further resolution. The availability of such treatment is an additional consideration in the choice of the most appropriate dose-mortality relationship for large accidental releases where the number of people potentially exposed to doses in the lethal range may be large.

The default value for the LD_{50} for bone-marrow irradiation used in HEALTH-MARC is 4 Gy. This value may be changed on input. It lies midway between the likely bounds of uncertainty in the LD_{50}, which reviews of the data[8,9] indicate to be between about 3 and 5 Gy; furthermore it is similar to the dose-effect relationship, assuming no supportive medical treatment, as proposed in the US-RSS[10].

The slope of the dose-mortality curve is also uncertain and, again, the value chosen reflects a significant measurement of judgement; the similarity in the slope of the dose-mortality relationships observed in a wide range of animal experiments is helpful in this respect[9] but, again, there are reservations as to the applicability of such data to the exposure of a heterogeneous human population. The shape of the curve has been approximated in MARC by a multi-step linear function, which is illustrated in Figure 3, for an LD_{50} of 4 Gy.

One final point warranting further consideration is how protraction of exposure influences the probability of early death. The data and dose-mortality relationships shown in Figures 2 and 3 are for brief exposure (up to a few hours). In general, where the exposure is protracted (greater than a few hours or days) a higher level of dose will be required to lead to the same probability of death. The mechanisms by which protraction of exposure influences survival are well understood but little effort has been directed towards the development of a generally applicable quantitative framework that can be used to determine the influence of protraction for the wide variety of patterns of exposure that might be encountered in accidents. A relatively simple, robust and conservative procedure was formulated for use in the US Reactor Safety Study[10] and has been adopted without change in a number of similar studies[11,12]. The dose used in conjunction with the dose-mortality relationship is as follows,

$$\left\{\begin{array}{c}\text{Dose accumulated in}\\ \text{the first 7 days}\end{array}\right\} + \frac{1}{2} \times \left\{\begin{array}{c}\text{Dose accumulated from}\\ \text{day 8 to 30}\end{array}\right\}$$

This assumes conservatively that exposure accumulated in the first 7 days is equally effective as a single brief exposure, while that accumulated from day 8 to 30 is half as effective; moreover, exposure accumulated beyond 30 days is regarded as ineffective in this context. There is no strong radiobiological justification for this relationship; it merely represents an empirical judgement. The formulation was arrived at with knowledge of the likely variation of dose with time following reactor accident releases and recognition that doses

accumulated at dose rates of several tens of mGy d^{-1} would not significantly influence the survival probability. In general, the dose rates after 30 days to those individuals exposed in the lethal range (and who had not already accumulated sufficient dose to cause death) would be, at most, of this same order and typically be very much less. In practice, it is more likely that the exposure of such individuals, at least from external exposure, would be terminated by evacuation within a short time of the release. In such circumstances the question of the precision of the above formulation for the effect of protraction becomes somewhat academic. This formulation has been adopted in HEALTH-MARC, while recognising its simplicity and potential limitations.

2.2 Death due to irradiation of the lung

There are no human data on which to base estimates of dose-mortality relationships for varying patterns of irradiation of the human lung. Appropriate animal data must therefore be used. The most relevant animal data, obtained from experiments with dogs, have been reviewed[8] and a series of dose-mortality relationships derived for various temporal patterns of dose accumulation. The dose-mortality relationship is very dependent on the pattern of dose accumulation; the more rapid the rate of accumulation, the smaller the dose required to produce a given risk of death. The pattern of accumulation of dose and the corresponding mortality data are shown in Figure 4. The data for yttrium-90 and the combined data for strontium-90 and cerium-144 probably encompass the upper and lower extremes of possible dose-effect relationships for accidental releases of radioactivity that might be encountered from nuclear installations. Dose-effect relationships based on these data are also shown in Figure 4. The form of these relationships shown in the figure is the model adopted in HEALTH-MARC.

In any application of HEALTH-MARC, it is important to ensure that the dose-effect relationship adopted is compatible with the pattern of dose accumulation for the release being considered. In the US-RSS[10], a dose-effect relationship between those for yttrium-90 and yttrium-91 was chosen as the most appropriate for the releases considered in that study. For those accidental releases of radioactivity from reactors, where death due to irradiation of the lung is important compared with that due to irradiation of the bone marrow, it is likely that the pattern of dose accumulation would be more similar to that of yttrium-91. Consequently, the dose-effect relationship appropriate to this pattern of dose accumulation has been adopted as the default option in HEALTH-MARC, although this can, and moreover should, be changed, depending on the release being considered.

The above dose-mortality relationships were derived using the dose accumulated to 365 days. The dose used in conjunction with these dose-mortality relationships in HEALTH-MARC is therefore taken to be that accumulated in the first year.

2.3 Death due to irradiation of the GI tract

There are few human data on the incidence of death due to irradiation of the GI tract on which to base a dose-effect relationship. Reviews[8,9] of the relevant animal data, again obtained from experiments with dogs, have proposed the dose-effect relationship shown in Figure 5. It is a simple linear function and has been adopted in this form in HEALTH-MARC. The slope and threshold of this relationship have also been adopted as the default values in HEALTH-MARC, although alternative values may be substituted.

Death from irradiation of the GI tract would only be of importance in comparison with death from irradiation of the bone-marrow for releases of radioactivity which result in a large internal dose to the GI tract. In this case, because of the normal clearance processes that take place in the gut, the majority of the dose would be delivered within the first 7 days. The dose used in the dose-mortality relationship for GI tract irradiation is therefore that accumulated within 7 days.

2.4 Morbidity due to irradiation of the lung

A further biological effect which needs to be considered following lung irradiation is the incidence of lung damage, particularly fibrosis, which may not be fatal but which may have serious consequences for the individual. Reviews of the experimental data on the incidence of radiation-induced fibrosis have been carried out[8,10] and dose-effect relationships proposed. The model used in HEALTH-MARC is based on the simple linear function of Kelly et al[8], which is shown in Figure 6. The default values for the slope and threshold dose are also those proposed in that report[8]; it is recognised, however, that this relationship is based on limited data, much of it from animal experiments for which there is a lack of quantitative information on the degree of fibrosis and hence the degree of health impairment.

