Risk Models in NIOSH-IREP

This document is part of the "View Model Details" help files provided with NIOSH-IREP, the computer code developed for the Division of Compensation Analysis and Support (DCAS) of the National Institute of Occupational Safety and Health (NIOSH), in support of their effort to implement a program created by the Energy Employees Occupational Illness Compensation Program Act of 2000 (EEOICPA) which provides compensation and medical benefits for nuclear weapons workers who may have developed certain work-related illnesses.

The probability of cancer causation (or assigned share) depends on the risk of cancer attributable to radiation exposure. In NIOSH-IREP, the radiation risk for a given cancer is estimated using risk models for cancer incidence developed by the National Cancer Institute (NCI; Land et al. 2002; 2003). With few exceptions, the risk models are obtained from the Life Span Studies (LSS) of Japanese atomic bomb survivors from Hiroshima and Nagasaki. The risk model for the incidence of thyroid cancer is obtained from an analysis of a pooled data set that contains information from the Japanese A-bomb survivors and from other studies of medical exposures to X rays (Ron et al. 1995). The risk model for lung cancer from exposure to radon is obtained from an analysis of uranium miners data (RECA 1996). This document presents a summary of the risk models applied for each cancer type (Table 1), as well as a brief description of the characteristics of each risk model.

The dose–response relationship was found to be linear for all cancer types other than for leukemia and for lung cancer from exposure to radon. The coefficient of the linear dose-response is referred to as the "*ERR/Sv*". The *ERR/Sv* coefficients obtained from analysis of the A-bomb survivors (who were exposed mainly to high doses of gamma radiation delivered at high dose rates) are adjusted when applied for an individual who was chronically exposed to low doses and low dose rates, by using a dose and dose rate reduction factor (DDREF). For leukemia, a linear-quadratic dose response is used for acute exposure to low-LET radiation (see details below). For exposure to radon, the risk of lung cancer is estimated using exposure expressed in working level months (WLM), rather than using radiation dose to the lung tissue (in Sv). Other adjustments that account for the reduced risk during the minimum latency period of the disease, for the transfer of risk from the Japanese to the U.S. population, and for the effectiveness of different radiation types are applied to the *ERR/Sv* coefficients as described by Land et al. (2003) and Kocher et al. (2008).

The *ERR/Sv* for each cancer type has an uncertainty obtained from the statistical analysis of the epidemiological data. This type of uncertainty is called "statistical uncertainty," and is given by a profile likelihood function. In some cases, the likelihood function is described by an analytical probability distribution function (e.g., lognormal). In other cases, the likelihood function is described as a cumulative probability function using a given set of percentiles (ranging from 0.25% to 99.75%). Cubic-spline interpolation between the given percentiles was used to obtain values for any percentile of the cumulative distribution (Press et al. 1992).

The cancer-specific risk models used in NIOSH-IREP can be divided into four broad categories:

1) Group 1 cancers: The *ERR/Sv* depends on both age at exposure and age at time of diagnosis of disease (attained age). For a given age at exposure and attained age, the uncertainty in the *ERR/Sv* is described by a lognormal distribution with a known geometric mean and geometric standard deviation.

2) Group 2 cancers: The *ERR/Sv* depends on both age at exposure and attained age, but the structure of the model describing this dependency is different from the model for Group 1. For a given age at exposure and attained age, the *ERR/Sv* is obtained using an age-independent *ERR/Sv* multiplied by an age-dependent modifying factor. The uncertainties in both the age-independent *ERR/Sv* and in the age-dependent modifying factor are used to obtain the uncertainty in the desired *ERR/Sv*.

3) Group 3 cancers: The *ERR/Sv* for cancers in this group does not depend on age at exposure or attained age. That is, for a given cancer, the *ERR/Sv* and its uncertainty is constant for all ages at exposure and attained ages.

4) Other cancers: Each cancer type in this group has a unique risk model and is treated individually.

