Defense Threat Reduction Agency
8725 John J. Kingman Road, MS-6201
Fort Belvoir, VA 22060-6201

DTRA-TR-16-054

TECHNICAL REPORT

Approximating the Probability of Mortality Due To Protracted Radiation Exposures

June 2016

HDTRA1-14-D-0003; 0005

Prepared by:

Applied Research Associates, Inc.
801 N. Quincy Street
Suite 700
Arlington, VA 22203

This page intentionally left blank.

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

PLEASE DO NOT RETURN YOUR FORM TO THE ABOVE ADDRESS.

1. REPORT DATE *(DD-MM-YYYY)*	2. REPORT TYPE	3. DATES COVERED *(From - To)*
10-06-2016	Technical Report	October 2005 to January 2009

4. TITLE AND SUBTITLE

Approximating the Probability of Mortality Due To Protracted Radiation Exposures

5a. CONTRACT NUMBER

HDTRA1-14-D-0003; 0005

5b. GRANT NUMBER

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)

McClellan, Gene
Crary, David
Oldson, Darren

5d. PROJECT NUMBER

5e. TASK NUMBER

5f. WORK UNIT NUMBER

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

Applied Research Associates, Inc.
801 N. Quincy Street, Suite 700
Arlington, VA 22203

8. PERFORMING ORGANIZATION REPORT NUMBER

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)

Nuclear Technologies Department, Attn: Dr. Blake
Defense Threat Reduction Agency
8725 John J. Kingman Road, Mail Stop 6201
Fort Belvoir, VA 22060-6201

10. SPONSOR/MONITOR'S ACRONYM(S)

DTRA J9NT

11. SPONSOR/MONITOR'S REPORT NUMBER(S)

DTRA-TR-16-054

12. DISTRIBUTION/AVAILABILITY STATEMENT

DISTRIBUTION A. Approved for public release: distribution is unlimited.

13. SUPPLEMENTARY NOTES

14. ABSTRACT

Probability of fatality plots in HPAC for whole body exposure due to nuclear weapons frequently indicate a median lethal dose (LD50) much higher than the prompt dose value, even for acute exposures. This apparent paradox is discussed in this paper. We conclude that the values generated by HPAC are reasonable in the MARCELL model, which includes the effects of bone marrow cell damage, repair and death. Values of fallout age-at-entry and exposure duration encountered in HPAC calculations typically generate exposures having LD50 ranging from 600 to 700 cGy, instead of the prompt value of 410 cGy. We present approximate methods for estimating the probability of mortality due to radiological environments from nuclear weapon detonations or from a radiological dispersal device.

15. SUBJECT TERMS

RIPD, MarCell, CHRNEM, Protracted Radiation Exposures, Health Effects, Mortality

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			Paul K. Blake, Ph.D.
U	U	U	U	31	19b. TELEPHONE NUMBER *(Include area code)* 703-767-3433

UNIT CONVERSION TABLE

U.S. customary units to and from international units of measurement*

U.S. Customary Units	Multiply by → / ← Divide by†		International Units
Length/Area/Volume			
inch (in)	2.54	$\times 10^{-2}$	meter (m)
foot (ft)	3.048	$\times 10^{-1}$	meter (m)
yard (yd)	9.144	$\times 10^{-1}$	meter (m)
mile (mi, international)	1.609 344	$\times 10^{3}$	meter (m)
mile (nmi, nautical, U.S.)	1.852	$\times 10^{3}$	meter (m)
barn (b)	1	$\times 10^{-28}$	square meter (m^2)
gallon (gal, U.S. liquid)	3.785 412	$\times 10^{-3}$	cubic meter (m^3)
cubic foot (ft^3)	2.831 685	$\times 10^{-2}$	cubic meter (m^3)
Mass/Density			
pound (lb)	4.535 924	$\times 10^{-1}$	kilogram (kg)
unified atomic mass unit (amu)	1.660 539	$\times 10^{-27}$	kilogram (kg)
pound-mass per cubic foot (lb ft^{-3})	1.601 846	$\times 10^{1}$	kilogram per cubic meter (kg m^{-3})
pound-force (lbf avoirdupois)	4.448 222		newton (N)
Energy/Work/Power			
electron volt (eV)	1.602 177	$\times 10^{-19}$	joule (J)
erg	1	$\times 10^{-7}$	joule (J)
kiloton (kt) (TNT equivalent)	4.184	$\times 10^{12}$	joule (J)
British thermal unit (Btu) (thermochemical)	1.054 350	$\times 10^{3}$	joule (J)
foot-pound-force (ft lbf)	1.355 818		joule (J)
calorie (cal) (thermochemical)	4.184		joule (J)
Pressure			
atmosphere (atm)	1.013 250	$\times 10^{5}$	pascal (Pa)
pound force per square inch (psi)	6.984 757	$\times 10^{3}$	pascal (Pa)
Temperature			
degree Fahrenheit (^oF)	$[T(^oF) - 32]/1.8$		degree Celsius (^oC)
degree Fahrenheit (^oF)	$[T(^oF) + 459.67]/1.8$		kelvin (K)
Radiation			
curie (Ci) [activity of radionuclides]	3.7	$\times 10^{10}$	per second (s^{-1}) [becquerel (Bq)]
roentgen (R) [air exposure]	2.579 760	$\times 10^{-4}$	coulomb per kilogram (C kg^{-1})
rad [absorbed dose]	1	$\times 10^{-2}$	joule per kilogram (J kg^{-1}) [gray (Gy)]
rem [equivalent and effective dose]	1	$\times 10^{-2}$	joule per kilogram (J kg^{-1}) [sievert (Sv)]

*Specific details regarding the implementation of SI units may be viewed at http://www.bipm.org/en/si/.

†Multiply the U.S. customary unit by the factor to get the international unit. Divide the international unit by the factor to get the U.S. customary unit.

2015-11-16

Table of Contents

List of Figures

List of Tables

Acknowledgements

This work was originally performed as part of the CBRNE Risk Assessment for Human Response and Health Effects contract, DTRA-01-03-D-0014-0015, under Mr. Eric Nelson at NTMP. That contract included a wide-range of tasks designed to support the Hazard Prediction and Assessment Capability (HPAC) and NBC Casualty and Resource Estimation Support Tool (NBC CREST).

Under the current Nuclear Survivability and Forensics contract, HDTRA1-14-D-0003; 0005, Dr. Paul Blake of DTRA/NTPR has supported the transition of this work into a technical report so that results based on the methodologies described here can be accessed.

Abstract

Probability of fatality plots in HPAC for whole body exposure due to nuclear weapons frequently indicate a median lethal dose (LD_{50}) much higher than the prompt dose value, even for acute exposures. This apparent paradox is discussed in this paper. We conclude that the values generated by HPAC are reasonable in the MARCELL model, which includes the effects of bone marrow cell damage, repair and death. Values of fallout age-at-entry and exposure duration encountered in HPAC calculations typically generate exposures having LD_{50} ranging from 600 to 700 cGy, instead of the prompt value of 410 cGy. We present approximate methods for estimating the probability of mortality due to radiological environments from nuclear weapon detonations or from a radiological dispersal device.

Section 1.

Introduction

In the Hazard Prediction and Assessment Capability (HPAC) software, probability of mortality for whole-body, protracted radiation exposure in the fallout field of a nuclear detonation is based on the Radiation-Induced Performance Decrement (RIPD) software developed by the Defense Nuclear Agency in the 1990s. The purpose of this paper is to better explain the qualitative and quantitative features of mortality due to protracted radiation exposure and to show how characteristic values for median lethal dose from fallout exposure arise in HPAC calculations.

A sometimes puzzling feature of the probability of mortality contours generated by HPAC for nuclear fallout is the occurrence of median lethal dose (LD_{50}) values between 600 and 700 cGy, significantly higher than the prompt dose value of 410 cGy. The puzzle is exacerbated when exposure durations of 24 or 48 hours are referred to as "acute" doses, implying that they ought to have an LD_{50} near that of a prompt dose. Biologically, prompt doses delivered in less than a minute have a significantly different effect than those delivered over periods lasting an hour or longer. The data and discussions below provide illustrations of this difference and some insight into the occurrence of LD_{50} values around 700 cGy.

Because the RIPD code is computationally intensive, it is useful to have an easier, approximate calculation for estimating probability of mortality due to protracted radiation exposure. The following discussion provides an overview of RIPD calculations for selected exposure histories and presents approximations that may be used for a range of situations related to nuclear fallout and other radiological exposures.