Studies in animals exposed to low-LET radiation have indicated that protraction of the dose reduces the degree of lung fibrosis. The effect of this protraction can be accounted for by use of a dose equal to the dose accumulated in the lung in the first 7 days plus one quarter of that accumulated between 7 days and 1 year[8]. This procedure is adopted in HEALTH-MARC. For α irradiation, the animal data suggest that protraction of the dose does not reduce the degree of fibrosis[8], and the dose accumulated in the lung in the first 5 years is used with the dose-morbidity relationship in HEALTH-MARC.

2.5 Prodromal vomiting

Following sufficiently large whole-body exposure, prodromal vomiting would occur and would be the cause of temporary discomfort. It is unlikely to recur or be the source of permanent injury, but the number of people affected would, in general, exceed the number that may experience other early effects, including early death and lung fibrosis. The data on the incidence of vomiting following exposure, among human patients undergoing treatment for cancer, have been

reviewed[10] and a dose-response relationship, based on the incidence within 48 hours, has been proposed. The model used in HEALTH-MARC approximates this relationship by a two-step linear function, which is shown in Figure 7. The default parameter values of the function are those shown, but alternative values may be specified.

Since the above dose-response relationship was based on the effects occurring within 2 days, it was assumed conservatively in the US-RSS[10] that the dose used in this relationship should be that accumulated within 2 days. This procedure has also been adopted in HEALTH-MARC.

2.6 Total effects

If each cause of early death was considered separately, the numbers of deaths could be overestimated, since the total probability of early death cannot exceed unity. Assuming that there are no synergistic effects between sub-lethal doses to several organs, the total probability of early death, P_{MT}, is given by

$$P_{MT} = P + (1 - P_1)P_2 + (1 - P_1)(1 - P_2)P_3$$

where P_1, P_2 and P_3 are the independent probabilities of death caused by irradiation of the bone marrow, lung and GI tract, respectively.

Similarly, the number of cases of lung morbidity will be influenced by the incidence of death. If f_1 is the independent probability of lung morbidity, the actual probability f_L is given by

$$f_L = (1 - P_{MT})\, f_1$$

Prodromal vomiting, on the other hand, is likely to occur before the death of those sufficiently exposed; the number of cases of prodromal vomiting has therefore been assumed to be unaffected by the incidence of mortality.

3. LATE SOMATIC EFFECTS

The most important late somatic effect to be considered following an accidental release of radioactivity is the increased incidence of fatal and non-fatal cancer, and both are included in HEALTH-MARC. Fatal cancer is used to denote those cancers for which the cure rate is low, whereas non-fatal cancer is used to indicate those cancers for which the cure rate is high, but for which there may be physical or psychological reasons for the quality of life being reduced.

Various reviews have been made of the increased incidence of cancer in irradiated populations[10,13,14,16]. Much of the human data derives from exposures at organ doses of between about 1 - 10 Gy. It has often conservatively been assumed that the risk at lower doses may be estimated by extrapolating the observed incidence at high doses linearly down to zero. The results of experiments with animals would, however, indicate that a dose-response relationship with an additional dose-squared term may be more realistic for low-LET radiation. Both linear and linear-quadratic models are incorporated into

HEALTH-MARC, and examples of these models and of the pure quadratic model (an extreme of the linear-quadratic) are shown in Figure 8. Values of the coefficients used in the different models for the types of cancer considered are discussed in more detail in the following sections.

As with early effects, the relative biological effectiveness of the radiation must also be taken into account. The default values used in HEALTH-MARC are 1 and 20 for β/γ and α radiation, respectively, as proposed by Clarke and Smith[14], although alternative values may be specified.

3.1 Linear model

A review of estimates of the increased incidence of cancer in irradiated populations has been made by Clarke and Smith[14] and risk coefficients proposed for the risk of excess cancers following irradiation of the whole body and important body organs. These risk coefficients are shown in Table 1. The values are those used by default in HEALTH-MARC, but alternative values may be substituted. Following irradiation the consequent health effects will appear over a prolonged period. The risk coefficients in Table 1 are applicable to a cohort which will live long enough for the total risk to be expressed. For calculating realistic expectations of health effects in irradiated populations, Clarke and Smith[14] have proposed a log-normal distribution of the incidence of cancer with time after irradiation, to allow for the observed delay between dose and effect. These distributions of the incidence of cancer with time after irradiation are shown in Figure 9. For the incidence of leukaemia, the proposed time distribution has a median time of appearance of 12.5 years and a standard deviation of 0.8; for the incidence of solid tumours it has a median time of appearance of 25 years and a standard deviation of 0.4. In HEALTH-MARC there is the option to use the risk coefficient of Table 1 unmodified, or to take account of this proposed time distribution as described below.

Because of the time distribution of the incidence of cancer, the time integrated risk coefficients, r_s, for the incidence of cancer in an exposed population with an age distribution typical of that of the UK, will be less than those given in Table 1, r, by a modifying factor, M, ie,

$$r_s = Mr$$

$$\text{where } M = \frac{\sum_{A} N(A) \sum_{i>A} (P_{i-1}(A) - P_i(A)) \int_0^{i-A} \phi(t)\, dt}{\sum_{A} N(A)} \qquad \ldots.(1)$$

where $N(A)$ is the number of individuals of age A in the population,

$P_i(A)$ is the probability of individuals of age A surviving to age i in the absence of the dose,

$\phi(t)$ is the normalised time distribution of the incidence of cancer, as shown in Figure 9.