Table 1 Risk models used in NIOSH-IREP

Cancer type[a]		Risk model used in calculation	
Name	ICD-9 code[a]	Category	ICD-9 code[b]
Oral Cavity and Pharynx	140-149	Group 2	140-149
Esophagus	150	Group 2	150
Stomach	151	Group 1, 2	151
Colon	153	Group 2	153
Rectum	154	Group 2	154
All digestive	150-159	Group 1	150-159
Liver	155.0	Group 1	155.0
Gallbladder	155.1,156	Group 2	155.1,156
Pancreas	157	Group 2	157
Trachea, bronchus and lung	162	Other cancers	162
Other respiratory (nasal cavity, larynx, other)	160, 161, 163-165	Group 2	160, 161, 163-165
Bone	170	Group 2	170-172, 175, 190, 194-199
Connective tissue	171	Group 2	170-172, 175, 190, 194-199
Malignant melanoma	172	Other cancers	173 (basal cell)
Non-melanoma (Basal cell carcinoma)	173 (basal cell)	Other cancers	173 (basal cell)
Non-melanoma (Squamous cell carcinoma)	173 (squamous cell)	Group 3	173 (squamous cell)
Breast-female	174	Group 1	174
Breast-male	175	Group 1	174
Ovary	183	Group 2	183
All female genital (except ovary)	179-182, 184	Group 3	179-182, 184
All male genitalia	185-187	Group 2	185-187
Bladder	188	Group 2	188
Kidney (+etc)	189	Group 2	188-189
Eye	190	Group 2	170-172, 175, 190, 194-199
Nervous system	191, 192	Group 2	191, 192
Thyroid	193	Other cancers	193
Other endocrine glands	194	Group 2	170-172, 175, 190, 194-199
Other and ill-defined sites	195	Group 2	170-172, 175, 190, 194-199
Lymphoma and Multiple Myeloma	200-203	Group 2	200-203
Leukemia (all except chronic lymphocytic)	204-208, less 204.1	Other cancers	204-208, less 204.1
Acute lymphocytic leukemia	204.0	Other cancers	204.0
Chronic lymphocytic leukemia	204.1	Group 2	200-203
Acute myelogenous leukemia	205.0	Other cancers	205.0
Chronic myelogenous leukemia	205.1	Other cancers	205.1

[a] Represents the cancer for which the probability of causation is estimated, using the risk models listed in the last two columns.
[b] Represents the cancer types used to derive the risk model from the epidemiological data. The risk models marked in red are obtained from a set of cancers different from the cancer for which the probability of causation is estimated.

GROUP 1 CANCERS

All digestive cancers (male and female), stomach (female), liver (male and female), and breast (male and female)

The risk models in NIOSH-IREP use a general formulation described by the following equation (Land et al. 2003; page 23; equation IV.D.1):

$$ERR = D \times \alpha \times \exp[\beta \times s + \gamma \times f(e) + \delta \times g(a)] = D \times ERR/Sv(s,e,a)$$

or, equivalently, for $\alpha > 0$:

$$ERR = D \times \exp[\ln(\alpha) + \beta \times s + \gamma \times f(e) + \delta \times g(a)] = D \times ERR/Sv(s,e,a)$$

where

- D = radiation equivalent dose (Sv) to the organ responsible for induction of cancer
- $\alpha, \beta, \gamma, \delta$ = parameters of the model associated with each modifier
- s = sex modifier ($s = 1$ for females and $s = 0$ for males)
- e = age at exposure
- a = attained age
- $f(e)$ = min[max(-15,e-30),0]
- $g(a)$ = min[ln(a/50),0]

The cancer sites in Group 1 have relatively large numbers of cases, exhibit strong evidence of dependencies with age, and strong correlations occur among various model parameters. For these cancers, a lognormal approximation of the *ERR/Sv* was found. In the logarithmic scale, the sex-specific *ERR/Sv* (at exposure age e and attained age a) is assumed to be normally distributed with the mean and variance of logarithms defined by the equations below.

$$\text{mean} = \ln(\alpha) + \gamma \times f(e) + \delta \times g(a) \text{ and}$$

$$\text{variance} = \text{var}(\ln \alpha) + 2 \times \text{cov}(\ln \alpha,\gamma) \times f(e) + 2 \times \text{cov}(\ln \alpha, \delta) \times g(a) + \text{var}(\gamma) \times f(e)^2 + 2 \times \text{cov}(\gamma,\delta) \times f(e) \times g(a) + \text{var}(\delta) \times g(a)^2$$

For a given attained age, the *ERR/Sv* for these cancer types decreases exponentially between ages at exposure of 15 and 30 and is constant outside this interval. Similarly, for a given age at exposure, the *ERR/Sv* decreases linearly with attained age, up to attained age 50, after which it remains constant. The age-at-exposure and attained-age dependencies are modeled by $f(e)$ and $g(a)$.

The *ERR/Sv* developed for female breast cancer is applied to estimate risk of breast cancer for males.