Section 2.

The MARCELL Model for Radiation-Induced Mortality

The probability of mortality in RIPD is calculated with the marrow cell (MARCELL) model (Jones *et al.*, 1993b) developed at Oak Ridge National Laboratory (ORNL) for the Defense Nuclear Agency. The model estimates mortality due to the hematopoietic syndrome of acute radiation sickness. In the MARCELL model, radiation exposure dynamically depletes the bone marrow cell population, the underpinning of the immune system. For any protracted exposure, an equivalent prompt dose (EPD) is estimated by finding the prompt dose that produces the same marrow cell population nadir (maximum cytopenia) as the protracted dose in question. This EPD is used in conjunction with a lognormal, dose-response curve for prompt doses to determine the probability of mortality for the protracted exposure.

MARCELL is a physiologically based, cell-kinetics model of the response of bone marrow tissue to radiation exposure (see the Appendix of Jones *et al.*, 1994a for a description of the model and equations). The model tracks the marrow cell population in three compartments: normal, injured, and killed cells. The compartment populations are described by a set of differential (rate) equations emulating normal cell turnover, radiation damage, repair of damage, cell killing, and tissue recovery. At zero dose rate, the set of equations has an equilibrium solution corresponding to a healthy individual. The equations contain the radiation dose rate as a driving term that creates cell damage, thereby depleting the marrow cell population. The cell population recovers either after the exposure ends or when the dose rate drops below a level where cell proliferation can compensate for the continuing cell depletion rate. Depletion resumes if the dose rate is increased again. This modeling approach accommodates temporally varying dose rates including combinations of prompt and fallout radiation exposures. The RIPD software numerically solves the MARCELL model equations for an exposure history defined by the user.

The development, testing, and application of the MARCELL model is extensively documented in a series of journal publications sponsored by the Defense Nuclear Agency (Jones *et al.* and Morris *et al.*, 1991-1997). In these references, the MARCELL model is applied to both data from animal experiments and data from relevant human sources. Animal experiments on a wide variety of species validate the way in which MARCELL models the effects of dose protraction on mortality. Parameters built into RIPD are the best available set for estimating human mortality (see Table 4 of Jones *et al.*, 1994a). The RIPD implementation of the MARCELL model has been verified against results published by the ORNL group (Morris *et al.*, 1994) and those calculated with an ORNL software version of the model named MarCel21.

Section 3.

Prompt Dose Mortality

The probability of mortality calculation in RIPD and RIPDLIPI (Radiation-Induced Performance Decrement Lethality Injury Probability Interpolation) is referenced to a lognormal dose-response curve for prompt doses (Anno *et al.*, 2003) having a median lethal dose (LD_{50}) of 410 cGy free-in-air (FIA) tissue kerma and a (probit) slope of 7.1 (using base-10 logarithms). Figure 1 compares this prompt dose-response curve with that used in the Combined Human Response Nuclear Effects Model (CHRNEM), which is based on the same LD_{50} value but a shallower slope of 4.4 (Levin and Fulton, 1992).

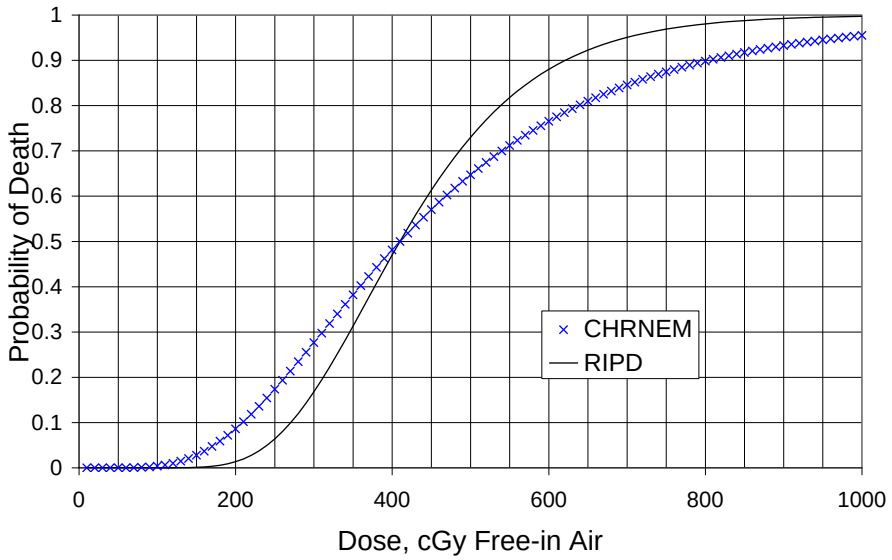

Figure 1. The prompt dose mortality curve used in RIPD has a steeper slope than that of the older CHRNEM software.

Section 4.

Exposure at Constant Dose Rate

One way to characterize a protracted exposure to ionizing radiation is by specifying two parameters: a (constant) dose rate and an exposure duration. Given values for these parameters, one can calculate integrated dose and estimate biological effect. In this section we describe how the MARCELL model relates integrated dose to biological effect, for the case of constant dose rate exposures.

For an individual in a constant dose rate field of moderate duration, the marrow cell population calculated from the MARCELL model falls monotonically, approaching a limiting population level that depends on the magnitude of the dose rate. At the end of the radiation exposure, the cell population begins a recovery to its normal equilibrium value. The resulting cell population nadir occurs at the end of the exposure when radiation damage ceases and repair processes continue. For a fixed total dose, increasing the exposure duration lowers the dose rate during exposure, resulting in less depletion of the cell population at the nadir. Therefore, the median lethal dose (LD_{50}) is larger for longer exposures. Figure 2 plots data from RIPD v2.0 showing the dependence of LD_{50} on the duration of exposure for exposures at constant dose rate. In the limit of zero duration of exposure, the LD_{50} equals 410 cGy, the value for a prompt exposure.

Note that in Figure 2 there is a different "constant" dose rate for each duration of exposure. The plot is a contour curve in the sense of biological effect: each point on the curve represents an exposure (a dose rate, exposure duration pair) that results in a 50% probability of mortality. Note that, by fixing biological effect, dose rate is a function of exposure duration, so only one parameter, exposure duration, is needed to specify the exposure scenario.

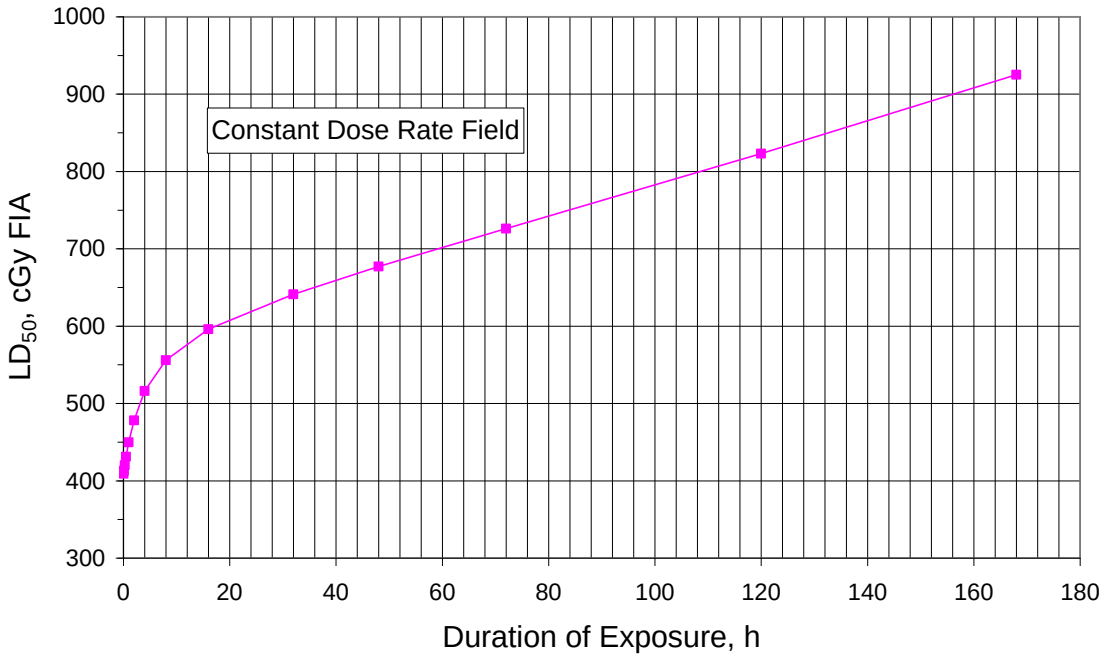

Figure 2. Because of biological repair and recovery processes, the median lethal dose for exposures at a constant dose rate increases with increasing duration of exposure.