This modifying factor will be applicable to the external γ-dose from the passing cloud and the external γ-dose from deposited activity, since in these cases it can be assumed that the age distribution of the exposed population is that of the population as a whole. In the case of external radiation from deposited activity, which may continue over extended periods, the modifying factor is strictly only correct if the age distribution of the exposed population group is assumed to remain essentially constant with time, although the individuals making up the population group will be continually changing due to births, deaths and movements. The number of cancers estimated in this way will include those appearing in the initially exposed population as well as those subsequently exposed.

In the case of internal irradiation from inhaled and ingested activity, however, further modifying factors are required. Some nuclides taken into the body may have an extremely long residence time; in such cases the dose to some organs may be delivered over tens of years following intake, a period which is comparable with the appearance of late effects and with life expectancy. A dose delivered at some time T years after intake will be delivered to a population group that has aged by T years, and the appropriate modifying factor will be

$$M(T) = \frac{\sum_{A} N(A) \sum_{i>A+T} (P_{i-1}(A+T) - P_i(A+T)) \int_0^{i-A-T} \phi(t)dt}{\sum_{A} N(A)}$$

A simplified procedure has been adopted in HEALTH-MARC for the evaluation of this expression. The dose accumulated within the first year after intake has been assumed to be delivered at the time of intake. The dose accumulated in subsequent 10-year periods has been conservatively assumed to be delivered at the beginning of that period, and a modifying factor appropriate to the beginning of the period applied. The periods considered and corresponding modifying factors are given in Table 2. These are based on the age distribution and life expectancy of the UK population in 1977[15]. The modifying factors are particular to the model chosen to represent the time of appearance of cancers, and different values are given for solid tumours and leukaemia. Their sensitivity to the pattern of appearance is unlikely to be large, at least for patterns that are consistent with the existing data on the incidence of radiation-induced cancers in man.

3.2 Non-linear models

Both the linear-quadratic model and the pure quadratic model are incorporated into HEALTH-MARC. In the linear-quadratic model, the increase in the

risk of cancer, R, due to a dose, D, is given by

$$R = \alpha D + \beta D^2$$

In the pure quadratic model, they are related by the expression

$$R = qD^2$$

The quadratic model may be thought of as one extreme of the linear-quadratic model with $\alpha = 0$, but has been included separately. Either model may be used instead of the linear model to estimate the increase in the incidence of cancer in an irradiated population.

To use these models, appropriate values of the parameters α and β, for the linear-quadratic model, and q, for the pure quadratic model, need to be specified for each of the organs listed in Table 1. If the risk coefficients, r, of Table 1 were based on observations of increased incidence of cancer at a unique dose, D_o, for each organ, this process would be relatively straightforward, since the risk given by the three models could be equated at this dose, ie,

$$\alpha D_o + \beta D_o^2 = qD_o^2 = rD_o$$

This equation would determine q and provide one constraint on the values of α and β. The remaining problem for the linear-quadratic model would then be of the relative magnitudes of α and β. In reality, however, the risk coefficients are based on an increased incidence of cancer, with large uncertainties, from exposure over a wide range of doses. Although some estimates of these parameter values have been made[16], the above procedure involves a significant measure of personal scientific judgement.

In addition, the use of non-linear dose-response models for late effects introduces other difficulties which remain unresolved. These include non-uniform irradiation of particular organs and protraction of exposure. Because of these difficulties, simplifications have been incorporated into the non-linear dose-response models currently included in HEALTH-MARC. No account is taken of the time of appearance of cancer following irradiation; instead, it is assumed conservatively that all the risk is expressed in the exposed population.

Any application of the non-linear models in HEALTH-MARC is, therefore, likely to involve many uncertainties, and the models are, as a result, deliberately simplistic in nature at this stage. Sensitivity analysis using these simple models[17] will be used as a first step in identifying the areas where further refinement may be required.

3.3 Total numbers of cancers

If the risk of death from cancer for each exposed organ were considered separately, the incidence of fatal cancer from large releases of activity could be significantly overestimated, because the probability of death from cancer cannot exceed unity. The actual probability of death from a radiation-induced cancer, C_T, where the independent probability of death from cancer in the nth organ is C_n, is given by

$$C_T = C_1 + (1-C_1)C_2 + \ldots\ldots\ldots + (1-C_1)(1-C_2)\ldots\ldots(1-C_{n-1})\,C_n$$

This expression applies where no other factors contribute to death. For releases of activity which give rise to early death, the actual probability of death from cancer will be further reduced due to prior depletion of the population by radiation-induced early deaths. In this case the probability of death from cancer will be

$$(1-P_{MT})C_T$$

where P_{MT} is the probability of early death. Similarly the actual probability of a particular non-fatal cancer has been assumed to be

$$(1-P_{MT})C_{NF}$$

where C_{NF} is the independent probability of a particular non-fatal cancer. This expression neglects the possible effect of prior depletion of the population by death from cancer.

4. HEREDITARY EFFECTS

The final important category of health effects considered in HEALTH-MARC is that of hereditary effects. Unlike somatic effects, which appear in the exposed individuals, hereditary effects manifest themselves in the descendants of these individuals. The serious hereditary disorders considered include those due to both gene mutations and chromosome anomalies. These will appear in succeeding generations with about half occurring in the first two generations. Only the total numbers of hereditary effects that occur in all subsequent generations are, however, evaluated in HEALTH-MARC.

There are no human data on the incidence of radiation-induced hereditary effects; estimates of their increased incidence are based entirely on animal studies. Although most estimates of the risk of hereditary effects have been based on a linear model[10,14], there is again evidence in favour of a linear-quadratic model for low-LET radiation, and both types of model are included in HEALTH-MARC.

As with other health effects, the relative biological effectiveness of the radiation must be taken into account. The values used by default in HEALTH-MARC are 1 and 20 for β/γ and α radiation respectively, as recommended by Clarke and Smith[14], although alternative values may be used.