GROUP 2 CANCERS

Oral cavity and pharynx (male and female), esophagus (male and female), stomach (male), colon (male and female), rectum (male and female), gallbladder (male and female), pancreas (male and female), other respiratory except lung (male and female), ovary (female), male genital (male), bladder (male and female), urinary organs less bladder (male and female), nervous system (male and female), lymphoma and multiple myeloma (male and female), chronic lymphocytic leukemia (male and female), other solid cancers (male and female).

The risk model for cancers in this group has the same general formulation as that for Group 1 cancers. However, cancers in Group 2 have a relatively lower number of cases, and no significant correlation was found between the α parameters and the age-at-exposure and attained-age parameters γ and δ. Since, the α parameters are practically independent of γ and δ, the *ERR/Sv* (at exposure age e and attained age a) is approximated by the following equation:

$$ERR/Sv(e,a) = ERR/Sv(s,e=30,a=50) \times F(e,a)$$

where the age-dependent modifying factor *F(e,a)* is assumed to be independent of the cancer site and is described by a lognormal distribution with a mean of logarithms and a variance of logarithms given by:

$$\text{mean} = \gamma \times f(e) + \delta \times g(a)$$

$$\text{variance} = \text{var}(\gamma) \times f(e)^2 + 2 \times \text{cov}(\gamma,\delta) \times f(e) \times g(a) + \text{var}(\delta) \times g(a)^2$$

In addition to the individual solid tumors listed above, a set of risk coefficients was derived for all other solid tumors as a group. The model for this group of cancers is called the "residual cancers" risk model, and it is applied to estimate the risk from exposure to radiation for the following cancer types: bone, connective tissues, eye, endocrine glands other than thyroid, and other ill-defined sites (ICD-9 code = 195). For these cancer types, the data were insufficient to derive individual cancer models.

GROUP 3 CANCERS

Female genitalia (less ovary), non-melanoma (squamous cell carcinoma; male and female), NIOSH-IREP lung model (male and female)

The risk model for these cancers shows no age dependency (i.e., $\gamma = \delta = 0$).

$$ERR = D \times ERR/Sv$$

The *ERR/Sv* for these cancers apply for all ages at exposure and all attained ages. For squamous cell carcinoma, same *ERR/Sv* are applied for both males and females (i.e., no gender dependency was observed). For NIOSH-IREP lung model the *ERR/Sv* depends on gender. See discussion on Group 4 cancers for more details about squamous cell carcinoma model and NIOSH-IREP lung model.

OTHER CANCERS (GROUP 4)

Malignant melanoma (male and female) and non-melanoma of skin (male and female)

The dose-response relationship for skin cancer is based on the Japanese A-bomb survivor cohort data. The only cancer subtype for which a significant dose response was obtained is basal cell skin carcinoma, which is a form of non-melanoma. The dose-response relationship for basal cell carcinoma shows only an age-at-exposure dependency. No gender or attained-age dependency was observed.

$$ERR = D \times \alpha \times \exp[\gamma \times f(e)] = D \times ERR/Sv(e)$$

where:

$$f(e) = \min[\max(-30, e-40), 0]$$

($f(e) = -30$ for $e \leq 10$, $= e-40$ for $10 < e < 40$, and $= 0$ for $e \geq 40$, which means that risk is constant with age at exposure for ages <10 and >40 and decreases exponentially between ages 10 and 40).

The dose-response for basal cell carcinoma is also applied to malignant melanoma cases. For non-melanoma skin cancers other than basal cell carcinoma (a group of cancers dominated by squamous cell carcinoma), no age dependency could be determined. A single profile for *ERR/Sv* is applied for all ages at exposure, all attained ages and both genders. Thus, in essence, the risk model for squamous cell carcinoma is a Group 3-type risk model.

Thyroid

The dose-response relationship for thyroid cancer was obtained by re-analyzing the pooled data of seven studies which include the studies of Japanese A-bomb survivors and of patients exposed to X rays (Ron et al. 1995). The dose-response from this analysis shows a strong dependency on age at exposure (e), and includes exposures in adulthood. Sex-dependency is not statistically significant,

but only marginally (p = 0.07). The *ERR/Sv* decreases exponentially with age at exposure from birth until age 50, after which the risk is considered constant with age at exposure.

$$ERR = D \times \alpha \times \exp[\gamma \times e] = D \times ERR/Sv(e)$$

The *ERR/Sv* for any given age at exposure (e) is described by a lognormal distribution with a geometric mean (*GM*) and a geometric standard deviation (*GSD*) provided by Land et al. (2003).