There are characteristic time constants associated with the physiological processes modeled in MARCELL. These characteristic times are associated with cell proliferation and the repair of radiation damage. Together they determine the shape of the curve in Figure 2. To display the effects of these characteristics times more clearly, Figure 3 shows the data of Figure 2 plotted on a logarithmic time scale. Below about 10 minutes, the dependence on exposure duration is weak; the LD_{50} differs little from that for a prompt exposure. For exposures longer than half an hour, the LD_{50} begins to rise significantly. For exposure durations between about one hour and 30 hours, the curve in Figure 3 is nearly straight, indicating an approximate power law dependence of LD_{50} on duration of exposure. For exposure durations longer than about 50 hours (or 2 days), the curve turns up more steeply.

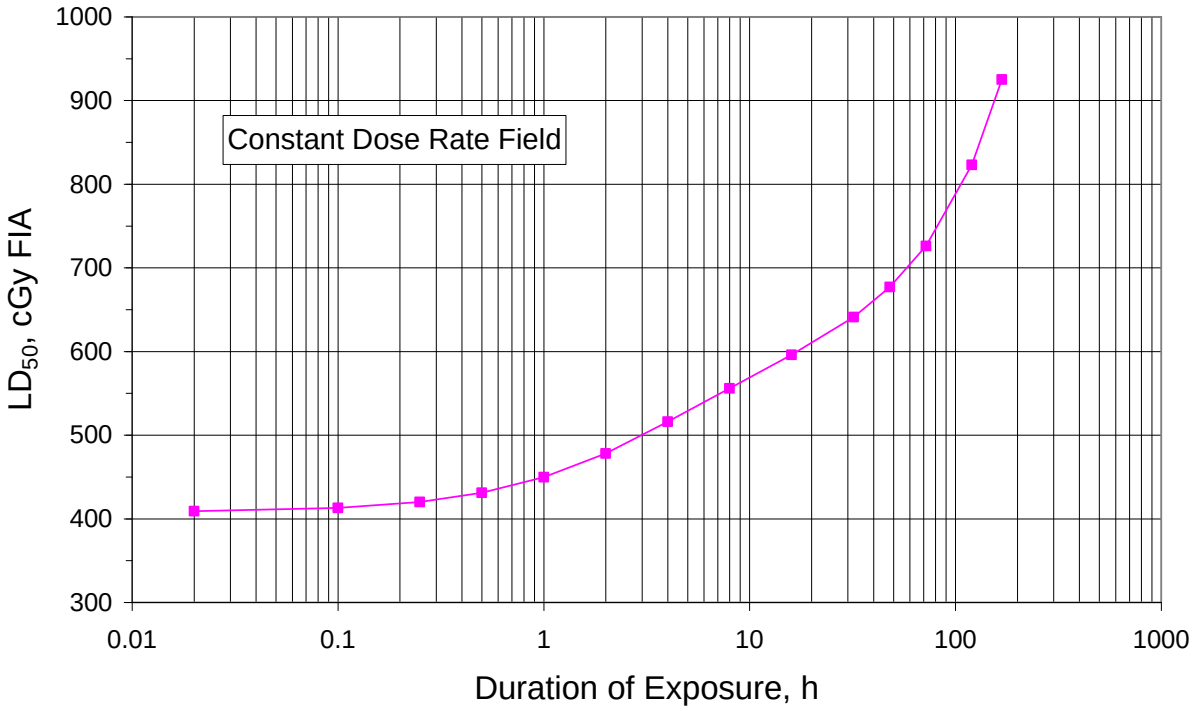

Figure 3. Data from Figure 2 plotted with a logarithmic time scale.

Figure 4 plots similar LD_{50} data calculated with RIPD v2.0 showing the LD_{50} for constant dose rate exposures as a function of dose rate rather than exposure duration. The straight solid line representing power law behavior is a regression fit to the five data points between dose rates of 20 and 300 cGy/h. In log-log space (base 10), the slope and intercept of the straight line are 2.9634 and -0.11918, respectively.

Figure 4 illustrates a principle having wide application in the theory and analysis of human response data, namely, that power law models are useful but extreme caution is required regarding extrapolation. It is common to use a power law model to analyze and describe experimental toxicity data when a suitable physiologically based model of response (either human or animal) is not available. Because experiments are costly, there is rarely enough data to map out the full range of response in terms of exposure rate or duration. Frequently, the data in the range of experimentation can be described quite well by a power law relationship between response and exposure rate or duration. Such is the case for the toxic load model of the effects of chemical exposure. Modelers must be keep in mind that these power law relationships can rarely be extrapolated with confidence to either long or short duration exposures (low or high dose rates). Figure 4 illustrates this point for a physiologically based model of the response to ionizing radiation exposure. The power law works quite well in a limited range of dose rates but fails to extrapolate correctly to either low or high dose rates.

The single data point labeled “Intermittent Exposure” in Figure 4 is the RIPD calculation of the LD_{50} for four equal doses of 1 h duration starting at 0, 5, 10, and 15 h, respectively, for a total exposure period 16 h. The LD_{50} for this fractionated exposure is 559 cGy or an average dose rate of 35 cGy/h during the 16 h period. The solid line in Figure 4 shows that the LD_{50} for a constant dose rate of 35 cGy/h is just over 600 cGy. From Figure 3, the LD_{50} for a single one hour

exposure is about 450 cGy. Therefore, the effect of fractionation on the LD_{50} is intermediate between that of a one hour exposure (corresponding to the length of individual fractions) and a sixteen hour exposure (corresponding to the total exposure period). The effect is closer to that of a sixteen hour exposure. The total time during which the exposure is non-zero for the fractionated exposure is four hours. From Figure 3, the LD_{50} for a four-hour exposure is about 520 cGy, so the effect of the fractionated exposure is midway between that for the four-hour and the 16-hour exposure periods. That is, the effect is midway between that of an exposure with the duration of the fractionated exposure's non-zero exposure rate and that of an exposure with duration equal to the fractionated exposure's total duration.

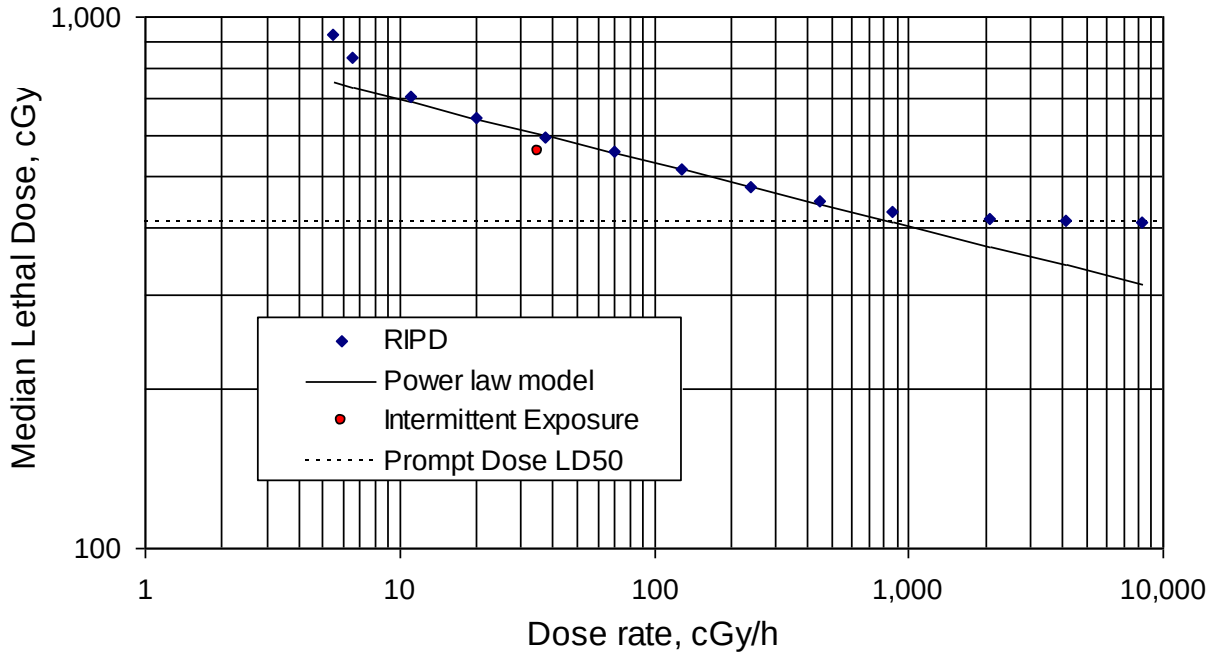

Figure 4. Median lethal dose for exposures at constant dose rate plotted as a function of dose rate (exposure duration is determined by dose rate, because biological effect is fixed).

