Let $\overline{Z}$ be the mean specific energy due to single hits only, for the fractional number of relevant cells hit. Since 2 is an average over only the critical volumes of the relevant cells hit while D is conceptually an average over all cells in the sample,

$$D = \frac{\mathrm{Z1} + \mathrm{Z2} + \mathrm{Z3} + ...\mathrm{ZH}}{\mathrm{Ne}} = \frac{\mathrm{Z1} + \mathrm{Z2} + \mathrm{Z3} + ...\mathrm{ZH}}{\mathrm{Nh}} = \frac{Nh}{Ne} = \overline{Z.\frac{Nh}{Ne}}$$

where NH and NE are the numbers of hit and exposed

cells respectively. With large amounts of radiation all

exposed cells are hit, i.e. Nh= Ne and thus D = $\overline{Z}$. With LLR, however, $\overline{Z}$ remains constant with increasing Φ or D, and only the proportion of cells hit increases with increasing Φ.

A better quantity to use in this context is the fluence of charged particles through the critical volumes. Only when all the cells have received at least one hit (i.e. at "doses" of ~1cGy for low-LET radiation and ~1 Gy for high-LET radiation) does dose become a suitable surrogate for charged-particle fluence. The incidence of cells hit I_h for a given exposure can be related to the fluence Φ by the equation

$$I_h = \Phi .\ \theta$$

where θ is the cross-section of the critical volume.
However, because at low levels of radiation (LLR) (i.e. those of significance in radiation protection) a large proportion of the cells will have received no radiation, the mean dose per cell represented by the average tissue dose is not the same as the mean dose per dosed cell.

The relevant results for a sample of human lung are shown in Figure 3.1(a-c). At the lowest fluence of alpha particles, only a very small volume of cells and an even tinier volume of nuclei are hit. Those targets that are hit receive higher specific energies than would be expected from the distribution to the tissue overall.