Leukemia

NIOSH-IREP estimates the assigned share/probability of causation for the following types of leukemia: acute myeloid leukemia (AML), chronic myeloid leukemia (CML), acute lymphocytic leukemia (ALL), and "leukemia" (as a group).

Despite its name, chronic lymphocytic leukemia (CLL) appears etiologically and clinically to be a lymphoma and is considered to be a form of Non-Hodgkins Lymphoma (NHL) by the U.S. National Cancer Institute (NCI 2009) and the World Health Organization (Harris et al. 1999). NIOSH has reviewed the current literature and epidemiological data on the relationship between radiation and CLL and proposed that CLL be considered as a radiogenic cancer under the Energy Employees Occupational Illness Compensation Program Act of 2000 (EEOICPA). For estimating probability of causation, a risk model for CLL similar to the IREP risk model for lymphoma and multiple myeloma has been developed (Trabalka and Apostoaei, 2010). The CLL risk model has passed the review process and has been incorporated in NIOSH-IREP as a Group 2 cancer, as of 01/24/2013.

The dose-response relationship for leukemia (as a group) is based on all cases of leukemia observed in the Japanese A-bomb survivor cohort, other than chronic lymphocytic leukemia (CLL). A linear quadratic dose-response is used to estimate risk of leukemia from exposure to acute low-LET doses of radiation (Land et al. 2003; page 27, equation IV.D.4).

$$ERR = (D + D^2) \times \alpha \times \exp(\beta \times e + \gamma \times t + \delta \times e \times t);$$

where

- D = radiation dose (Sv)
- $\alpha, \beta, \gamma, \delta$ = parameters of the model associated with each modifier
- e = age at exposure
- t = time since exposure

A linear dose response is used in all other exposure situations (i.e., high-LET and low-LET radiation delivered at low dose rates).

$$ERR = D \times \alpha \times \exp(\beta \times e + \gamma \times t + \delta \times e \times t)$$

The dose response for leukemia does not depend on gender. The model parameters α, β, γ, and δ are obtained from statistical analysis of the data. The parameter α corresponds to the excess relative risk for age at exposure $e = 0$, and time since exposure $t = 0$, given that $D + D^2 = 1$ (for acute exposures to low-LET radiation), or the dose is $D = 1$ Sv (for all other exposure situations). For a

given time since exposure, the risk decreases exponentially with increasing age at exposure up to age 55, and is constant after wards. For a given age at exposure, the risk decreases with time since exposure until 50 years after exposure[1], and is constant afterwards.

In addition to all types of leukemia as a group, individual types of leukemia were studied using the same dose response model.

Acute Myeloid Leukemia (AML)

The risk of Acute Myeloid Leukemia from exposure to radiation decreases exponentially with time since exposure (t) until 50 years after exposure, and is constant afterwards. No dependency of the risk on age at exposure (β and $\delta = 0$) or gender was observed for AML in the A-bomb survivor cohort.

Chronic Myeloid Leukemia (CML)

The risk of Chronic Myeloid Leukemia from exposure to radiation decreases exponentially with time since exposure (t) until 50 years after exposure and is constant afterwards. The risk for males decreases more rapidly than the risk for females. No dependency on age at exposure (e; β and $\delta = 0$) was determined for CML.

Acute Lymphocytic Leukemia (ALL)

When exposure occurs before age 20, the risk of Acute Lymphocytic Leukemia from exposure to radiation decreases exponentially with time since exposure (t) until 50 years after exposure, and is constant afterwards. No time-since-exposure dependency was identified for exposure in adulthood. Also, no gender dependency was determined for ALL.

Lung cancer from exposures to sources of radiation other than radon

As of 2/28/06, NIOSH-IREP automatically runs lung cancer claims under both the NIOSH-IREP risk model and the risk model used by the National Institutes of Health in their version of IREP (NIH-IREP). The 99th percentiles of PC produced by the two models are compared and the higher PC is reported. The higher PC value determines the claim outcome. The lower PC value is also reported, but only for information purposes and it has no bearing on the claim outcome. The NIOSH-IREP and NIH-IREP risk models for lung cancer are described below. The main differences between these two models are summarized in Table 2.

[1] This statement ignores the increase in risk with increasing time since exposure during the latency period of leukemia. The latency period for leukemia may last between 1 and 5 years.