Figure 5 examines the behavior of the MARCELL model from the viewpoint of a fixed total dose of 410 cGy delivered at different fixed dose rates (*i.e.*, different exposure durations). Figure 5 shows that the equivalent prompt dose for mortality approaches 410 cGy at high dose rates as it must to be biologically reasonable. At low dose rates, the EPD decreases rapidly, consistent with the fact that dose rates approaching natural background will have negligible effect on mortality. In the intermediate range between 10 and 1000 cGy/h, the dependence of EPD on dose rate is well described by a power law. The regression fit to the six points between 10 and 500 cGy/h results in an intercept and slope of 2.3494 and 0.085302, respectively, in log-log space (base 10). The single point labeled "Intermittent Exposure" is for the same fractionated schedule as that in Figure 4 with a dose rate of 102.5 cGy/h during each pulse and an average of 25.63 cGy/h for the 16 hour period of exposure.

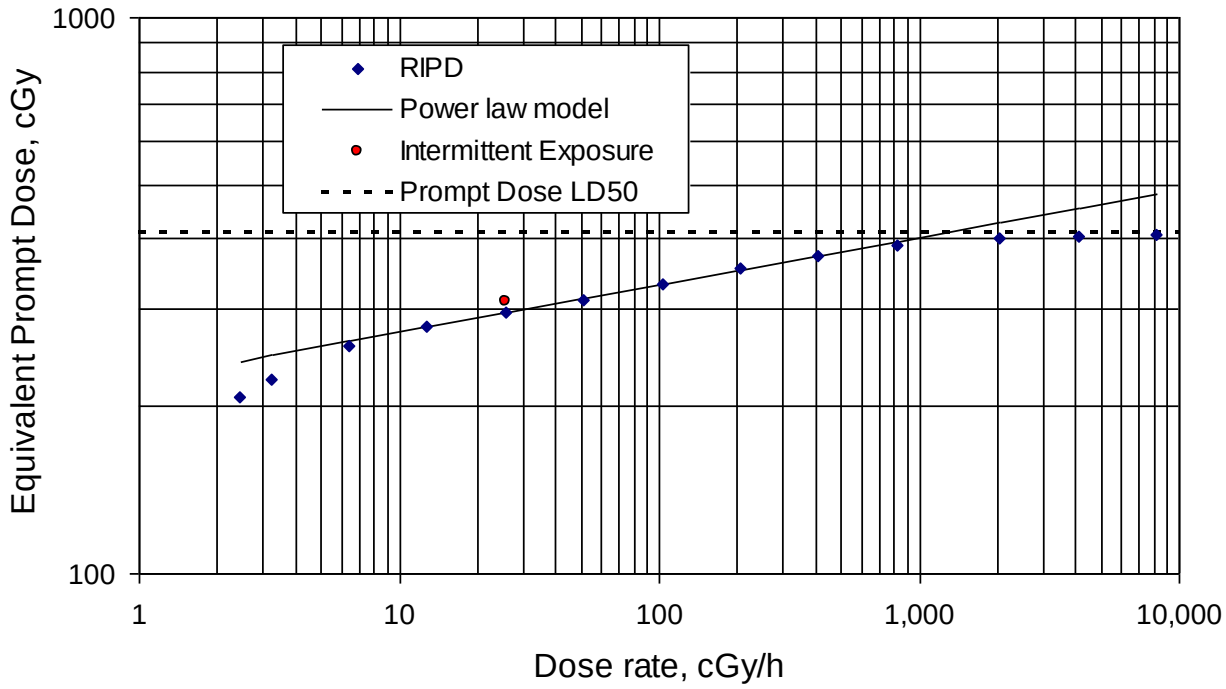

Figure 5. The EPD for a 410 cGy dose delivered at constant dose rate also has a power behavior over a limited range of dose rates.

Section 5.

Dose-Response Approximations for Exposure at Constant Dose Rate

This section presents two methods for approximating the lethal effects of exposure at constant dose rate described in the previous section. It is useful to have information on the shape of the dose response curve as well as the median lethal dose. In this regard, Figure 6 plots as a function of duration of exposure the 10% and 90% lethal doses (LD_{10} and LD_{90}, respectively) for exposures at constant dose rate as well as the LD_{50} described previously.

Table 1 lists the data plotted in Figure 6 as well as the ratio of LD_{90} to LD_{10}, which is determined by the slope of the dose response curve. The relatively small variation of this ratio over the more than three orders of magnitude change in duration of exposure indicates that variation of the slope might be neglected in constructing a simple model of the effect of dose protraction on the dose-response curve for mortality. The ratio changes by only 13% while the LD_{50} increases by 65% over this range.

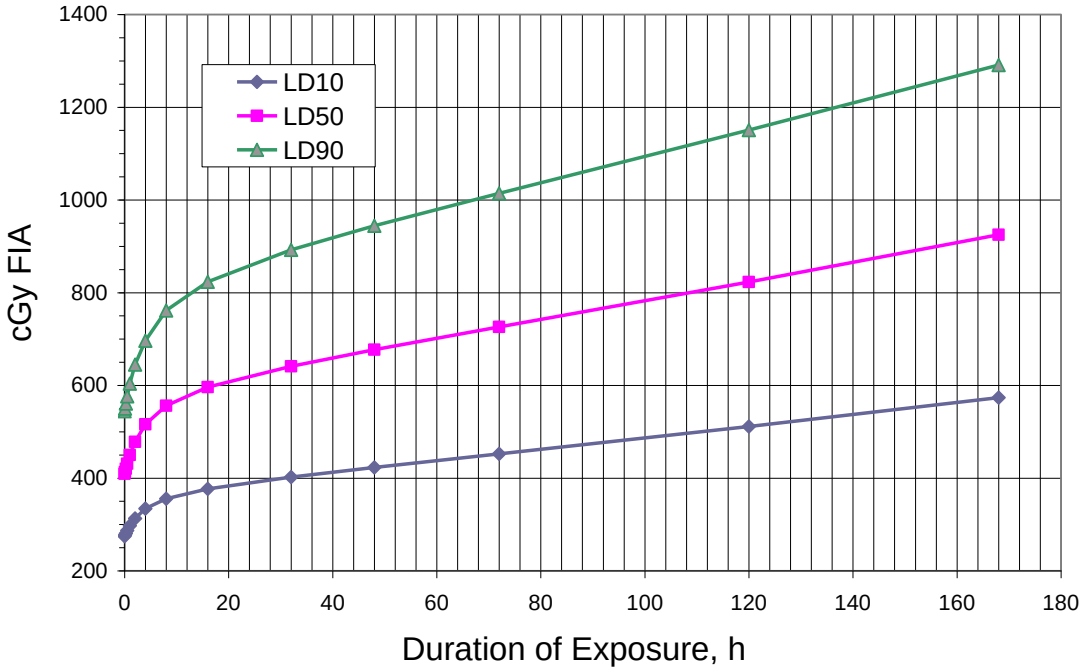

Figure 6. Response parameters for constant dose rate exposure.

Table 1. Data from Figure 6 showing the relatively small variation in the slope of the dose response curve as measured by the ratio of the LD_{90} to the LD_{10}.