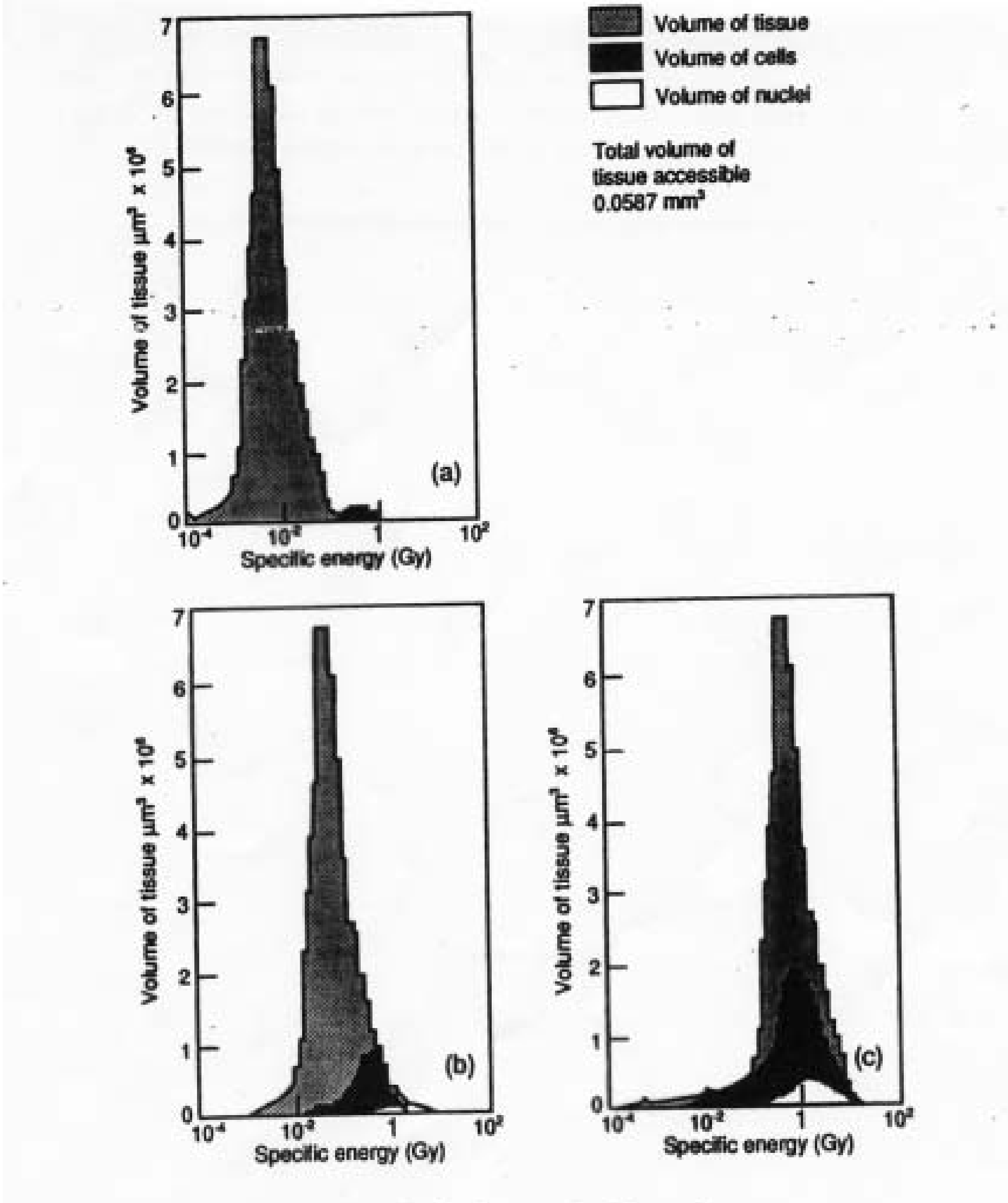

Figure 3.1. *Results for a sample of human lung.*

A linked study on workers who were occupationally exposed to radium showed that the incidence of malignant neoplasms could be fitted by a log-normal distribution as a function of dose (46 sarcomas, 19 carcinomas). Most significantly, none of the 1400 subjects with average skeletal doses of less than 10 Gy had developed any malignancy.

Cancer Registrations among Plutonium Workers at Sellafield

Plutonium Dose plus External Dose (mSv)

Cancer Site	**<10**	**10-19**	**20-49**	**50-99**	**100-199**	**200-399**	**400+**
Stomach	1(0.6)	1(0.4)	1(1.6)	2 (2.0)	3 (3.1)	4(3.1)	2(2.3)
Colon	0(0.5)	0(0.4)	1(1.8)	3(2.1)	2(2.8)	4(3.3)	4(3.1)
Lung	0(0.5)	1(0.8)	3(2.4)	4(4.0)	4(8.5)	17(15.5)	41(38.4)
Prostate	0(0.4)	0(0.3)	1(1.1)	4(1.5)	2 (2.3)	2 (2.8)	2 (2.8)
All Lymphatic and Haemotopoietic	1(1.4)	0(0.6)	3(1.5)	1(1.7)	0(2.6)	3(3.4)	7(3.8)

Authors' Conclusion:

For no cancer site was there a significant excess of cancer registrations compared with rates for England and Wales.

R.Z.Omar, J.A.Barber and P.G.Smith *British Journal of Cancer* 1999 79 1288-1301

The possible hazards from plutonium have been investigated by Voelz and his co-workers over a long period of time. Their studies covered some 5000 workers whose systemic burden ranged from 7 pCi to 230 pCi. The *452* observed deaths were significantly fewer than the 832 expected from all causes. The 107 deaths due to malignant neoplasms were also significantly fewer than the 167 expected from these diseases. Further detailed analysis revealed that that a threshold value of at least 230 uCi (i.e., at least six times the maximum permissible body burden) must exist for the induction of disease by plutonium.

Review of the 26 white male workers who did the original plutonium research at Los Alamos

(Voelz et **al.,** *Health Physics* 73611-619(1997)

After fifty years, 7 individuals died compared with an expected 16 deaths based on mortality rates of white males in the general population. The SMR was 0.43.
When compared with 876 unexposed Los Alamos workers of the same period, the plutonium workers' mortality rate was still only 0.77.