Table 2 Summary of differences between NIOSH-IREP and NIH-IREP versions of lung cancer risk models

Areas	NIOSH-IREP	NIH-IREP
Risk model	• No age dependency (parameters γ and $\delta \equiv 0$ under all conditions)	• Age dependency ($\gamma \neq \delta \neq 0$ under all conditions)
Parameter uncertainty	• Sex-specific likelihood profiles for parameter α (given as an average over all smoking categories)	• Sex-adjusted likelihood profiles for parameter α (given for never smokers) • Sex (β) and age (γ,δ) parameters are correlated
Transfer to the US population	• Trapezoidal probability distribution which assigns a uniform weight between the additive and multiplicative transfer model	• 50% weight assigned to the additive model • 50% weight given to the trapezoidal distribution which assigns a uniform weight between the additive and multiplicative transfer model
Radiation-smoking interaction	• $[x + (1 - x)W_S^*]$, where x is always described as a triangular distribution (0, 1, 1.1). Multiplicative model has a heavier weight than the additive model.	• $[x + (1 - x)W_S^*]$, • For exposure to alpha particles, x is described as a triangular distribution (0, 1, 1.1). Multiplicative model has a heavier weight than the additive model. • For exposures to radiation types other than alpha particles, 50% weight is assigned to case where x follows a triangular distribution (0, 1, 1.1), and 50% weight to case where $x \equiv 0$ (additive model).

NIOSH-IREP risk model for lung cancer

The NIOSH-IREP risk model for lung cancer was derived from epidemiologic studies of a cohort of both smokers and non-smokers Japanese A-bomb survivors. The model is described by the following equation (page 25 in Land et al. 2002):

$$ERR = D \times \alpha(s) \times \exp[\gamma \times f(e) + \delta \times g(a)] = D \times ERR/Sv$$

where

- D = radiation dose (Sv) delivered to lung
- α, γ, δ = parameters of the model associated with each modifier
- s = sex modifier
- $f(e)$ = min[max(-15,e-30),0] where e is the age at exposure
- $g(a)$ = min(ln(a/50),0) where a is the attained age

No age effects are included in the NIOSH-IREP risk model for lung cancer (i.e. $\gamma = \delta = 0$). The profile for α was obtained for males and females separately. Thus, in essence, the NIOSH-IREP risk model for lung cancer is a Group 3-type risk model. In particular, α is the *ERR/Sv* for any age at exposure and any attained age, for a population of both smokers and non-smokers.

The NIOSH-IREP risk model is used to estimate risk of lung cancer in the U.S. population by accounting for the differences in the baseline rates of lung cancer in the Japanese and U.S. population. The transfer from the Japanese to the U.S. population can be done using an additive transfer model, a multiplicative transfer model, or a transfer model which predicts a risk that falls between the risk predicted by the additive and multiplicative models. The NIOSH-IREP lung model assumes complete lack of knowledge about the correct model for transfer between populations. That is, the transfer between populations is modeled using a trapezoidal probability distribution that assigns uniform weight between the additive and multiplicative models, and a small probability (4.5%) that the transfer model can be sub-additive or super-multiplicative.

To estimate probability of causation for different smoking categories, including never smokers, the *ERR/Sv* is multiplied by the term: $[x + (1 - x)W_S^*]$, where W_S^* is the smoking adjustment factor[2]. The uncertainty distribution for x allows the *ERR/Sv* for lung cancer to range from that obtained with an additive interaction ($x = 0$) to that obtained with a multiplicative interaction ($x = 1$). The parameter x is described by a triangular distribution with a minimum value of 0, a mode of 1.0 and a maximum value of 1.1. This triangular distribution gives more weight to the multiplicative model, with a probability of about 0.10 for a super-multiplicative interaction ($x > 1$).

[2] There are 3 major choices for smoking history in NIOSH-IREP: (1) never smoker, (2) former smoker, and (3) current smoker. The current smoker category is further subdivided according to the number of cigarettes smoked per day. Values of the smoking adjustment factor W^* are provided for each smoking category, normalized to a population of both smokers and non-smokers. These values are presented under the "Transfer to U.S. population" area of the "View Model Details" help files in NIOSH-IREP, and they can also be found in Land et al. (2003).