Exposure Duration	LD_{10}	LD_{50}	LD_{90}	LD_{90}/LD_{10}
h	*cGy*	*cGy*	*cGy*	
0.02	274.7	409.5	544.3	1.98
0.1	276.9	413.4	550.1	1.99
0.25	280.9	420.4	560.6	2.00
0.5	286.9	431.2	576.6	2.01
1	297.3	449.7	603.9	2.03
2	312.8	477.6	644.9	2.06
4	334.2	516.0	696.2	2.08
8	355.5	555.7	761.4	2.14
16	376.9	595.8	823.2	2.18
32	402.1	641.5	892.3	2.22
48	422.7	677.0	944.1	2.23
72	452.0	726.0	1014.2	2.24
120	511.1	823.3	1150.5	2.25
168	573.4	924.7	1291.1	2.25

Radiological weapons ("dirty bombs") will in most cases disperse radionuclides whose half-life is long enough that the dose rate in contaminated areas will be essentially constant over most emergency response scenarios. Therefore the data presented here is directly applicable to resulting whole-body gamma ray exposures. For a quick estimate of the median lethal dose (or the LD_{90} or LD_{10}) for a given scenario, one may simply use Table 1 as a look-up table, interpolating relative to exposure duration if desired. Alternatively, the data in Table 1 may be fitted with an analytic curve to provide an estimate at any exposure duration within the given range of durations. We tested several alternatives and found that a sixth-order polynomial relating $y = \log_{10}$(*dose* in cGy) to $x = \log_{10}$(*time* in hours) gave a good compromise between simplicity and accuracy. Therefore, we have the expression

$$y = \sum_{n=0}^{6} c_n x^n \,. \tag{1}$$

The coefficients c_n for the three sets of data shown in Figure 6 are listed in Table 2. The smooth curves in Figure 6 are generated by the Microsoft Excel plotting utility. Curves generated by Equation 1 with the coefficients from Table 2 are plotted for LD_{50}, LD_{10}, and LD_{90} in Figure 7, Figure 8, and Figure 9, respectively. Equation 1 may be used between 0.1 and 168 h.

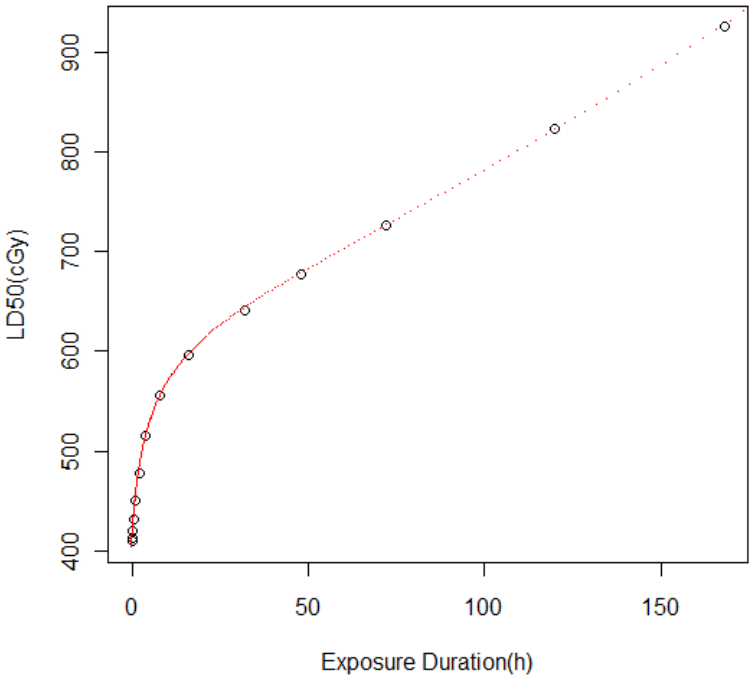

Figure 7. Curve shows sixth-order polynomial fit to the LD_{50} data (circles) from Table 1. Coefficients of the polynomial are listed in Table 2.

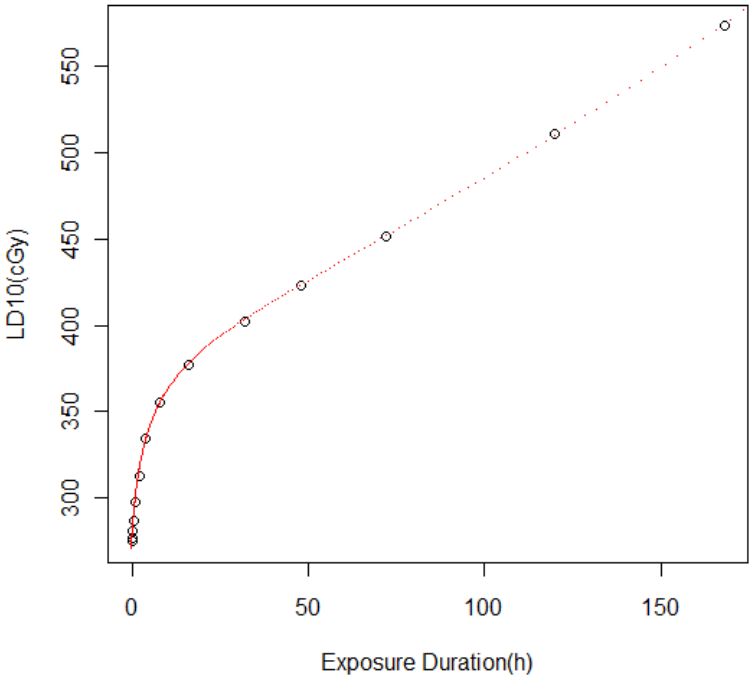

Figure 8. Curve shows sixth-order polynomial fit to the LD_{10} data (circles) from Table 1. Coefficients of the polynomial are listed in Table 2.

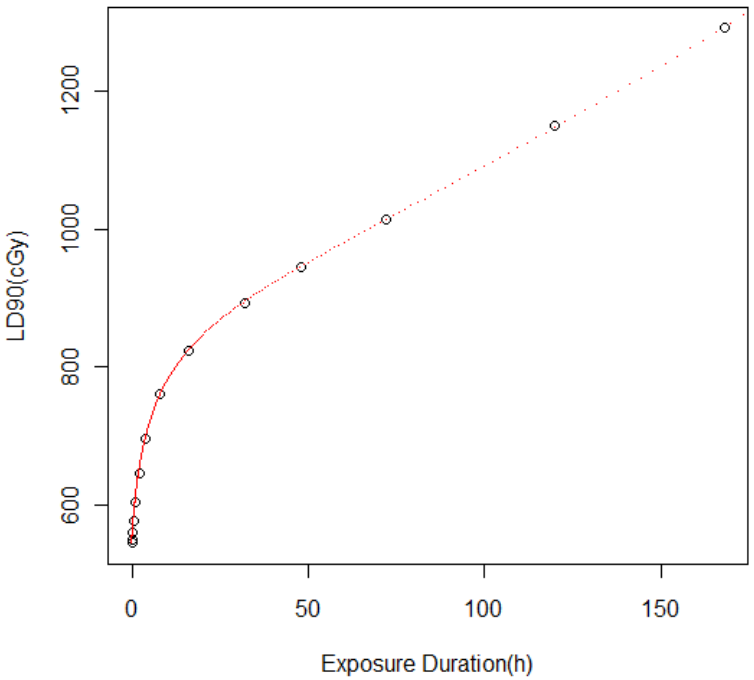

Figure 9. Curve shows sixth-order polynomial fit to the LD_{90} data (circles) from Table 1. Coefficients of the polynomial are listed in Table 2.

Table 2. Coefficients for sixth-order polynomial fits to the data in Table 1. Independent variable is $\log_{10}$(*time* in hours). Polynomial gives $\log_{10}$(*dose* in cGy).

Coefficient:	c_0	c_1	c_2	c_3	c_4	c_5	c_6
LD_{90}	2.781	0.08238	0.04383	-0.008851	-0.01075	0.001748	0.001609
LD_{50}	2.654	0.07762	0.03944	-0.01164	-0.009152	0.002541	0.001309
LD_{10}	2.474	0.06620	0.03308	-0.01106	-0.007633	0.002600	0.001161

The LD_{50} data in Table 1 has also been fit as a function of dose rate (Oxford, 2016), using the following formula:

$$LD_{50}(DR) = \frac{LD_{50}(0.02)}{c_0 \cdot c_1^{DR} \cdot DR^{c_2} + c_3}. \qquad (2)$$

Where $LD_{50}(DR)$ is the value of the LD_{50} (in Gy) at dose rate DR (in Gy/h). The coefficients for this fit are given in Table 3, below.

This functional form was derived using open-source curve-fitting software which is available at the website https://github.com/zunzun/pyeq2. This software fits a large number of linear and non-linear functions to a given data set, using a genetic algorithm to find initial values for the fits. It then ranks the fits, using a measure such as Akaike Information Criteria (AIC), and reports these results back to the user. The form of Equation 2 was selected from the top three

ranked functions returned by the software, and was the only function which gave reasonable values when extrapolating outside the dose rate range of the fitted data (Oxford, 2016).