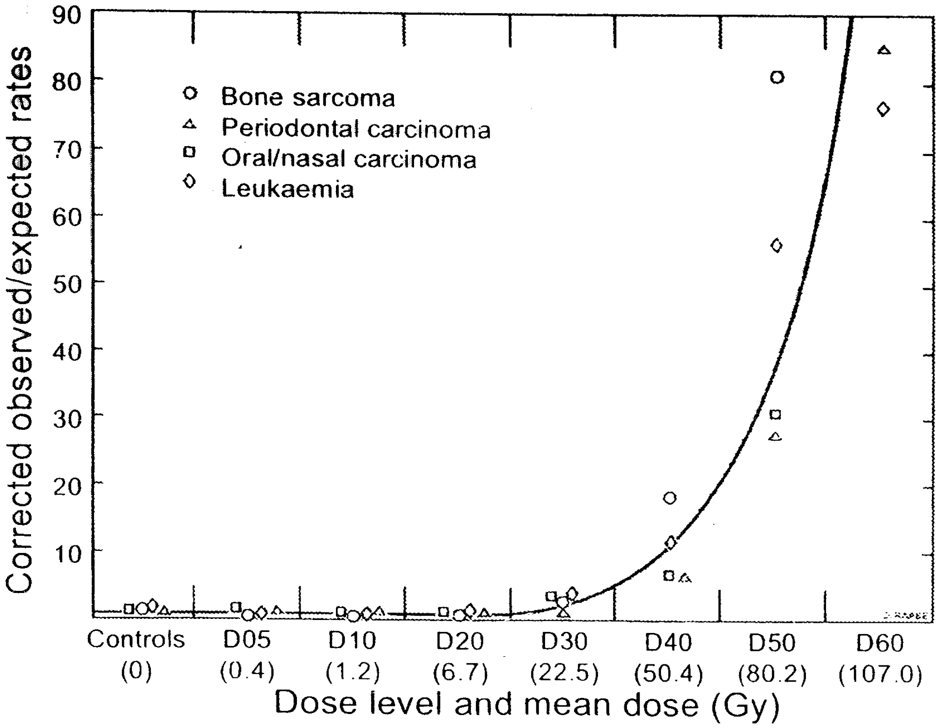

Figure 4. Incidence of fatal cancer as a function of dose group in beagles fed ^{90}Sr at the University of California, Davis[12].

These results indicate that intake levels as large as *50,pCi* of either isotope of radium produce bone changes that cannot be distinguished from changes sometimes appearing in unexposed individuals. Only when the intake levels are significantly larger do bone changes appear that indicate the presence of radium within the body.

Rowland, R.E., (1994), *Radium in Humans; A Review of US. Studies.*

The induction of liver tumors by ^{239}Pu citrate or
^{239}PuO2 particles in Chinese hamster.

Radiation Research, 96, 135-15 1 (1983)

In terms of average dose, the non-uniform irradiation was *2.5* times less carcinogenic than the uniform exposure.

ICRP Publication 31: Biological effects of inhaled radionuclides (1980). None of the many animal experiments with inhaled transuranic elements showed any evidence for an enhanced risk when the material is deposited in the form of discrete particles rather than dispersed.

"Mayak" Workers; Lung Cancer Rates as a Function o

Alpha-particle Dose to the Lung

**(From Table VI in Paper by Koshuurnikova et al.
Radiation Research, 149 366-371 (1998)**

Dose Range (Sv)

	<0.5	0.5 -	1.8-	4.0-	7.0-	13.0-	20.0-	30.0-	60.0-	120+
Average Dose (Sv)	.27	1,03	2.75	5.36	9.51	16.2	24.6	39.9	81.4	260.7
Persons	360	474	268	143	89	45	45	28	18	9
Observed/ Expected	1.41	1.04	1.70	2.55	5.12	6.86	11.0	9.86	19.9	35.13
(95% CI)	0.62	0.50	0.71	0.86	1.30	1.15	1.75	0.91	0.90	0.2
	3.25	2.18	4.07	7.57	20.1	40.8	69.4	107	439	∞