NIH-IREP risk model for lung cancer

The risk model for lung cancer adopted by NIH in their version of IREP (NIH-IREP) is based on epidemiologic studies of never-smoker Japanese A-bomb Survivors (Pierce et al. 2003). The model is described by the following equation:

$$ERR = D \times \alpha \times \exp[\beta \times s + \gamma \times f(e) + \delta \times g(a)] = D \times ERR/Sv$$

where

- D = radiation dose (Sv) delivered to lung
- $\alpha, \beta, \gamma, \delta$ = parameters of the model associated with each modifier
- s = sex modifier (s = + 0.5 females and - 0.5 for males)
- $f(e)$ = min[max(-15,e-30),0] where e is the age at exposure
- $g(a)$ = min(ln(a/50),0) where a is the attained age

Parameter α is assumed to be uncorrelated with β, γ, and δ. Parameters β, γ, and δ are correlated. The joint distribution of β, γ, and δ is a multivariate normal with specified means and covariance matrix (this distribution corresponds closely to the profiles for these parameters). This approach is similar to the treatment of cancers in Group 2, except for the sex parameter, β. Parameter α applies to both males and females, and gender differences are introduced by the sex modifier s. Thus, there is a unisex profile for parameter α for lung cancer, not separate ones for each sex. In particular, α is the *ERR/Sv* for age at exposure 30 and attained age 50, and represents never smokers.

$$ERR/Sv(e,a) = ERR/Sv(e=30,a=50) \times F(s,e,a) = \alpha \times F(s,e,a)$$

The modifying factor $F(s,e,a)$ is assumed to be independent of α and is described by a lognormal distribution with a mean and variance of logarithms given by:

$$\text{mean} = \beta \times s + \gamma \times f(e) + \delta \times g(a), \text{ and}$$

$$\text{variance} = \text{var}(\beta) \times s^2 + \text{var}(\gamma) \times f(e)^2 + \text{var}(\delta) \times g(a)^2 + 2 \times \text{cov}(\beta,\gamma) \times s \times f(e) + 2 \times \text{cov}(\beta,\delta) \times s \times g(a) + 2 \times \text{cov}(\gamma,\delta) \times f(e) \times g(a)$$

For a given attained age, the *ERR/Sv* decreases exponentially between ages at exposure of 15 and 30 and is constant outside this interval. Similarly, for a given age at exposure, the *ERR/Sv* decreases linearly with attained age, up to attained age 50, after which it remains constant.

In the NIH-IREP risk model, the transfer between populations is assumed to be more consistent with the additive model, as indicated in the analysis by Pierce et al. (1993). Thus, when the NIH-model is applied to the U.S. population, a 50% weight is given to the additive model and 50% weight is given to the trapezoidal distribution used by the NIOSH-IREP risk model (which assigns a uniform weight between the additive and multiplicative model).

To estimate probability of causation for different smoking categories, the *ERR/Sv* for never smokers

is multiplied by the term: $[x + (1 - x)W_S^*]$, where W_S^* is the smoking adjustment factor[3]. The uncertainty distribution for x allows the *ERR/Sv* for lung cancer to range from that obtained with an additive interaction ($x = 0$) to that obtained with a multiplicative interaction ($x = 1$). For exposure to radiation types other than alpha particles, a 50% probability is given to the purely additive model (where $x = 0$) and 50% to a triangular distribution with a minimum value of 0, a mode of 1.0 and a maximum value of 1.1. This triangular distribution gives more weight to the multiplicative model, with a probability of about 0.10 for a super-multiplicative interaction ($x > 1$). In cases of exposure to alpha particles, the interaction between radiation and smoking is modeled by assigning 100% weight to the triangular distribution described above (minimum = 0, mode =1.0 and maximum = 1.1) which gives more weight to the multiplicative model, as indicated by uranium miners studies.

Lung cancer from exposure to radon

The risk from exposure to radon is derived from studies of uranium miners (RECA 1996). The risk model depends on the level of exposure to radon (expressed in working level months – WLM) rather than on radiation dose to the lung tissues.

$$ERR = \alpha \times WLM^{0.82}$$

where *WLM* is the number of working level months from exposure to radon provided by the user. Parameter α represents the risk from exposure to 1 WLM. Alpha was found to decrease exponentially with the attained age (a) for $45 \le a \le 75$ and with the time since last exposure (TSLE) for 5 years $\le$ TSLE $\le$ 25 years. Outside these limits, α is constant and equal to the α at the respective limit. Parameter α is dependent on smoking history and is defined for two smoking categories (smokers and never smokers). The values for α contain a bias correction for the random errors in doses estimated for the uranium miners.

[3] There are 3 major choices for smoking history in NIH-IREP: (1) never smoker, (2) former smoker, and (3) current smoker. The current smoker category is further subdivided according to the number of cigarettes smoked per day. Values of the smoking adjustment factor W^* are provided for each smoking category, normalized to never smokers. These values are presented under the "Transfer to U.S. population" area of the "View Model Details" help files in NIOSH-IREP, and they can also be found in Land et al. (2003).

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