4.1 Linear model

Estimates of the incidence of hereditary effects following a dose of radiation have been reviewed by Clarke and Smith[14]; a risk coefficient of 2×10^{-2} Gy^{-1} (low-LET) for gonad irradiation is recommended for individuals who are irradiated before the start of reproductive age and subsequently have the average number of children. This figure includes the risk of hereditary effects in all generations, with about half occurring in the first two generations. This risk coefficient is that adopted by default in the linear model of HEALTH-MARC.

The population averaged risk of hereditary effects, r_s, can then be expressed as

$$r_s = Mr$$

where r is the risk coefficient for individuals who have the full opportunity to produce children, and the modifying factor, M, is given by

$$M = \frac{\sum_A N(A) \sum_{i>A} C_i \, P_i(A)}{\left(\sum_A N(A)\right)\left(\sum_A C_A\right)} \qquad \ldots\ldots (2)$$

where $N(A)$ is the number of people in the standard population of age A,

$P_i(A)$ is the probability of surviving to age i, given survival to age A,

C_i is the probability of becoming a parent when age i.

The denominator in the above expression gives the total number of children that would be born if everyone in that population had the average number of children.

As for the incidence of cancer, this modifying factor applies to the external γ dose from the passing cloud, and the external γ dose from deposited activity, where the age distribution of the exposed population can be assumed to be typical of that in the UK. In the case of internal irradiation from inhaled and ingested activity, however, the dose to the gonads may be delivered over an extended period depending on the characteristics of the radionuclides inhaled. A dose delivered at some later time after intake will have a reduced effect due to ageing of the population and an overall reduction in child expectancy. For a dose delivered T years after intake, the modifying factor becomes

$$M(T) = \frac{\sum_A N(A) \sum_{i>A+T} C_i \, P_i(A+T)}{\left(\sum_A N(A)\right)\left(\sum_A C_A\right)}$$

A similar procedure to that used to evaluate the modifying factor for the incidence of cancer has been adopted in evaluating the above expression. The dose delivered within the first year has been assumed to be delivered at the time of intake, and the dose delivered in subsequent ten-year periods has been assumed to be delivered at the beginning of that period. The modifying factors used and corresponding periods are given in Table 3. The factors are based on the age distribution and child expectancy of the UK population[15].

4.2 Non-linear models

As with the estimation of cancers, both linear-quadratic and pure quadratic models are included in HEALTH-MARC to estimate the increased incidence of hereditary effects. The problems described in the application of non-linear models to the estimation of the increased incidence of cancer apply equally to hereditary effects.

It has been assumed, for simplicity, in the use of these models, that all of the dose is delivered to a population with an age distribution and child

expectancy typical of that of the UK. Thus, the risk estimated using these models is modified by the factor M given in equation (2). For accidental releases typical of those postulated for nuclear reactors, a large proportion of the dose to the gonads is from external radiation, and this assumption is, therefore, not unreasonable. Other simplifying aspects of these models will be the subject of further analyses.

4.3 Total numbers of effects

In evaluating the incidence of hereditary effects, account is taken of their possible reduction by radiation-induced early deaths. No account has, however, been taken of the possible influence of early morbidity or late effects on child expectancy. For late effects, in particular, this is likely to be minimal.

5. SUMMARY

The MARC suite of modules has been developed to provide a comprehensive methodology for the evaluation of the radiological consequences of accidental releases of radioactivity. This report has described the models currently incorporated into HEALTH-MARC, the health effects module in the methodology. Options on the values of parameters in the models adopted and on the choice of models have been provided throughout HEALTH-MARC to enable the user to select those most appropriate for any intended application. A number of simplifications are currently included in some areas of HEALTH-MARC; these are the subject of continuing analyses, and refinements will be made, where appropriate, in the future.

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Table 1

Risk coefficients of the increased incidence of cancer for use in the linear model

Organ	Risk coefficient (Gy^{-1}) low-LET
a) Fatal cancers[1]	
Breast	2.5×10^{-3}
Red bone marrow	2×10^{-3}
Lung	2×10^{-3}
Thyroid	5×10^{-4}
Bone surface	5×10^{-4}
Liver	1×10^{-3}
LLI	1×10^{-3}
Remainder tissues	3×10^{-3}
Skin	1×10^{-4}
b) Non-fatal cancers[1]	
Thyroid	1×10^{-2}
Skin	1×10^{-2}
Breast	2.5×10^{-3}
c) Hereditary effects[2]	
Gonads	2×10^{-2}

Notes:

1. The risk coefficients are applicable to a population which will live long enough for the total risk to be expressed.
2. The risk coefficient is applicable to individuals irradiated before reproductive age and who subsequently have the average number of children.

Table 2

Modifying factors to be applied to the risk coefficients for the incidence of cancer for doses delivered in various periods following the intake of activity

	Period, y							
	0-1	1-10	11-20	21-30	31-40	41-50	51-60	61-70
Leukaemia	0.76	0.75	0.62	0.49	0.37	0.25	0.15	0.06
Solid tumours	0.63	0.61	0.49	0.36	0.24	0.13	0.05	0.01

Table 3

Modifying factors to be applied to the risk coefficients for the incidence of hereditary effects in all future generations for doses delivered in various periods following the intake of activity

	Period, y							
	0-1	1-10	11-20	21-30	31-40	41-50	51-60	61-70
Modifying factor	0.40	0.39	0.26	0.11	0.02	2×10^{-3}	2×10^{-4}	0.0