Table 3: Coefficients for fits of function given in Equation 2 to LD_{50} data given in Table 1. Independent variable is dose rate in Gy/h. Equation 2 gives the value of LD_{50} in Gy.

Coefficient:	c_0	c_1	c_2	c_3
LD_{50}	-0.2351	0.8946	-0.2876	-0.9947

LD_{50} as a function of dose rate from the data in Table 1 and from the fit given by Equation 2 is shown in Figure 10.

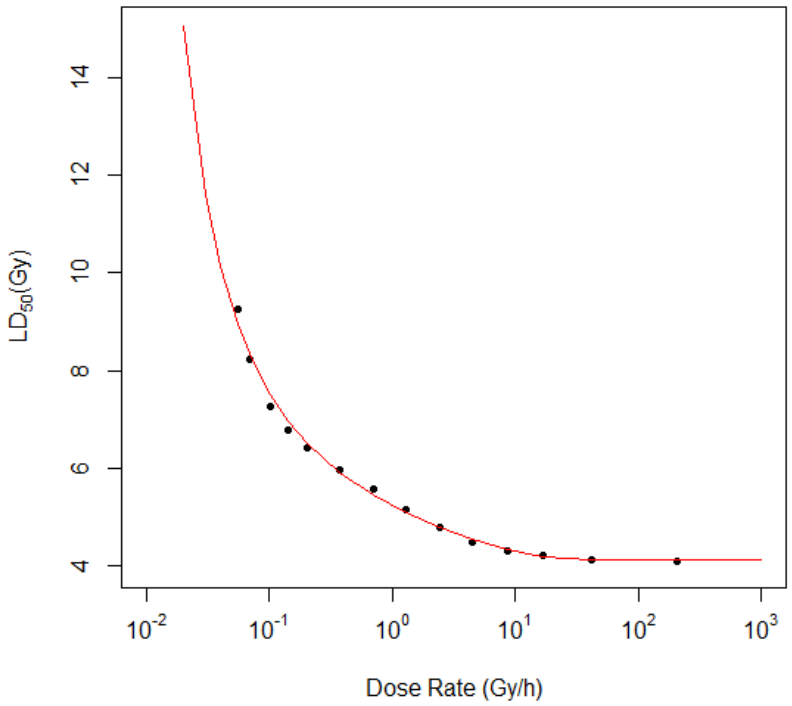

Figure 10: Curve shows Equation 2 fitted to the LD_{50} values (circles) from Table 1. Coefficients for Equation 2 are given in Table 3.

Section 6.

Exposure in a Nuclear Fallout Field

At minimum, one extra parameter is needed to characterize exposure to a nuclear fallout field. For a constant dose rate exposure, the two independent variables describing the exposure are the dose rate and the exposure duration. For a fallout exposure, the corresponding variables are the dose rate at entry into the fallout field and the exposure duration; however, an additional independent variable is needed to specify the rate of decrease of the dose rate, that is, the dose rate decay. Because the decay of the various radionuclides begins at the time of nuclear detonation and because short-lived radionuclides become negligible quickly, the rate of decrease of the dose rate during the exposure period depends strongly on the age of the fallout at the time of entry. The average radionuclide decay rate is much faster soon after the detonation than it is later. Therefore, the age of the fallout at the time of entry is needed as a third independent variable to fully specify the dose rate history for a fallout exposure.

Over the range of time-after-detonation and exposure durations considered in this paper, the decay of radionuclides gives a dose rate history that is well approximated by a power law as a function of time. Commonly, the dose rate decay has been approximated by the function $t^{-1.2}$. More recently, (as in HPAC v4.x), the curve is being approximated with $t^{-1.3}$. RIPDLIPI in HPAC 4.x is based on RIPD calculations with $t^{-1.3}$. The calculations illustrated in this paper are based on Version 2.1 of RIPD, which used $t^{-1.2}$. The features of human response to fallout field exposure described in this paper are not significantly affected by this small difference in exponent.

Operationally, two factors will determine the fallout age-at-entry. The first, obviously, is the time of personnel movement into the fallout field either from a remote location or from a fallout shelter. This movement may occur soon after fallout deposition or many months later. The second factor is the atmospheric transit time from the point of detonation to the location of interest. The transit time consists of the time needed for the radionuclides to rise with the nuclear fireball, drift downwind, and fall to the ground at the location of exposure. This atmospheric transit time places a lower limit on the fallout age at the beginning of exposure. It depends on both meteorology and weapon yield. For kiloton yields it may be as small as a few minutes but for megaton yields several hours or longer depending on range downwind. Because of spatial dispersion of the nuclear cloud during atmospheric transit, the deposition of fallout is not instantaneous but rather occurs over a period of time during which the dose rate rises to a maximum at a given location. For present purposes, this deposition time is assumed to be short compared to the duration of exposure and is neglected.

Because three independent variables are needed to specify the dose rate dependence during exposure to a fallout field rather than the two needed for exposure at constant dose rate, it is convenient for comparisons to fix one of the three variables for the fallout exposure. Figure 11 shows such a comparison fixing the age-at-entry equal to one hour for the fallout exposure. Figure 11 plots RIPD (MARCELL) predictions for LD_{50} as a function of duration of exposure (given age-at-entry equal to one hour, the remaining exposure parameter, dose rate at entry, is determined by duration of exposure). There is little difference between the fallout and constant

dose rate curves for duration of exposure less than 100 hours. For longer durations, the constant duration exposure is less damaging (LD_{50} is higher) because it does not have the high dose rate peak at early times characteristic of fallout exposure. (Similar to Figure 2, Figure 11 is a contour curve in the sense of biological effect: each point on the curve represents an exposure (i.e., specified values of fallout age at entry, exposure duration, and dose rate at entry) that results in a 50% probability of mortality. By fixing biological effect and fallout age at entry, dose rate at entry is a function of exposure duration, so only one parameter, exposure duration, is needed to specify the exposure scenario).

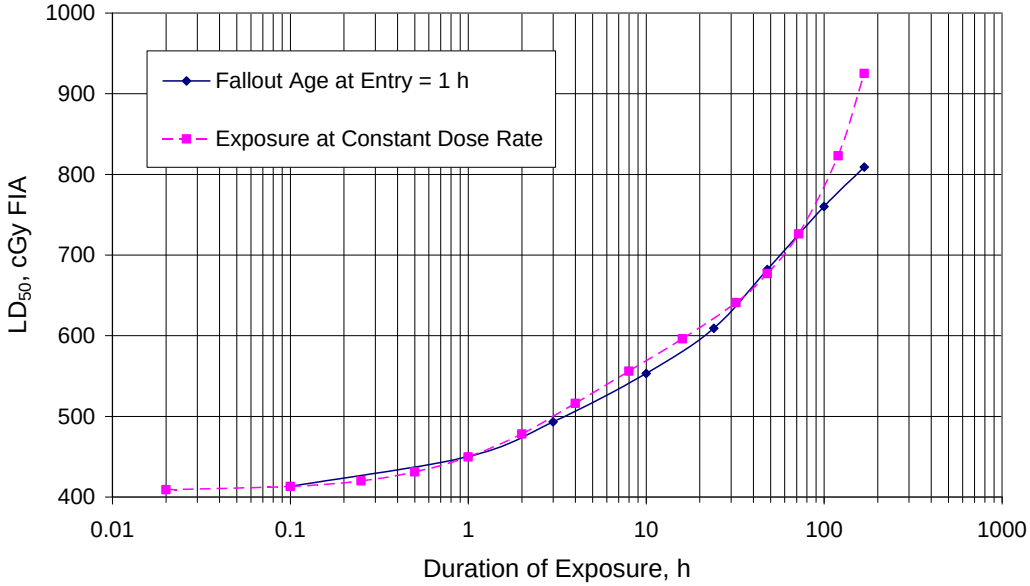

Figure 11. With fallout age fixed at 1 h, the median lethal dose as a function of exposure duration is similar to that for a constant dose rate exposure when the exposure duration is less than four days.

Figure 12 shows the LD_{50} as a function of fallout age-at-entry for exposure duration fixed at 48 hours. As the age-at-entry increases, the dose rate during the exposure becomes flatter, and will eventually approach a constant during the fixed 48 hour period. Figure 2 shows that the LD_{50} for a constant dose rate exposure is about 680 cGy so, as expected, the curve in Figure 12 levels out at about that value for increasing fallout age-at-entry. As age-at-entry decreases, the dose rate becomes quite peaked at the beginning of exposure, leading to an LD_{50} tending toward the prompt dose value as shown in Figure 12.