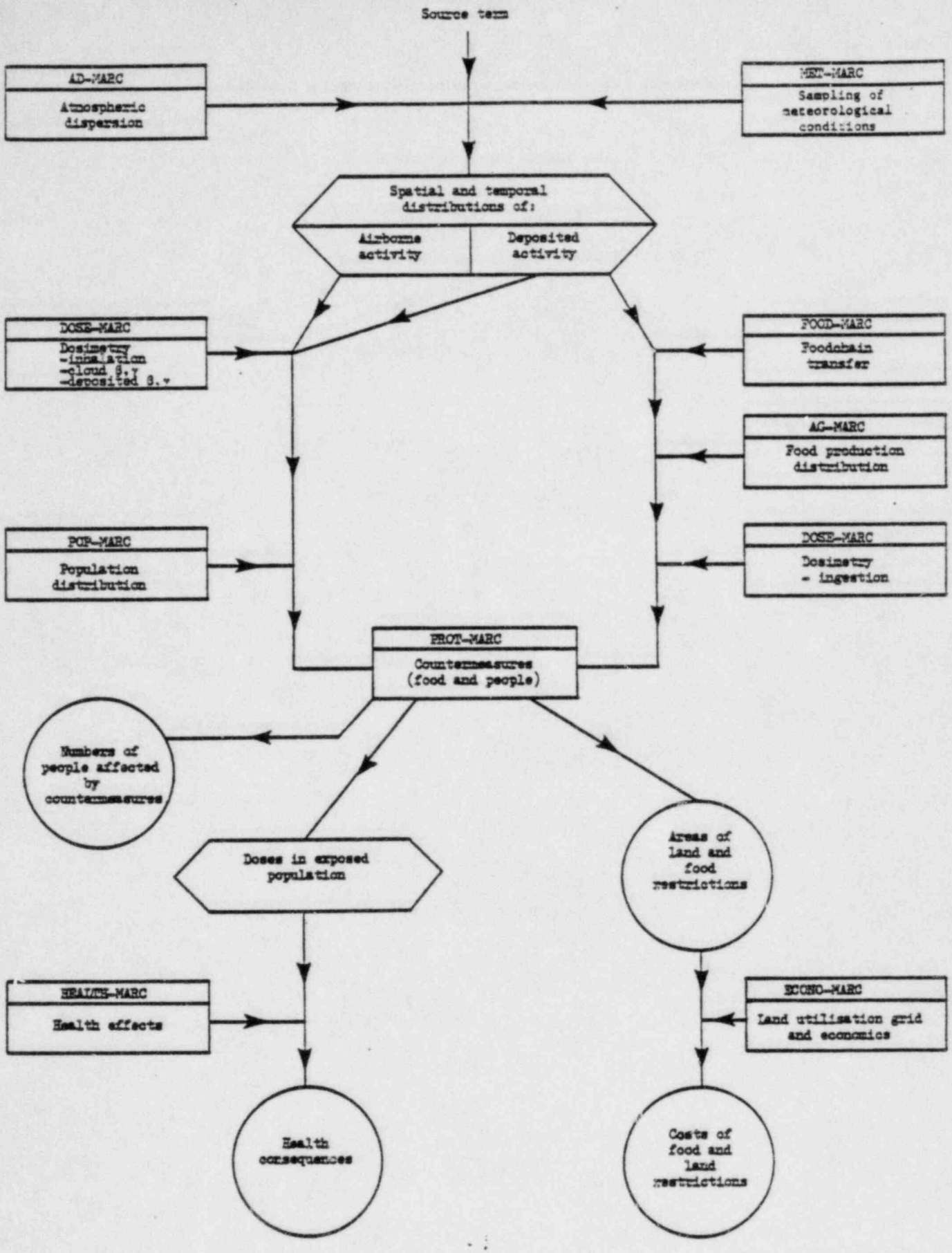

Figure 1 Schematic diagram of the major components of MARC

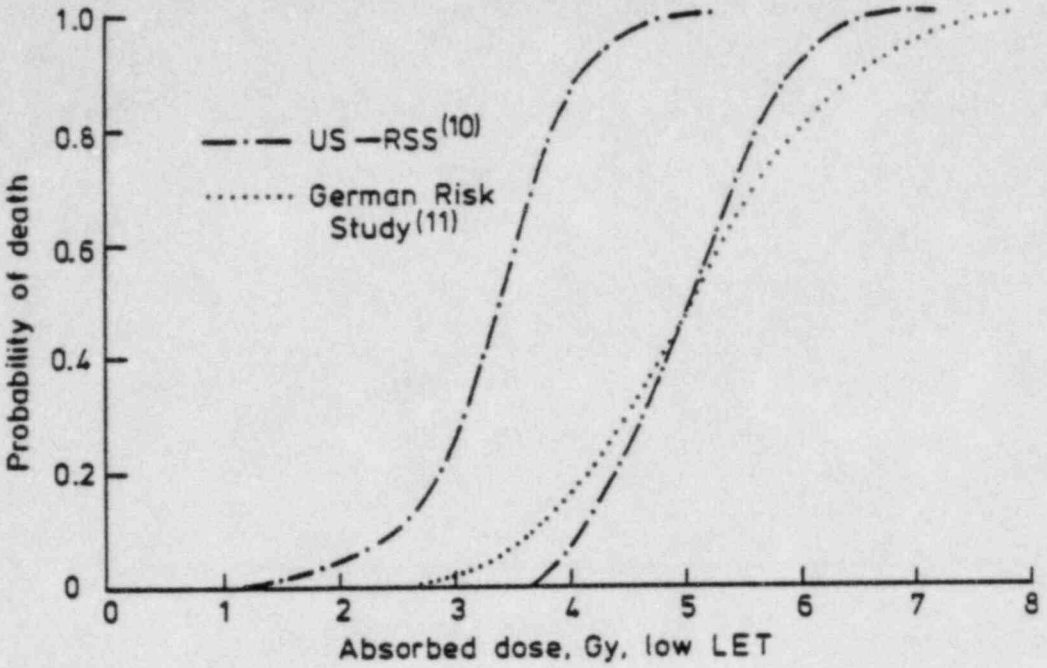

Figure 2 Examples of mortality relationships for bone marrow irradiation used in other studies