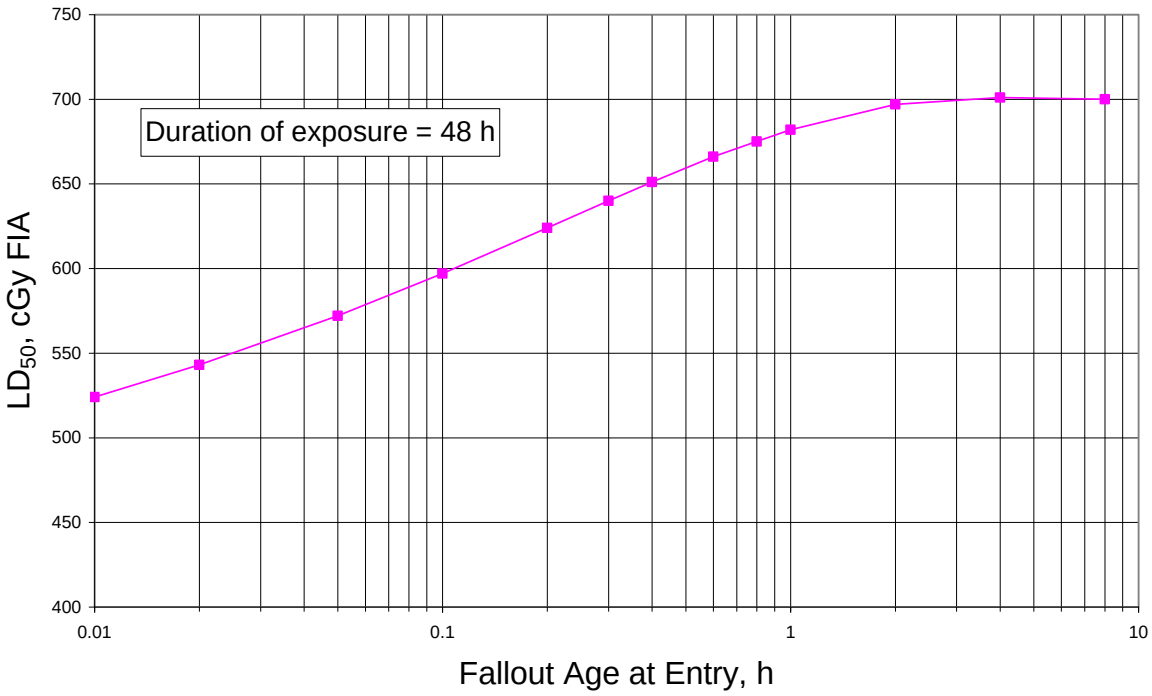

Figure 12. The median lethal dose for a 48 h exposure in a fallout field depends on the age of the fallout at the time of entry into the fallout field.

Section 7.

Dose-Response Approximation for Exposure in a Fallout Field

To provide a first approximation for the probability of mortality due to a single exposure period in the fallout field of a nuclear detonation, we can estimate the LD_{50} and probit slope for the dose response curve for such an exposure using information presented in the two previous sections. Our method consists of using the approximation for exposure at constant dose rate and making a correction for the effect of the decay of the dose rate over time. Although this correction actually varies somewhat with age of fallout, we use a single correction independent of age that gives a good approximation for ages of operational interest for those in the open downwind of a surface burst in the aftermath of a surface or near surface detonation.

Because of cloud stabilization time and deposition time of the fallout, most fallout exposures at ground level (away from the base surge) will begin at fallout ages of at least 10 to 20 minutes. In that case, Figure 12 shows that the LD_{50} will be closer to that of a constant dose rate exposure than a prompt dose. As an approximation to the LD_{50}, we can use a weighted average of the prompt dose value and the constant dose rate value for the exposure duration of interest. Focusing on the data for a 48-h duration of exposure and a fallout age at entry of 0.3 h (*i.e.*, 18 min.), we find that weighting the constant dose rate value six times heavier than the prompt dose value provides a good estimate. The formula for the weighted LD_{50} as a function of duration of exposure is then:

$$weightedLD_{50}(duration) = \frac{LD_{50}prompt + 6 \times LD_{50}const(duration)}{7}. \qquad (3)$$

This "rule of thumb" formula yields a value of 639 cGy for the weighted LD_{50} in good agreement with RIPD calculations as illustrated in Figure 13.

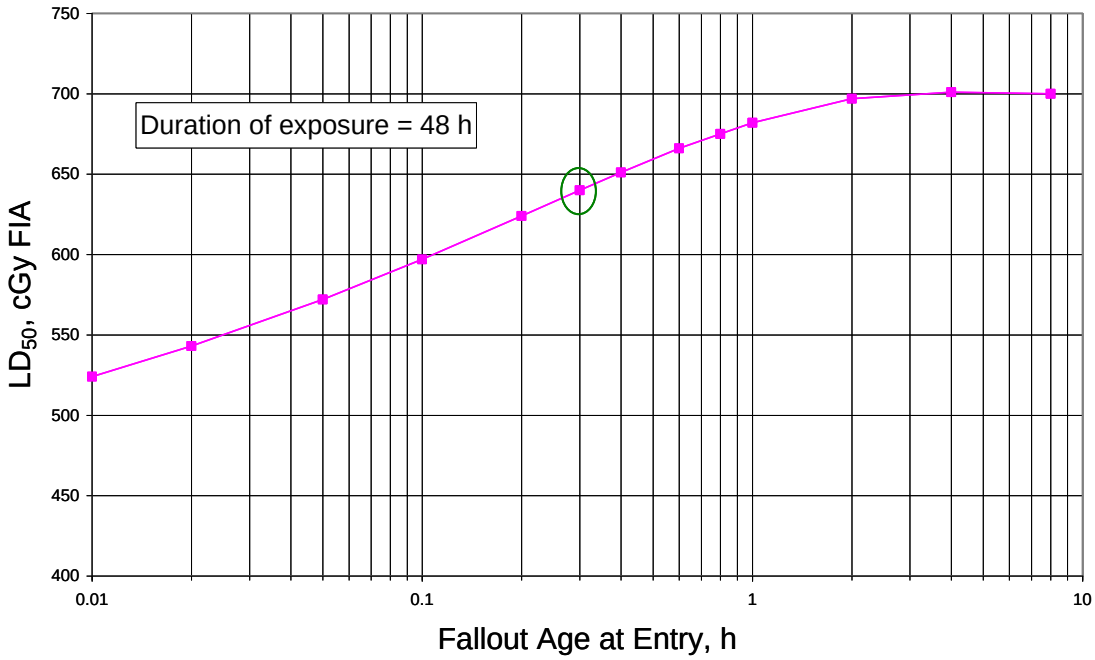

Figure 13. The rule of thumb gives an LD_{50} of about 640 cGy (circled point) for a 48 h exposure, in quite good agreement with the fallout calculation from RIPD for a fallout age at entry of 0.3 h.

To illustrate the effect of neglecting fallout age in the rule of thumb, Table 4 compares weighted values of LD_{50} given by the rule of thumb with RIPD calculations for 0.2 h and 1.0 h ages at entry. The comparison shows that the rule of thumb values reflect the RIPD values quite closely for fallout age at entry of 10 to 20 minutes. The rule of thumb underestimates the LD_{50} somewhat for age at entry of 1 h. This underestimate of the LD_{50} provides a defense-conservative estimate of casualties.

Table 4. Rule of thumb provides results intermediate to fallout ages of 0.2 and 1.0 h.

	LD50 (Free-In-Air, whole-body gamma)			
Exposure Duration h	*Steady Exposure cGy*	*Fallout Age 0.2 h (RIPD) cGy*	*Rule of Thumb cGy*	*Fallout Age 1.0 h (RIPD) cGy*
2	478	468	468	476
4	516	491	501	506
24	618	578	588	609
48	677	624	639	683

In summary, an approximate method for estimating the probability of mortality due to fallout exposure in the aftermath of a nuclear detonation accounting for dose protraction is the following:

1. Choose a fallout exposure duration appropriate to the desired scenario, but not exceeding 4 days nor less than 0.1 h.
2. Determine the LD_{50} for an exposure of that duration at constant dose rate (use Table 1 or Equation 1).
3. For early fallout exposure (*i.e.*, beginning 0.2 to 1 h after detonation), use Equation 3 to estimate the LD_{50}.
4. For later exposures (starting several hours after detonation), use the constant dose rate value from Step 2 for the LD_{50}.
5. Use the same value of the probit slope as for prompt doses (see Section 3).