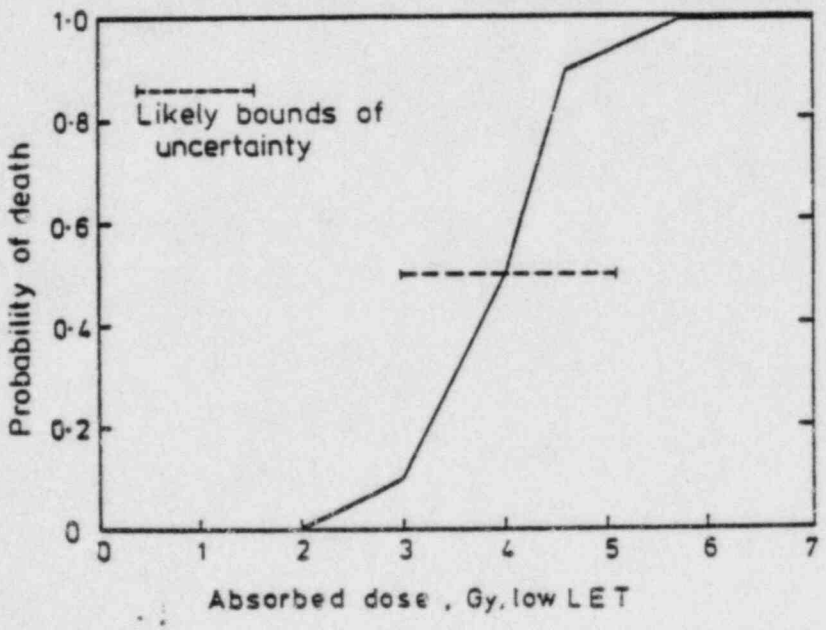

Figure 3 Dose mortality relationship for bone marrow irradiation

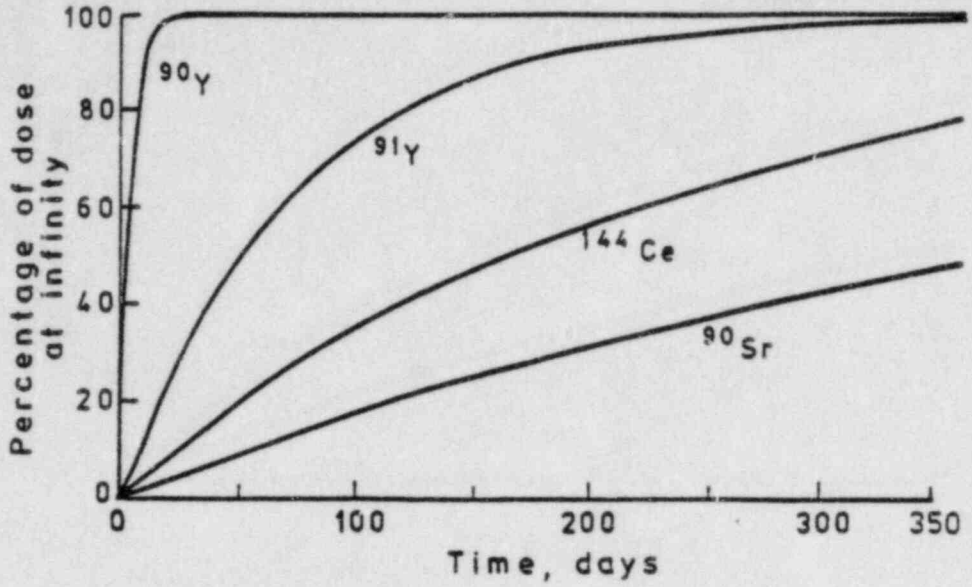

Figure 4a Pattern of accumulation of lung dose for dogs exposed to various inhaled insoluble radioactive aerosols

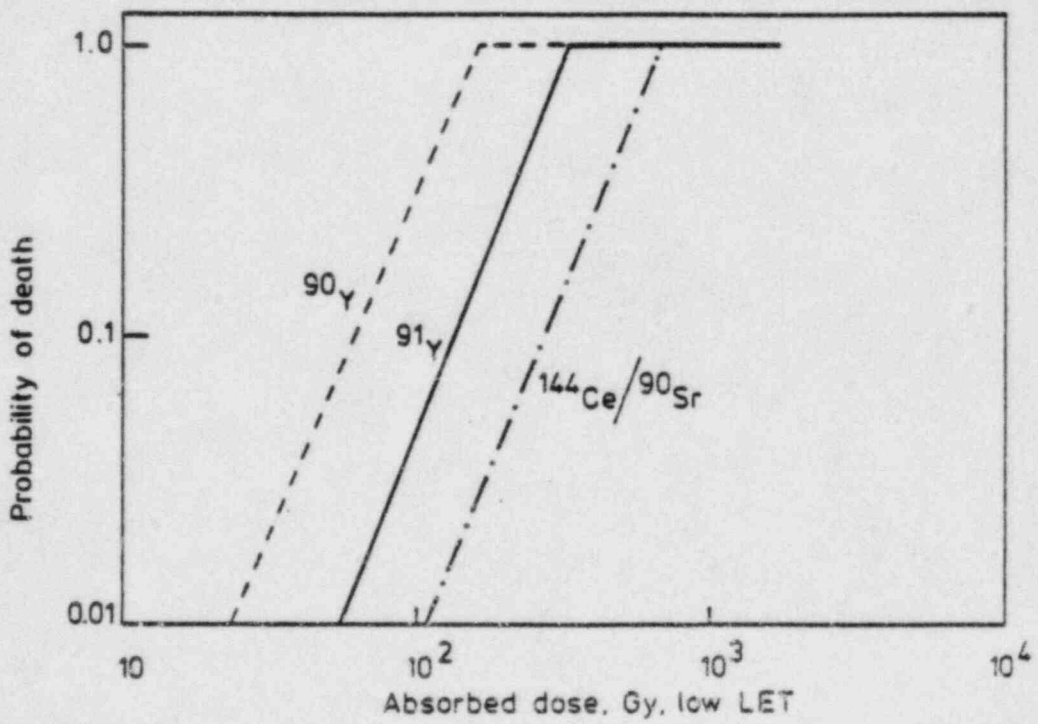

Figure 4b Dose mortality relationship for lung irradiation

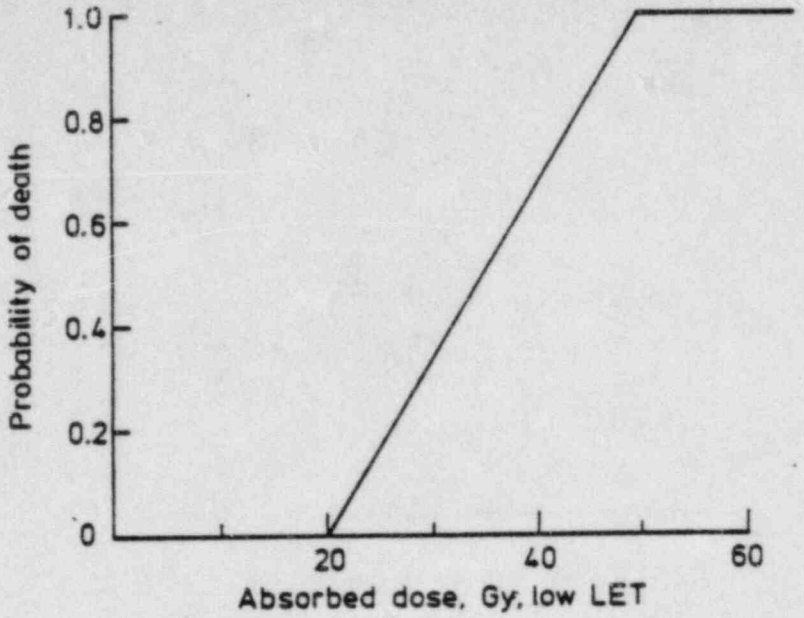

Figure 5 Dose mortality relationship for G I tract irradiation

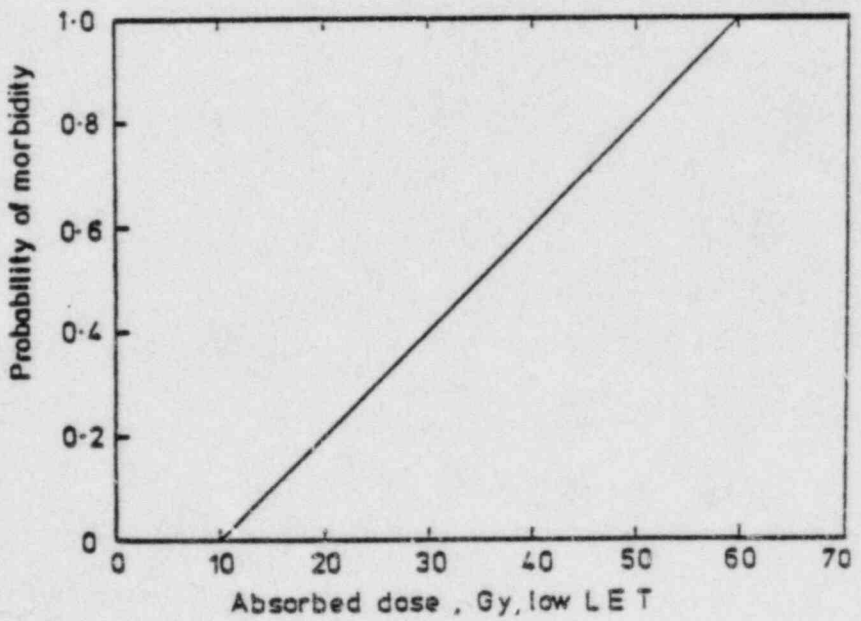

Figure 6 Dose morbidity relationship for lung irradiation