For radiological scenarios, an approximate method is the following:

1. Choose an exposure duration appropriate to the desired scenario, but in the range for 0.1 h to 168 h.
2. Determine the LD_{50} for an exposure of that duration at constant dose rate (use Table 1 or Equation 1).
3. Use the same value of the probit slope as for prompt doses (see Section 3).

Section 8.

Conclusion

Aside from continuing improvement of documentation of models in HPAC, a primary motivation for this paper is to better understand why the Probability of Fatality plots in HPAC for exposure to nuclear weapon fallout so frequently indicate median lethal doses in the vicinity of 700 cGy rather than the prompt dose value of 410 cGy as illustrated in Figure 14. This result has seemed counter-intuitive to some. However, the analysis presented in this paper shows that such values are quite reasonable in the context of the MARCELL model. It seems that typical values of fallout age-at-entry and exposure duration encountered in HPAC calculations tend to generate protracted exposures having LD_{50} ranging from 600 to 700 cGy.

As a byproduct of this analysis, we present approximate methods for estimating the probability of mortality due to radiological environments from nuclear weapon detonations or from a radiological dispersal device.

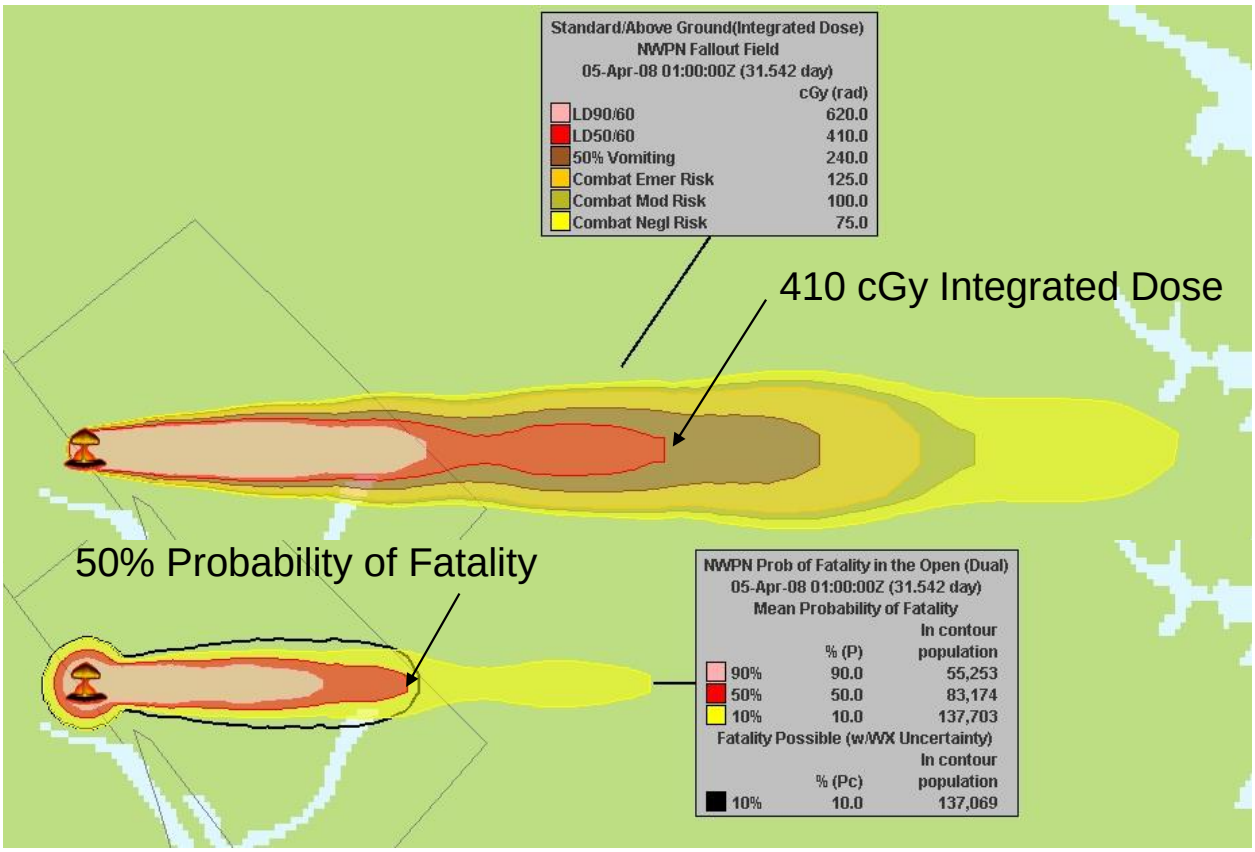

Figure 14. HPAC comparison of 48-hour integrated dose from fallout and the resulting probability of fatality shows the reduction in mortality caused by dose protraction for a 10 KT ground burst. (Note that the probability of fatality includes prompt weapon effects while the integrated dose does not.)

Section 9.

References

Anno, G. H., G. E. McClellan, M. A. Dore, and S. J. Baum, "Biological Effects of Protracted Exposure to Ionizing Radiation: Review, Analysis, and Model Development," DNA-TR-90-157, Defense Nuclear Agency, Alexandria, Virginia 22310-3398, November 1991.

Anno, G. H., G. E. McClellan, and M. A. Dore, "Protracted Radiation-Induced Performance Decrement, Volume 1 – Model Development," DNA-TR-95-117-V1, Defense Nuclear Agency, Alexandria, Virginia 22310-3398, May 1996.

Anno, G. H. et al., "Dose Response Relationships for Acute Ionizing-Radiation Lethality," *Health Phys.* 84, 5:565, 2003.

Jones, T.D., M.D. Morris, and R.W. Young, "A Mathematical Model for Radiation-Induced Myelopoiesis," *Radiation Research* 128: 258-266, 1991.

Jones, T.D., M.D. and Morris, and R.W. Young, "Mathematical Models of Marrow Cell Kinetics: Differential Effects of Protracted irradiations on Stromal and Stem Cells in Mice," *Int. I Radiat. Oncol. Biol. Phvs.* 26: 817-830, 1993a.

Jones, T.D., M.D. Morris, and R.W. Young, Kehlet, R.A., "A cell-kinetics model for radiation induced myelopoiesis," *Experimental Hematology* 21: 816-822, 1993b.

Jones, T. D., M. D. Morris, and R. W. Young, "Dose-Rate RBE Factors for Photons: Hematopoietic Syndrome in Humans vs. Stromal Cell Cytopenia," *Health Phys.* 67:495-508; 1994a.

Jones, T.D., M.D. Morris, and R.W. Young, "Do stem or stromal cells control hematopoietic recovery after irradiation?," *Experimental Hematology* 22: 3-4, 1994b.

Jones, T.D., Morris, M.D., R.W. Young, and R.A. Kehlet, "Neutron RBEs for Cytopenia and Repopulation of Stroma and Hematopoietic Stem Cells: Mathematical Models of Marrow Cell Kinetics," *Health Physics* 72(4): 530-543, 1997.

Levin, S.G., and J. W. Fulton *Consolidated Human Response Nuclear Effects Model (CHRNEM),* Technical Report, DNA-TR-93-45, Defense Nuclear Agency, Alexandria, VA, September 1992

Matheson, L. N., M. A. Dore, G. H. Anno, and G. E. McClellan, "User's Manual: Radiation-Induced Performance Decrement (RIPD) – Version 2.0," DNA-TR-95-91, Defense Nuclear Agency, Alexandria, Virginia 22310-3398, 1996.

Matheson, L. N. and G. E. McClellan, " HPAC Effects Module RIPDLIPI: Radiation-Induced Performance Decrement (RIPD) Lethality Injury Probability Interpolation," HPAC V&V Report, Defense Threat Reduction Agency, Alexandria, Virginia 22310-3398, June 30, 2004.

Morris, M.D., T.D. Jones, and R.W. Young, "Estimation of Coefficients in a Model of Radiation Induced Myelopoiesis from Mortality Data for Mice following X Ray Exposure," *Radiation Research* 128: 267-275, 1991.

Morris, M.D., T.D. Jones, and R.W. Young, “Bone marrow equivalent prompt dose from two common fallout scenarios,” *Health Physics* 67: 183-186, 1994.

Oxford, S. *Private communication*, Institute for Defense Analyses, 2016.