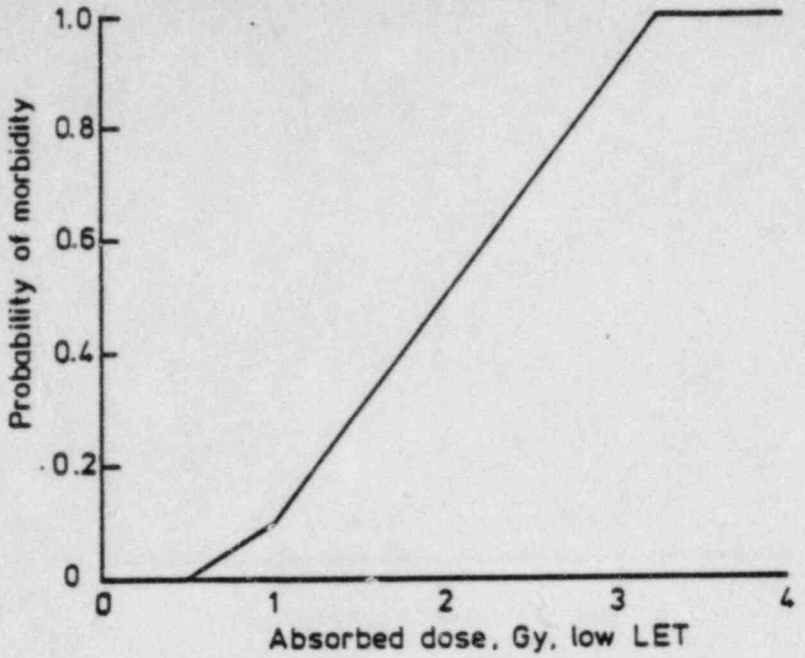

Figure 7 Dose morbidity relationship for prodromal vomiting

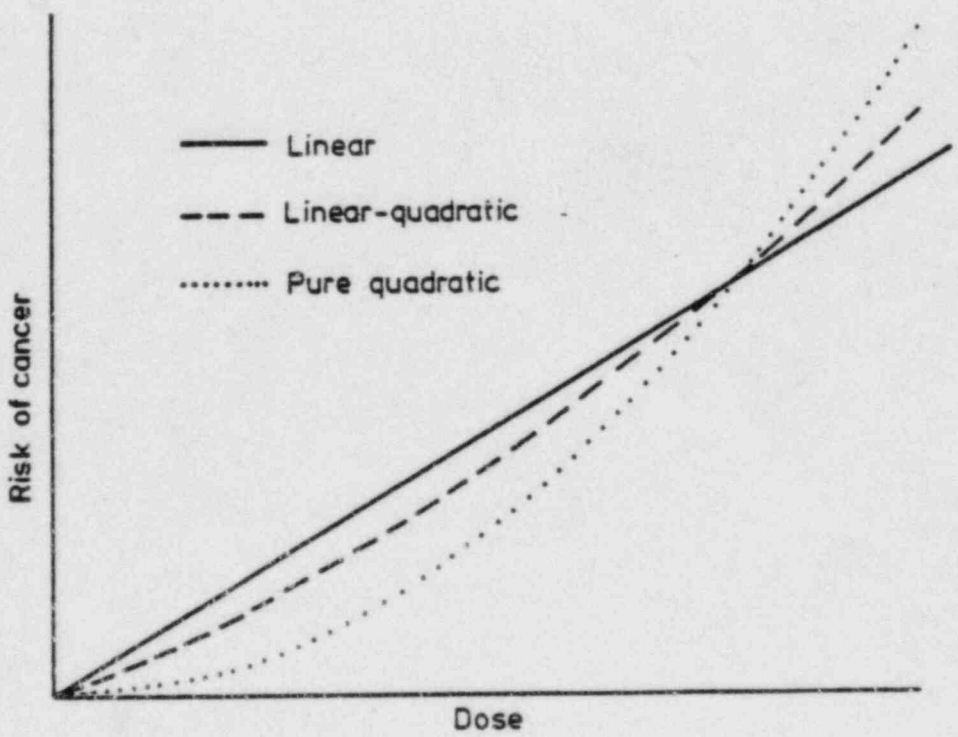

Figure 8 Examples of the models for estimating the increased incidence of cancer

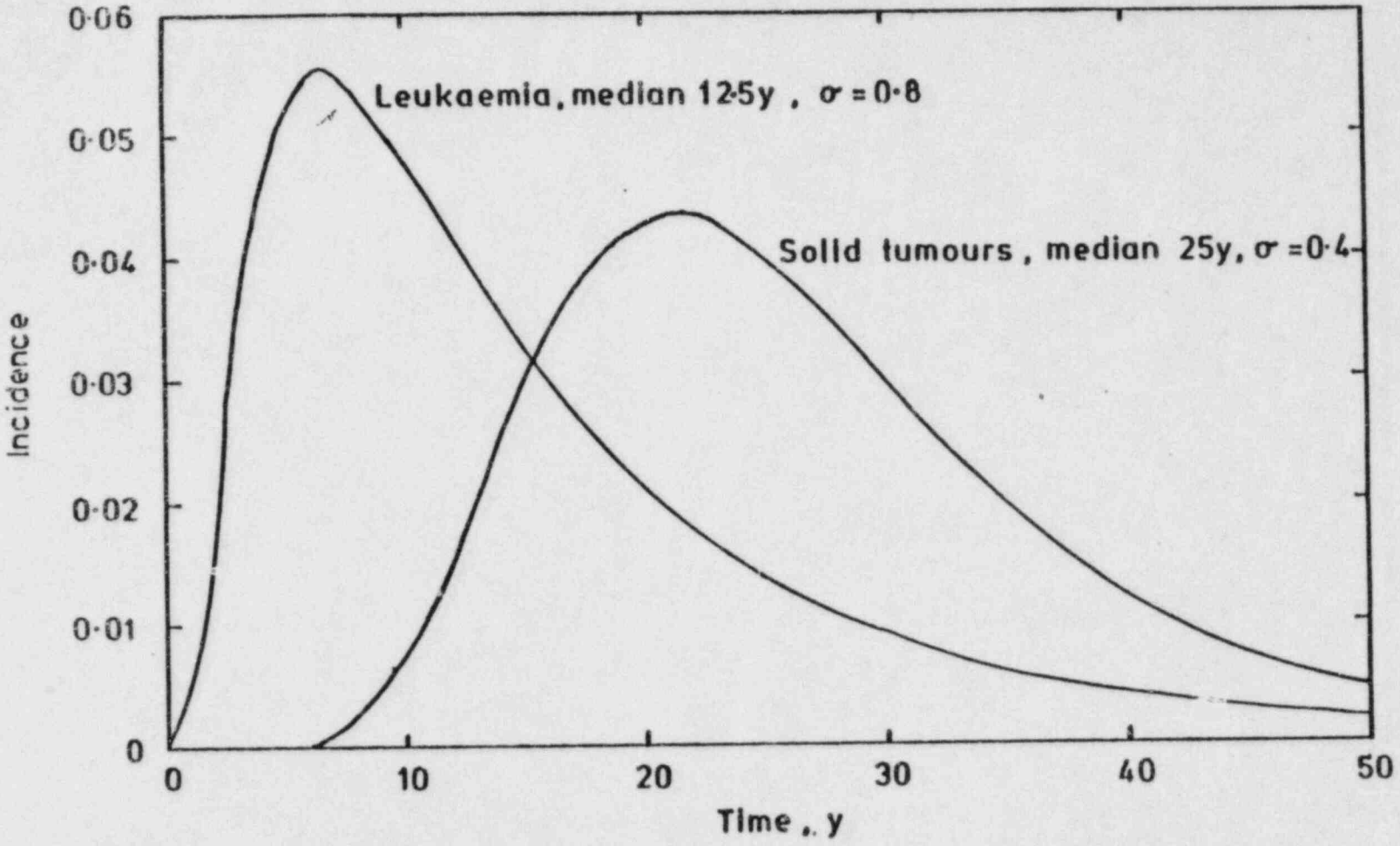

Figure 9 Incidence of cancers following an increment of irradiation